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EDITOR'S NOTE

Avoiding the High Cost of Peer Review Failures

I am a firm believer in the positive value of the peer review system. Not because it is perfect, but because of its potential to ensure the high value of published literature—a critical legacy of the scientific community. The conceptual merits of peer review are clear. It is a system serving both as gatekeeper to keep out inadequate content and a method of materially improving the quality of any work that is published. It is maintained through the shared effort of qualified experts with a vested interest in the authority of the official record. It is able to reach beyond local and interpersonal politics to deliver impartial evaluations of professional work. In its best form, it is objective, focusing on the product of academic labors without bias. Properly administered and supported by the research community, it can be a great example of the proverb "many hands make light work."

Although the benefits of robust peer review are clear, its effective execution can be undermined by all 4 groups critical to scientific publishing: authors, peer reviewers, editors, and publishers. Although many authors appreciate that peer review can confirm, and often strengthen, their professional efforts, some chafe at the obligation. They may believe that their work is beyond contestation or that the additional input and double-checks are not worth the aggravation.

Many peer reviewers understand that their diligent efforts will improve the literature and may enhance the ability of authors to contribute to it in the future. They accept, and may even welcome, the obligation to lead through an indirect form of mentorship. They know that the closer the work is to their own area of specialization, the greater their responsibility to deliver thorough, thoughtful, and critical evaluations. Some, however, are not sufficiently motivated to fully engage. Deficient reviews may reflect inadequate training or time pressure, but they can also reflect disregard or disinterest. Although it would be most appropriate for invitees to pass on review invitations that they are not willing and able to discharge with a high level of performance, reviewer credit can be claimed regardless of the effort delivered, creating a potential conflict for those wanting solely to document "productivity."

The role of the publisher has evolved over time. Historically, journals were developed by learned societies to disseminate the intellectual product of their membership. They served as the centerpiece of discipline-specific scientific endeavor and repositories of knowledge. Authors published their work to be debated in letters and rebuttals in the same journals and at scientific meetings. Parent societies provided oversight and served as publishers, with printers simply tasked with the physical creation of documents. Over time, however, the independent business of publishing has grown, with expanding services offered to remove the burden from often largely volunteer societies, or in some cases to replace the societies completely. Editors may now work for learned societies or publishers, with priorities potentially blurred.

Modern publishers are happy to publish high-value content that will be widely read and respected, but the business model thrives on volume to keep the presses turning. Although some societies maintain absolute control over published content, others have sold off their journals to independent publishers. An increasing number of new publications have also been developed with no learned society oversight. The push for open-access literature has had unexpected consequences, with "pay-to-play" (predatory) models becoming increasingly attractive for their profitability. Charging to publish work without the complicated multi-step processes and time required for high-quality peer review and revision can be appealing to those more interested in the financial benefits than in the literature record. It is important to recognize that many journals have maintained or have been developed with strong peer-review processes to ensure high-value scientific content, but others much less so. Publishers may also demonstrate different levels of commitment to peer-review rigor for different publications in their portfolio. Ultimately, the dedication to meaningful peer review can no longer be assumed, and both the authors submitting to journals and the readers of published work must be much more mindful of potential compromise.

The challenges to peer review are not new, but the surprising thing is how poorly we address these realities in training our students and even in interacting with our colleagues. It is this failure, whether intentional or unintentional, that allows the problem to grow.

It is possible that journals employing predatory practices outnumber those that do not. The legitimacy of journals cannot be confirmed by name or impact factor scores, and often not by promises made regarding peer-review standards or editorial board membership lists. Many predatory journals have credible and even inspiring names. They can also manufacture or manipulate impact factor scores and blatantly mislead regarding peer-review practices. Manuscripts that are submitted in sound form make deficient practices less obvious. Similarly, although some "editorial boards" are wholly fabricated, others may include individuals who have agreed to serve with the best intentions, with no awareness of shortcomings. Finally, some reviewers may be invited to conduct reviews without knowing that they are window dressing that will not alter editorial decisions. Collectively, these things can lend an air of respectability, or at least raise reasonable doubt that allows the fraud to continue.

Mindfulness, and more than a small degree of cynicism, is necessary to critically evaluate the legitimacy of any journal. Most importantly, this mindfulness must be passed on to students, and to colleagues where warranted, to avoid the compromise of peer review. Careful scrutiny is required because deficiencies that suggest predatory practices may not immediately be recognized. It is possible, for example, for an author to have had what is perceived to be a positive previous experience with a given journal. Publication fees can be confused with immediate access fees, and getting through "peer review" with no more than trivial editorial comments may seem reasonable for the person or team thinking that their words are gold. Being invited to review may also confer an aura of legitimacy. Such events could result in additional manuscripts being submitted to the same journal. Hard questions need to be asked within research teams early in manuscript development to ensure a shared valuation of peer review and of any target journals.

The inherently independent nature of researchers can lead to avoidance of conversations regarding research publication. Although the journal fit is obvious for some articles, odd choices might be attributed to journal shopping to get weaker manuscripts through, which may discourage discussion. This tacit acceptance through silence must be fought, however, to avoid supporting disreputable publications. Administrative units within institutions, working with library services, might be best positioned to raise awareness. Concerns over questionable peer-review practices should be discussed and guidelines established to ensure a low likelihood of being trapped by predatory journals.

There is a very practical reason for concern regarding journal selection. Institutions grappling with issues of predatory journal practices can raise questions that will be uncomfortable for authors. For example, did a person or team publish in such a journal inadvertently or to get around research weaknesses? Should full (or any) credit be given for publications in journals found to be predatory? Should job candidates with a history of publication in predatory journals be considered? Should articles published in journals employing predatory practices count in tenure packages? What scrutiny of the effort of flagged authors is warranted? Ultimately, the best way to avoid publication remorse is to ensure that the provenance of published work cannot be questioned.

Predatory publications will exist as long as the financial incentives remain enticing. Practice guidelines to protect institutional credibility are likely to develop in response to this reality. To reduce the risk of compromise, students and rising professionals must be sufficiently informed of the realities and hazards in publishing to ensure smart decision-making. All should learn not only to identify questionable publications, but also to carefully evaluate published work and to ensure the highest standard for their own efforts.

Reputable journals should welcome uninvited letters to the editor for open debate on perceived deficiencies in the work published. Maintaining an open forum is one of the best ways to hold authors and journals accountable. The most credible and responsible peer-reviewed journals should be supported through submissions, high-value review efforts, and promotion among colleagues and trainees. In the same way that poor literature should not be cited, journals that do not provide rigorous peer review and open debate should be shunned. The goal is to make their deficiencies more evident and to make them more easily avoided.

Wilderness & Environmental Medicine maintains a high standard for peer review and will continue to do so to benefit authors, readers, and the research record. Letters to the editor are welcome to promote open scientific discourse.

Neal W. Pollock, PhD
Editor-in-Chief

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ORIGINAL RESEARCH

The Role of ProBNP on Prognosis in Scorpion Stings

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Introduction—Scorpion stings are a major health problem with potentially fatal consequences. Children under the age of 10 y especially face a great risk. Predicting the prognosis is important in reducing mortality and morbidity because it enables the use of early treatment options. In this study, we examine the relationship between proBNP and prognosis in scorpion stings.

Methods—This is a retrospective analysis of patients aged ≤18 y who were admitted to the child emergency service with a scorpion sting. We examined the demographical data, clinical findings, laboratory records, treatments, and results of the patients. We classified stage 1 and stage 2 scorpion envenomation as group 1 (mild-moderate) and stage 3 and 4 as group 2 (severe). A *t* test was used for normally distributed data, and the Mann-Whitney U test was used for nonnormally distributed data. The correlation analysis was done using the Spearman test.

Results—There were 32 (74%) patients in the mild-moderate group and 11 (26%) in the severe group. ProBNP 1 was significantly higher in the severe group at admission (P=0.016). There was no difference between the troponin I values (P=0.051). ProBNP 2 (12th hour) and proBNP 3 (24th hour) were higher in the severe group (P=0.001 and P=0.032, respectively). There was a negative correlation between proBNP and echocardiographic findings involving ejection fraction and shortening fraction (r=–0.703, P=0.002).

Conclusions—In our study, the first proBNP values were significantly higher in the severe group. This suggests that proBNP may be beneficial in predicting prognosis.

Keywords: poisoning, BNP, venom, scorpionism, troponin I

Introduction

Approximately 1 to 1.5 million scorpion stings occur around the world annually, of which 3000 result in death.[1] Children and adolescents under the age of 10 y particularly face a great risk of scorpion envenomation.[2] Of the scorpions found in North Africa, the Middle East, Israel, Lebanon, Iran, and Turkey, *Leiurus* spp often cause damage that leads to autonomic dysfunction.[3] Alpha toxins in scorpion venom act by binding to sodium channels in the cell membrane and inhibiting action potential inactivation. This effect, through a synergistic effect with other ingredients in scorpion venom, causes the prolongation of depolarization and excessive release of acetylcholine from parasympathetic ganglia as well as excessive release of epinephrine and norepinephrine from sympathetic ganglia and the adrenal glands. Excessive release of these neurotransmitters results in an autonomic storm. Cholinergic findings are bronchore, salivation, sweating, bronchospasm, priapism, lacrimation, vomiting, diarrhea, and bradycardia. Hypertension, tachycardia, and agitation develop with sympathetic stimulation. Severe poisoning can cause direct myocardial damage, leading to arrhythmia, myocarditis, pulmonary edema, cardiogenic shock, and multisystem organ failure. It is believed that myocarditis occurring as a result of scorpion envenomation may be caused by increased catecholaminergic effect or through toxins directly affecting the heart muscle.[1]

Staging systems have been established to assess regional scorpion envenomation that causes autonomic storms. Accordingly, stage 1 has been defined as local

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effect only, stage 2 as systemic autonomic effects, stage 3 as cardiotoxicity including heart failure with cardiogenic shock or acute pulmonary edema with hypotension, and stage 4 as progressive cardiogenic shock with coma, seizure, or multiorgan failure.[2]

Natriuretic peptides are protein molecules released from the ventricles in response to volume or pressure load. Brain natriuretic peptide (BNP) is synthesized and released in the myocytes of the left ventricle, mainly in response to the volume expansion of the ventricle or the stretching of myocytes owing to pressure loading.[4] Initially, the hormone is released as pre-proBNP and splits into proBNP. ProBNP also dissociates into active BNP and inactive NT-proBNP.[5] BNP is a biologically active molecule that improves myocardial relaxation by causing natriuresis, vasodilation, and diuresis.[6]

In our study, we examined the demographic, clinical, and laboratory findings of patients who were admitted with scorpion stings. We aimed to evaluate cardiac enzymes and proBNP to show cardiac damage in scorpion stings.

Methods

We retrospectively analyzed patients aged ≤18 y who were admitted to Kahramanmaraş Sütçü İmam University Medical Faculty child emergency department and Kahramanmaraş Necip Fazıl city hospital child emergency service for scorpion sting between January 1, 2019 (the first case was on May 20, 2019) and October 31, 2019 (the last case was on August 26, 2019). Research approval was received from the ethics committee of Kahramanmaraş Sütçü İmam University medical faculty of medicine (2019/18-05). We were able to retrospectively access the medical records of patients from our center and Kahramanmaraş Necip Fazıl city hospital. We examined the demographic data, clinical findings, laboratory records, treatments, and results of the patients.

We divided the patients into 2 groups according to factors that can help in predicting poor prognosis. We classified stage 1 and 2 as group 1 (mild-moderate) and stage 3 and 4 as group 2 (severe). The patients were treated with antivenom produced by the Public Health Institution of the Republic of Turkey (Scorpion Antivenom, thsk.tglab@saglık.gov.tr). This antivenom contains immunoglobulins that neutralize *A crassicauda* venom. It also has a polyvalent effect and has the power to neutralize other scorpion venom. Each 1 mL of immunocerum contains immunoglobulin that neutralizes scorpion venom of ≥50 LD (lethal dose) *A crassicauda* species.

Table 1. Demographic and clinical parameters

Parameters	*Mild-moderate n (%)*	*Severe n (%)*	*P*[a]
Sex			
Female	15 (47)	5 (45)	0.93
Male	17 (53)	6 (55)	
Age (y)			
0–5	8 (25)	9 (82)	<0.05
6–10	10 (31)	1 (9)	
11–18	14 (44)	1 (9)	
Site of scorpion sting			
Head/Neck	2 (6)	0 (0)	0.69
Trunk	1 (3)	1 (10)	
Upper extremity	13 (41)	5 (50)	
Lower extremity	16 (50)	4 (40)	
Stings (n)			
1	28 (88)	10 (91)	0.92
2	4 (13)	0 (0)	
3	0 (0)	1 (9)	
Time of arrival at hospital (h)	1 (0.5)[b]	1(0)[b]	0.31
Length of hospital stay (d)	0.5 (2)	3 (1)	0.0
Mortality	No	No	

[a]Mann-Whitney U test or χ^2 test.
[b]Median (interquartile ratio).

STATISTICAL ANALYSIS

The data obtained in the study were produced using SPSS version 25 (IBM SPSS Statistics 25). Categorical variables were expressed as numbers and percentages and numerical variables as median and interquartile values. Categorical variables between groups were compared using the χ^2 test. Normality of numerical data was evaluated through the Kolmogorov-Smirnov test. Normally distributed data were compared with the *t* test, and nonnormally distributed data were compared using the Mann-Whitney U test. The correlation analysis was done using the Spearman test. Statistical significance was accepted as $P<0.05$.

Results

The data of 43 patients who were monitored for scorpion stings were analyzed. The demographic data of the patients are presented in Table 1. The patients and their families described the scorpion responsible for the stinging. Usually, the scorpions that stung the patients were killed by families. We classified stage 1 and stage 2 scorpion poisoning as group 1 and stage 3 and 4 as group 2 according to factors that can help in predicting poor

Table 2. Drugs given to patients

Patient no.	*Antivenom* (*1 vial: 1 mL*)	*Adrenaline* ($\mu g \cdot kg^{-1} \cdot min^{-1}$)	*Dobutamine* ($\mu g \cdot kg^{-1} \cdot min^{-1}$)	*Milrinone* ($\mu g \cdot kg^{-1} \cdot min^{-1}$)	*Doxazosine* ($mg \cdot kg^{-1}$)
8	1	0.2	10		
21	1	0.2	10	0.2	0.03
32	2				0.03
28	2				0.03
38	1				0.03
16	1				
40	1	0.2			0.03
17	1				
33	1				0.03
34	1				
23	1				

prognosis. There were 32 (74%) patients in the mild-moderate group and 11 (26%) in the severe group. The median age of the mild-moderate group was 9.75 y (Q1=5.0, Q3=14), and the median age of the severe group was 3.75 y (Q1=2.0, Q3=5.0). The median weight of the mild-moderate group was 28 kg (Q1=18.25, Q3=46.75), and the median weight of the severe group was 15 kg (Q1=13, Q3=27). The severe group had lower body weight (P=0.018). The time to admission to the hospital was not different between the groups (P=0.31). There was also no difference between the groups in terms of the site of the scorpion sting and the number of stings (P=0.64 and P=0.84, respectively). The most common finding was localized pain. Vomiting was present in 1 patient in the mild-moderate group and 6 patients in the severe group. There was syncope present in 1 patient, sweating in 1 patient, and dizziness and abdominal pain in 1 patient. Compartment syndrome developed in 1 patient. Fourteen patients initially had hypertension during admission, and 2 patients had hypotension and hypothermia. The 2 patients with hypotension and hypothermia were followed up for pulmonary edema. There was no difference in hypertension between the groups (P=0.75). Three patients had sinus tachycardia.

One vial of antivenom was given to all patients with systemic involvement, and 2 vials were given to 2 patients in the severe group. Along with the administration of antivenom, steroids and antihistamines were administered to the patients. The patients did not develop a reaction against antivenom treatment. Depending on the patient's clinical condition, other drugs were given, as shown in Table 2. One patient received adrenaline and dobutamine; 1 patient received adrenaline, dobutamine, milrinone, and doxazoin; and 1 patient received adrenaline and doxazosin. Six patients were given doxazosin in total.

Laboratory findings are shown in Table 3. Leukocytosis, hyperglycemia, and transaminase levels were found to be high in the severe group (P=0.002, P=0.003, and P=0.001 for AST (aspartat aminotransferase); P=0.008 for ALT (alanin aminotransferase)). In the blood gas analysis, although pH, HCO3, and base excess were different in the severe group, there was no difference between the groups in terms of lactate and pCO2 (P=0.000, P=0.003, P=0.005, P=0.59, and P=0.12, respectively). Thrombocytosis was not observed in our patients at the time of first admission (P=0.14). There was no difference between the groups in terms of electrocardiographic findings.

In the blood samples taken at the time of first admission, there was no difference between troponin I values (0th hour) (P=0.051) (Figure 1). ProBNP 1 (0th hour) was significantly higher in the severe group (P=0.016) (Figures 2 and 3). In blood samples taken 12 h and 24 h after the first admission, proBNP 2 (12th hour) and proBNP 3 were higher in the severe group (P=0.001; P=0.032, respectively). Troponin I (12th hour) was high enough to make a difference in the severe group, whereas troponin I (24th hour) was not different (P=0.011 and P=0.84, respectively). In terms of echocardiographic findings (Table 4), the ejection fraction and shortening fraction were significantly lower in the severe group (P=0.006 and P=0.005, respectively). There was a negative correlation between proBNP and echocardiographic findings involving ejection fraction and shortening fraction (r=–0.703, P=0.002).

Discussion

Cardiopulmonary complications are frequently the cause of death in envenomation related to scorpion stings.[7] Autonomic storm is generally observed in scorpion sting cases in our country, and the follow-up and treatment are arranged according to autonomic storm.[3] In a study with 64 patients in Turkey, the death rate was 12%;

Table 3. Laboratory results

	Mild/Moderate median (IQR)	*Severe median (IQR)*	*P Mann-Whitney U test*
White blood cell ($10^3 \cdot mL^{-1}$)	9595 (4995)	24330 (20160)	0.002
Neutrophile ($10^3 \cdot mL^{-1}$)	5050 (3735)	10330 (12080)	0.012
Lymphocyte ($10^3 \cdot mL^{-1}$)	3505 (3553)	3020 (13760)	0.38
Hemoglobin ($g \cdot dL^{-1}$)	13 (1.45)	12.2 (1.7)	0.35
Platelets ($10^9 \cdot mL^{-1}$)	305000 (124750)	363000 (276000)	0.14
Glucose ($mg \cdot dL^{-1}$)	106 (20)	128 (126)	0.003
BUN ($mg \cdot dL^{-1}$)	16.4 (21.2)	19.3 (18.8)	0.34
Creatinine ($mg \cdot dL^{-1}$)	0.43 (0.33)	0.40 (0.23)	0.27
Aspartate transaminase ($U \cdot L^{-1}$)	25.5 (11.5)	35 (6.5)	0.001
Alanine transaminase ($U \cdot L^{-1}$)	15 (7)	22 (23)	0.008
Uric acid ($mg \cdot dL^{-1}$)	3.6 (1.3)	5 (0)	0.033
pH	7.39 (0.05)	7.34 (0.09)	0.000
pCO_2 (mm Hg)	42.5 (7)	37.1 (6)	0.12
HCO^3 ($mmol \cdot L^{-1}$)	24.8 (2.8)	18.5 (5.2)	0.003
Base excess	1.49 (4.3)	–7.8 (7.7)	0.005
Lactate ($mmol \cdot L^{-1}$)	1.8 (1.0)	1.5 (2.9)	0.59
ProBNP 1 (0 h) ($ng \cdot L^{-1}$)	69 (7)	349 (33)	0.016
ProBNP 2 (12 h)	221 (21)	3070 (777)	0.001
ProBNP 3 (24 h)	191 (18)	447 (1056)	0.032
Troponin-I 1 (0 h) ($\mu g \cdot L^{-1}$)	0.01 (0.0)	0.02 (0.07)	0.051
Troponin-I 2 (12 h)	0.01 (0.0)	0.1 (0.11)	0.011
Troponin-I 3 (24 h)	0.05 (0.11)	0.31 (0.28)	0.84
Creatinine kinase 1 (0 h) ($U \cdot L^{-1}$)	144 (134)	336 (238)	0.006
Creatinine kinase 2 (12 h) ($U \cdot L^{-1}$)	90 (76)	370 (506)	0.005
Creatinine kinase 3 (24 h) ($U \cdot L^{-1}$)	50.5 (15)	310 (282)	0.004

BUN, blood urea nitrogen; IQR, interquartile ratio.

the death rate in another study was reported as 0%.[8,9] Similarly, none of the patients in our research died (0%). So far, 13 species from 8 families have been identified, including Buthidae, Chacthida, Iurida, and Scorpionidae families in Turkey. *Androctonus crassicauda* and *Leiurus quinquestriatus*, which are members of the Buthidea family, are usually seen in Kahramanmaraş.[10] Recently, a new yellow scorpion species named *Leirus abdullahbayrami* has been reported in Kahramanmaraş.[11,12]

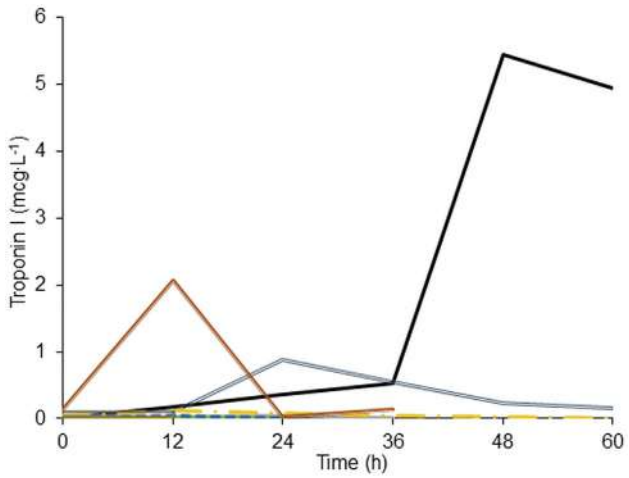

Figure 1. Troponin I levels in patients in the severe group.

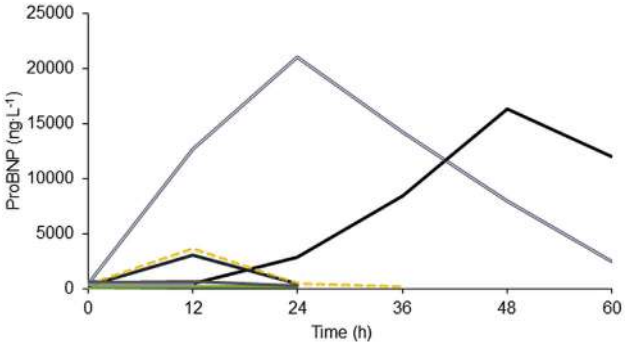

Figure 2. ProBNP levels in patients in the severe group.

In our study, because the patients described the scorpions that stung them as being yellow and black, it was presumed that the black scorpion is *Androctonus crassicauda* and the yellow *Leiurus quinquestriatus* or *Leirus abdullahbayrami*, in line with previous studies, although the species distinction was not made in accordance with the patients' clinical results.

In this research, hypertension was observed in 14 (33%) patients, and there was no statistically significant difference between the mild-moderate and severe groups. Although thrombocytosis was suggested as a marker of clinical deterioration in a previous study, our study found, in accordance with the literature, that thrombocytosis was not observed at the time of first presentation.[3,13,14] BNP and NT-BNP may be elevated in patients with myocarditis.[15] In 1 study, proBNP levels were evaluated in scorpion-associated myocarditis, and it was suggested that serial proBNP monitoring might be important in scorpion poisoning and that further studies should be conducted on the topic.[16] In our study, proBNP was observed to be elevated without significant troponin I elevation. However, there was a difference between the second troponin I values taken at the 12th hour. This shows that the proBNP value is important during early admission for predicting the prognosis of scorpion stings. Thus, we suggest that proBNP can be an important parameter in terms of directing prognosis, follow-up, and treatment in scorpion stings.

In previous studies, a reduction in ejection fraction was detected in patients stung by scorpions.[17] In our study, echocardiography was performed on all patients in the first hour after admission to the hospital. The ejection fraction was significantly lower in the severe group. The shortening fraction was also significantly lower in the severe group. A significant negative correlation was found between proBNP and ejection fraction and

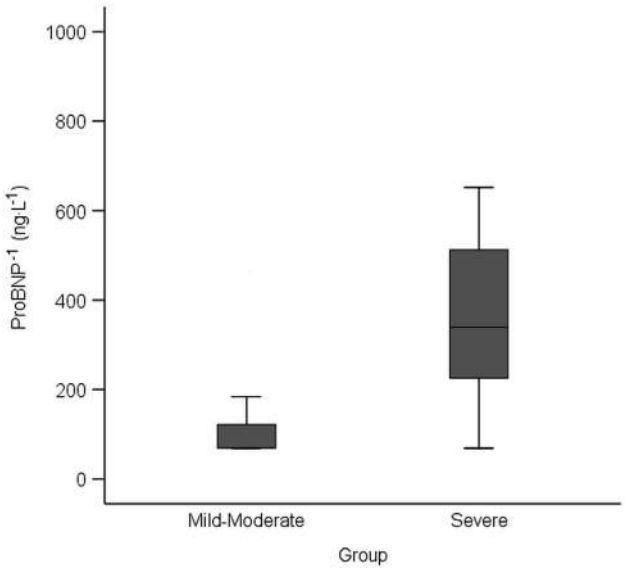

Figure 3. ProBNP levels in at first admission between groups. ProBNP levels were higher at the time of first admission in the severe group.

Table 4. ECHO and ECG results

	Mild/Moderate[a]	*Severe*[a]	*P*[b]
ECG			
Heart rate (beats·min^{-1})	91 (41)	110 (63)	0.30
QTc (s)	0.39 (0.03)	0.38 (0.03)	0.47
Echo			
EF (%)	76 (7)	68 (9)	0.006
SF (%)	43 (6)	35.5 (8)	0.005

ECG, electrocardiography; ECHO, echocardiography; EF, ejection fraction; IQR, interquartile ratio; SF, shortening fraction.
[a]Median (IQR).
[b]Mann Whitney U test.

shortening fraction. This shows that the higher the proBNP, the more severe the cardiac involvement can be. In addition, the initial proBNP value can predict prognosis and show whether the patient should be referred to advanced centers. It is believed that patients with high proBNP will require a longer intensive care stay because prognosis will be poor.

LIMITATIONS

The retrospective nature of our study and the low number of patients constitute the limitations of our study. Our data were limited to what was recorded on the patient records. The envenoming scorpion was determined by the descriptions provided by patients and their relatives. Prospective studies with a higher number of cases are needed to address the deficiencies in our study.

Conclusions

At the time of initial hospital admission, proBNP may be more useful than troponin I in predicting the prognosis.

Author Contributions: Study concept and design (UUG, Sİ); acquisition of the data (SD, TD, SY); analysis of the data (Sİ); drafting of the manuscript (UUG, Sİ, TD); critical revision of the manuscript (UUG, Sİ); approval of final manuscript (Sİ, UUG, SD, SY, EA).
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ORIGINAL RESEARCH

Snakebites Reported to the Kentucky Regional Poison Control Centers for the Years 2012-2016

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Introduction—Snake envenomations cause significant morbidity and mortality. The goals of this study were to assess the epidemiology of snakebites in Kentucky and treatment strategies used by physicians reporting to the Kentucky Regional Poison Control Centers.

Methods—This was a descriptive epidemiologic study compiling clinical data on snakebites reported to the Kentucky Regional Poison Control Centers from 2012 to 2016. We built a database of the patient demographics, treatment, and clinical course of each snakebite reported in the study period. Attention was paid to all antivenom interventions and use of contraindicated therapies.

Results—We compiled 674 total records. Patient age was 34±18 y (mean±SD), with males (71%) predominating. Most (97%) bites were to a distal upper or lower extremity. The majority (78%) occurred at a private residence. Most reports came between May and September (88%). Of the 674 patients, 24% (n=159) were classified as moderate or severe. Two hundred thirty (34%) patients were admitted to the hospital. Forty-six patients (7%) received surgical consultation, and 12 (2%) underwent surgical intervention. One hundred fifty-three patients received antivenom, with 6±3 vials used per patient. Length of stay was 3±2 d when antivenom was administered and 2±2 d when not administered. Six cases of coagulopathy were noted. Multiple contraindicated therapies were noted.

Conclusions—Snake envenomations are a prevalent public health concern for residents of Kentucky, chiefly during summer months. Significant medical intervention is being performed for many patients, with a high prevalence of contraindicated therapies. More data are needed to fully characterize the epidemiologic impact and appropriateness of the interventions being applied.

Keywords: envenomation, antivenom, copperhead, pit viper, debridement, rattlesnake

Introduction

The Centers for Disease Control and Prevention estimates that >7000 people per year sustain a snakebite, although studies have shown a range between 5000 and 9000.[1,2] When these bites are the result of a venomous snake, they represent an uncommon but important illness owing to the potential for prolonged morbidity and, rarely, mortality.[1,3] Although death is a rare sequela of snakebites, with 0 to 4 reported per year, the number would likely be higher without proper medical care.[4,5]

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Victims are most often bitten on their distal extremities, lower more than upper, and are more likely to be Caucasian and male.[1,4] The most common age range of a snakebite patient is 20 to 30 y, although there is a small peak in children below the age of 12 y.[1,4,6,7] Bites typically occur at dawn and dusk, during the snake's natural predatory activities, and in the warmer months of May through October.[7] There are multiple species of venomous snakes in the United States, including rattlesnakes, copperheads, cottonmouths/water moccasins, and coral snakes.[7] The state of Kentucky itself is home to 4 types of venomous snakes, all in the Crotalinae subfamily of Viperidae, commonly referred to as "pit vipers": copperheads (*Agkistrodon contortrix*), cottonmouths (*Agkistrodon piscivorus leucostoma*), timber rattlesnakes (*Crotalus horridus*), and the western pygmy rattlesnake (*Sistrurus miliarius streckeri*).[8] Copperheads and timber

rattlesnakes are widely distributed throughout the state, but both the western cottonmouth and western pigmy rattlesnake are generally located only in the most western parts of the state.[8]

Snake venom is a complex mixture of biologically active compounds that vary by region, species, diet, and genetics.[7] Crotalinae venom in particular typically contains a variety of compounds that have cytotoxic, myotoxic, and hemotoxic compounds that can result in both local tissue damage and systemic effects such as hemolysis and coagulopathies.[9,10] Although less common, crotaline venom may also contain neurotoxic compounds that can result in weakness, cranial nerve palsies, and potentially respiratory failure.[5,7] Timber rattlesnakes are an example of one such snake that commonly possesses a neurotoxin.[7] Typical local symptoms include pain, erythema, and edema that can potentially encompass an entire limb.[4,11-13] These localized symptoms may worsen in the first few days after envenomation and persist for 14 to 21 d, and occasionally longer.[4]

Envenomations are classified into 4 broad categories: dry bite, mild envenomation, moderate envenomation, and severe envenomation.[7,14] Dry bites result in no localized or systemic symptoms and no coagulopathy on serial measures. Mild to severe envenomations are a spectrum of progressive localized and systemic symptoms, with moderate and severe envenomations potentially having signs of coagulopathy. Severe envenomations can result in severe localized edema and pain, gross coagulopathies on laboratory assessment, clinically significant bleeding, hypotension, angioedema, or anaphylaxis.

Crotaline antivenom is given as an initial starting dose of 4 to 6 vials with repeat dosing until initial control is obtained. Once initial control is obtained, maintenance dosing of 2 vials at 6, 12, and 18 h is recommended.[11,15] Expert consultation with a local poison control center is available to aid in the management of patients to help guide providers on antivenom administration and avoidance of contraindicated therapies.

Several therapies are not recommended because they do not improve outcomes over antivenom and can potentially result in more harm to patients. The use of ice, local incision, suction at the bite site, tourniquets, compression bandages/pressure immobilization, and prophylactic fasciotomies can result in more tissue damage without any benefits.[16] Use of nonsteroidal anti-inflammatory drugs should be avoided for pain control in favor of opiates because they may potentiate venom's effects on platelet aggregation and coagulation. Antibiotics should also be avoided because secondary infections of snakebites are rare and antibiotics have not shown any benefit in outcomes.[17] Despite these recommendations, individual practice still varies.[7,14]

The purpose of this study was to identify the demographics of snakebite incidents, snakebite victims, and outcomes in the state of Kentucky. We also made note of the use of antivenom, the specific doses of antivenom given, and any documented use of a contraindicated therapy.

Methods

This study was reviewed by the University of Louisville institutional review board and was determined to be exempt according to 45 CFR 46.101(b). We accessed records from the Kentucky Regional Poison Control Center (KRPCC) with a diagnosis of snakebite between the years of 2012 and 2016, which provided 674 unique records. From these records, multiple data points were extracted and entered manually into a database by the authors. Patient characteristics including age, sex, and potential intoxication were recorded. Envenomation information including location on body; time, date, and geographic location of the bite; and type of snake reported was also recorded. Location on body was grouped into the following categories: head and neck; torso, including chest, abdomen, and back; proximal upper extremity, defined as proximal to and including the elbow; distal upper extremity, defined as distal to the elbow; proximal lower extremity, defined as proximal to and including the knee; and distal lower extremity, defined as distal to the knee. Location where the bite occurred was grouped into the following categories: private residence, public area or park, zoo, or other. Type of snake reported was broken down into the following categories: copperhead, cottonmouth, rattlesnake, unknown, and other. Signs and symptoms of the bite were also obtained from the description provide,d including pain, swelling, ecchymosis, blister or bullae, nausea and/or vomiting, paresthesia, fasciculations, and necrosis. Severity of bite was defined as dry if there was no pain or swelling, mild if localized pain/swelling did not cross a joint, moderate if pain or swelling crossed >1 joint but ≤2 joints, and severe if the swelling affected >2 joints, a coagulopathy was detected, or hypotension occurred. Coagulopathy was defined as an international normalized ratio greater than 2.0, prothrombin time greater than 20, platelet count less than 50,000, or fibrinogen less than 50.

To help ensure inter-rater reliability, the first 25 cases were reviewed by the 4 reviewers as a group to ensure each rater was familiar with the format of KRPCC records and the aforementioned bite severity definitions. In the event that KRPCC records presented a questionable case, group consensus of the 4 reviewers was obtained. Data are presented as mean±SD or percentage, as appropriate.

Table 1. Victim age distribution of snakebite reported for the years 2012 to 2016

Victim age (y)	*Number*	*Percentage*
<18	137	20
18–35	220	33
36–55	199	29
>55	94	14
Unknown age	24	4

Results

DEMOGRAPHICS

Males comprised 71% of snakebite victims reported to the KRPCC (Table 1). Age was 34±18 y for males and 34±20 y for females. Ninety-seven percent of bites were to a distal upper or lower extremity (48% each). Males were more often bitten on the upper extremities (61%), and females were more often bitten on the lower extremities (75%). Seventy-eight percent of bites occurred at a private residence. Seventeen people were reportedly intoxicated at the time the snakebite occurred. Eighty-eight percent of snakebites reported occurred in the months of May through September (Figure 1). July had the most snakebites for each year examined. The time of day for calls to the KRPCC was approximately 5±6 hours after noon.

Copperheads made up 53% (n=358) of reported snakebites in which the snake could be identified. Rattlesnakes made up 3% (n=20). Thirty-seven percent (n=46) of snakebites reported were from an unknown type of snake. The remainder was grouped into an "other" category that included nonvenomous snakes only.

CLINICAL COURSE

One hundred fifty-nine (24%) snakebites reported were classified as moderate or severe based on the symptoms reported to the KRPCC (Table 2). Symptomatically, 88% of patients reported pain, with 71 and 28% of patients reporting swelling and ecchymosis, respectively. Nausea and vomiting were the most common systemic symptoms at 9%. Six cases of coagulopathies were noted; 5 of these were from rattlesnake bites, and 1 was from an unknown type of snake.

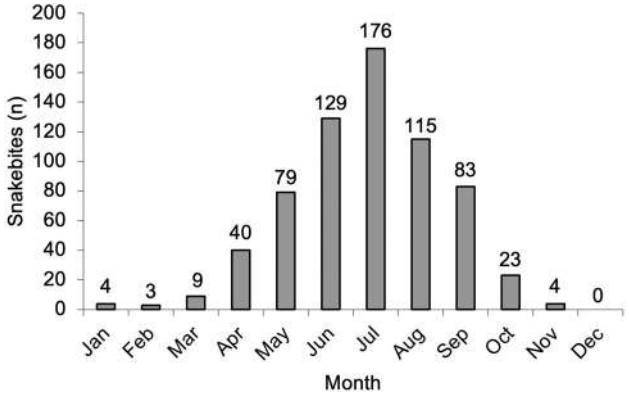

Figure 1. Distribution of snakebites reported by month.

Thirty-four percent, or 230 patients, were admitted to the hospital; 69 (10%) of those were admitted to the intensive care unit. Fifty-one percent of patients who presented to the emergency department were discharged home, with another 4% signing out against medical advice. Twenty patients returned to the emergency department within 24 h (6% of those discharged from the emergency department). Forty-six patients received surgical consultation, and 12 underwent surgical intervention. Seven patients underwent fasciotomies, and 1 patient received a partial digit amputation. Of the patients who received fasciotomies, 5 bites were from copperheads and 2 were from an unknown type of snake. The amputation resulted from a bite from an unknown type of snake. Five patients received debridement. Debridement was performed on 2 copperhead envenomations, 1 rattlesnake envenomation, and bites from 2 unknown snakes. Fifty percent of patients who received surgical intervention did not receive antivenom. One death was reported in a person who was bitten on a distal extremity by a rattlesnake.

Forty-one percent of envenomations received antivenom in this cohort. One hundred fifty-three patients received antivenom, with an initial and total dose of 4±1 and 6±3 vials, respectively. The maximum number of vials given was 21. Of bites described as moderate or severe, 55 and 79% received antivenom, respectively. Length of stay was 2±2 d, with lengths of stay of 3±2 d and 2±2 d if antivenom was administered and not administered, respectively. Contraindicated therapies including initial antibiotic administration, application of ice, nonsteroidal anti-inflammatory drug or aspirin administration, tourniquet application, steroids, and incision can be seen in Table 3. Antibiotics were the most commonly used contraindicated therapy.

Two hundred thirty (34%) patients were admitted to the hospital. Length of stay in the hospital was 3±2 and 2±2 d with and without antivenom, respectively. Sixty-four percent of those who were admitted and received antivenom had moderate or severe snakebites, compared to 38% of those who were admitted without receiving antivenom.

Discussion

DEMOGRAPHICS AND LOCATION WITHIN KENTUCKY

The male sex of victims and distal extremity predominance of envenomations is consistent with prior studies in the United States.[18-20] A concentration of envenomations

Table 2. Classification of snakebite severity for the years 2012 to 2016

Type of bite	*Definition*	*Incidence*	*Percentage of total bites*
Dry	No pain or swelling	239	36
Mild	Local pain/swelling crossing ≤1 joint	273	40
Moderate	Pain/Swelling crossing 2 joints	141	21
Severe	Pain/Swelling crossing >2 joints, coagulopathy, hypotension	19	3
Unable to determine		2	0

in the summer months is also consistent with prior studies.[19,20] The proclivity of males to be bitten on an upper extremity and females to be bitten on a lower extremity was consistent with data from the North American Snakebite Registry.[20] Hospital admission rates were similar to the nationwide figure of 29%.[21]

The concentration of snakebites reported to the KRPCC around the state of Kentucky is heat mapped in Figure 2. Metropolitan areas demonstrated a higher concentration of snakebites reported. This is possibly secondary to their higher populations. The mountainous areas of southeastern Kentucky also demonstrated higher relative concentrations of snakebites compared with the rest of the state, despite not containing populous areas. The cause of this concentration is unclear. One possibility is the practice of snake handling in certain religious practices that are common in Appalachia.

ANTIVENOM ADMINISTRATION

Previous studies have shown that 72% of copperhead bites and 84% of all pit viper bites received antivenom.[19] This was significantly higher antivenom administration than in this cohort. Bite severity between these groups is difficult to compare given the available data. Increased bite severity correlated highly with antivenom administration, with 79% of severe bites receiving antivenom and less than 1% of dry bites receiving antivenom. Extremes of age did not seem to be a factor: The distribution of antivenom administration to patients aged <18 and >65 y was similar to the distribution of those ages in the cohort. The often-recommended initial dose of 4 to 6 vials[2] is consistent with the KRPCC recommendation and is consistent with what was administered in this cohort.

CONTRAINDICATED THERAPIES

Several therapies were reported frequently in our study despite typically being considered contraindicated therapies that show no benefits and occasionally cause harm[7,14] (Table 3). Antibiotics were the most commonly used contraindicated therapy at 21% of snakebites reported. Several contraindicated therapies were administered by emergency medical service personnel, including incision, suction, tourniquets, and application of ice. Incision and suction in particular have been associated with local tissue injury and should be avoided when treating snakebites.[14,22,23] Nonsteroidal anti-inflammatory drugs, although contraindicated owing to concern about platelet dysfunction, have little evidence to suggest they should be avoided in copperhead bites, the main source of venomous snakebites in Kentucky.[14,24] Tourniquets, although generally not recommended, have mixed data.[14,22]

Antibiotics are relatively contraindicated because crotaline envenomations are considered as at low risk for secondary infection, but antibiotic administration is associated with side effects and potential harm.[17] The appropriateness of antibiotic use in this cohort is difficult to determine given the limited clinical information obtained through KRPCC records. Inflammation versus infection can be challenging to assess when determining whether to administer or prescribe antibiotics. The timing of the presentation in relation to the bite and expert consultation are important factors in determining whether antibiotics are indicated.[14] Previous studies have shown a much lower rate of antibiotic use (8 vs 22%) than what was administered per the KRPCC records.[20]

LIMITATIONS

KRPCC's records are not standardized; therefore, much of this data was collected from free text fields in KRPCC's database. These data represent an incomplete medical record and do not provide a complete clinical picture of each snakebite. Data were also heterogenous to

Table 3. Contraindicated therapies reported for the years 2012 through 2016

Therapy	*Incidence*	*Percentage*
Ice	28	4
Incision	5	1
Suction	7	1
Tourniquet	19	2
Steroids	16	2
Aspirin/Nonsteroidal anti-inflammatories (ASA/NSAIDS)	20	3
Antibiotics	146	22

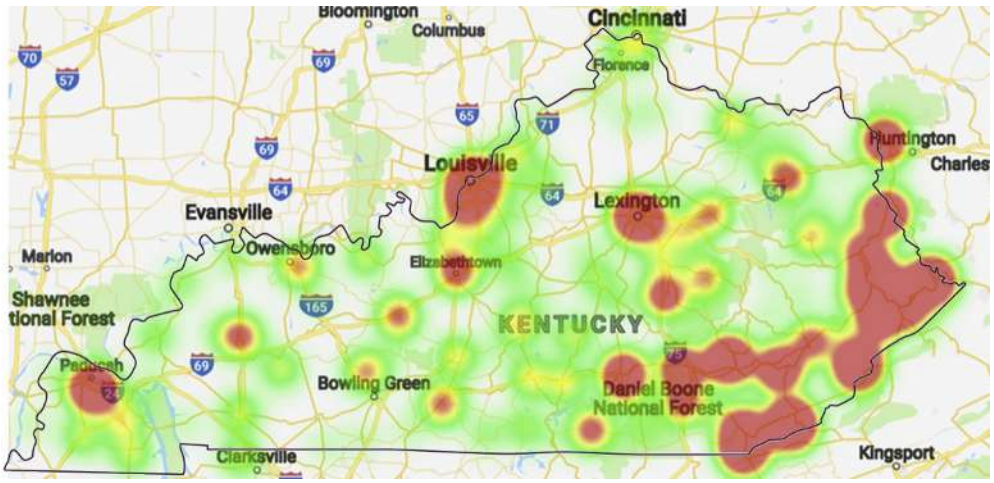

Figure 2. Heat map representing the concentrations of snakebites reported to the Kentucky Regional Poison Control Centers mapped by the zip code of the caller. Map generated from Google Maps and reproduced in accordance with their permissions guidelines.

a large extent given the free text nature of the records and the difficulty of obtaining follow-up information from busy hospital staff. Although efforts were made to increase interrater reliability, there was some potential for heterogeneity in snakebite severity assigned across raters.

Snakebite figures are likely underreported to KRPCC. A comparison of snakebite-related Missouri poison control center calls and emergency department visit data revealed that only 18% of snakebite-related emergency department visits had a corresponding call to the state poison control center.[25] Snake species were often self-reported, and the reliability of a person's ability to properly identify snake species was not determined in this study. A previous study determined laypersons could distinguish between a venomous and nonvenomous snake 81% of the time.[26] The 5-y time period for which data was collected may be insufficient to properly encompass trends in snakebites in Kentucky.

Reporting bias may affect the distribution and number of snakebites reported to KRPCC. Hospitals in certain areas may call the KRPCC to report bites more frequently than other hospitals. Individual providers who work in certain areas may also be more inclined to report snakebites to the KRPCC than others. Local resident knowledge of KRPCC's availability to assist with snakebites may also affect reporting to KRPCC.

Conclusions

This study demonstrated that crotaline envenomations continue to be a public health issue for residents of Kentucky during the summer months. Significant medical intervention is being performed for many of these Kentucky residents. Our data are in line with previous studies in regard to the demographics of crotaline envenomation victims. Despite numerous guidelines recommending against some therapies, they continue to be used in the state of Kentucky, particularly antibiotics. Emergency medical service personnel likely need additional training given the contraindicated therapies they are performing. More data are needed to fully characterize the full epidemiological impact and appropriateness of interventions being applied.

Author Contributions: Study concept and design (JB, JT, MH); acquisition of the data (JB, JT, CH, LK); analysis of the data (JB, JT, CH, MH); drafting of the manuscript (JB, JT, CH, MH); critical revision of the manuscript (JB, JT, CH, MH); approval of final manuscript (All).

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ORIGINAL RESEARCH

Evaluation of Environmental Conditions on Self-Selected Work and Heat Stress in Wildland Firefighting

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Introduction—The purpose of this study was to evaluate heat stress occurring in wildfire management activities with variable environmental conditions.

Methods—Direct observation and real-time wireless physiological monitoring allowed for weather and physiological metrics, including heart rate, core temperature (T_c), skin temperature, and physiological strain index (PSI), of male (n=193) and female (n=28) wildland firefighters (WLFFs) to be recorded during wildfire management activities. Accelerometry data were used to categorize intensity level of activity.

Results—Ambient temperature and relative humidity values were used to compute the heat index (HI; n=3891 h) and divided into quartiles (Q1: 13.3–25.1°C; Q2: 25.2–26.4°C; Q3: 26.5–28.9°C; Q4: 29.0–49.1°C). Activity levels remained relatively constant across all HI quartiles. The percentage of time spent performing moderate/vigorous activities was lowest during the hotter Q4 (Q1: 3%; Q2: 2%; Q3: 2%; Q4: 1%). Heart rate, T_c, PSI, and skin temperature associations with HI varied by resource type. Sixty-one percent of WLFFs (n=134) experienced a T_c ≥38.0°C, and 50% of WLFFs (n=111) experienced a PSI ≥6.0.

Conclusions—Heat stress was prevalent as WLFFs performed job tasks of varying intensities in all ambient conditions. Spontaneous bouts of arduous labor, duration of work shifts, and other occupation characteristics present the possibility for substantial durations of hyperthermia, although no heat-related injuries occurred in this study. Despite chronic exposure to rugged sloped terrain, load carriage, and environmental conditions, self-regulation and individual attention to managing work:rest appears to be the primary management strategy in mitigating excessive accumulation of body heat in this occupation.

Keywords: hyperthermia, physiological strain

Introduction

According to the National Institute for Occupational Safety and Health, heat stress is the net heat load to which an individual is exposed as a result of the combination of metabolic heat, environmental factors, and clothing worn, which results in an increase in heat storage in the body.[1] Heat stress can evoke heat strain, which is the physiological response to heat load.[1] For wildland firefighters (WLFFs), the metabolic generation of heat is inevitable as they hike over rough terrain, construct firelines, lay hose, operate chainsaws, and clear brush during wildfire management and training activities.[2] These wildfire management activities are often performed in hot environments, which increases the associated strain on heat loss mechanisms during these activities.[3] High values of water turnover (4.5–9.6 $L \cdot d^{-1}$) and caloric expenditure values ranging from 12.6 to 26.2 $MJ \cdot d^{-1}$ (3000–6260 $kcal \cdot d^{-1}$) are further evidence of the strenuous physical requirements of a typical 12 to 14 h workshift.[4-7]

Studies have varied in description of tasks performed in the occupation; accelerometry data have demonstrated that WLFFs spend 49±8%, 39±6%, and 12±2% of their workshifts in sedentary, light, and moderate-vigorous intensity categories, respectively.[8] A more recent classification of these tasks using heart rate (HR) data suggests

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that WLFFs spend 43, 9, 19, and 28% of their workshifts performing sedentary, light, moderate, and high-intensity physical activity.[9] Correspondingly, these intensity levels of physical activity have shown increases in core temperature (T_c) of 0.1, 0.2, and 0.4°C for light, moderate, and high-intensity tasks, respectively.[9]

Prior research has demonstrated that heat stress limits exercise performance[10,11] and that social and behavioral factors often influence the intensity and duration of bouts of activity that WLFFs endure.[12,13] Unfortunately, factors such as military or industrial discipline, commercial pressures, team effort, or personal motivation can deter a behavioral response to thermal stress.[14] Although research in simulated environments can provide insight into the contributing factors that influence physiological strain,[15-17] an emphasis on real-time documentation during wildfire suppression should become the primary driver of policy change because scripted laboratory or simulated data do not account for self-selected modifications to work rates in field settings.[18] Although prior research has evaluated real-time physiological stress in WLFFs, these previous studies were conducted on only 2 fires in Colorado and Washington.[7,8,19] The purpose of this study was to assess the real-time heat stress in WLFFs during wildfire management activities across variations in geographic and ambient weather conditions.

Methods

PARTICIPANTS

Participants were recruited on large wildfires in the western United States, where they typically work 14-d shifts. Participants were asked to volunteer to be observed for 1 work shift on varied days of their respective assignments. Before taking part in the study, participants signed a written, informed consent form approved by a University of Montana institutional review board.

To obtain a comprehensive sample of the WLFF population, we included both interagency hotshot crews (IHCs) and other resource types (type II crews; 46 and 54% of the total sample, respectively). On wildfire incidents, IHCs are a common type I, or typically more experienced, resource, usually composed of 20 to 22 crewmembers. In contrast, other resource types can range from 3 to 20 crewmembers, depending on their purpose.[20]

EXPERIMENTAL DESIGN

Field data were collected during the fire season (May to October) in the western United States from 2013 through 2016. Before data collection, the US Department of Agriculture-Forest Service's National Technology and Development Program trained a cohort of WLFFs in direct observation methodologies. Continuous observations of each participant permitted the research team to classify tasks and relate physiological responses to each specific task a participant performed using this methodology, as seen in prior studies.[9,21] Participants were allowed to eat and drink ad libitum throughout the day.

On the morning of each trial (≈0600), 2 or 3 participants from a WLFF resource type arrived at the field laboratory after an overnight fast and ingested an initialized wireless thermometer capsule (Jonah ingestible sensor, Mini Mitter, Bend, OR). Participants were fitted with a Hidalgo Equivital physiological monitor, which communicates with the thermometer capsule and records HR and skin temperature (T_{sk}). This device consists of a chest harness with shoulder straps worn around the chest to situate sensors inferior to the pectoral muscles on the anterior chest surface. Participants then ate breakfast before their workshift to promote T_c capsule progression into the digestive tract.

PHYSICAL MEASUREMENTS

The Hidalgo Equivital physiological monitor continuously monitored and recorded HR, T_c, and T_{sk} throughout the entire workshift using the previously mentioned methodology. Raw HR, T_c, and T_{sk} values were trimmed according to the CI associated with the Hidalgo physiological monitor. A threshold of 85% CI was used based on manufacturer recommendations and evaluation of raw data. The data above 85% CI consisted of values associated with population and age-adjusted normal values. Maximal HR (HR_{max}) was estimated using the equation $HR_{max}=208-0.7\times age$.[22]

An Atago 4410 PAL-10S digital pocket refractometer was used to measure urine specific gravity (USG). Before each shift, a first void urine sample was collected from each participant to provide a baseline USG. Before the evening meal, another void sample was obtained from each participant to measure post-shift USG in the fire camp.

Equipment weight was obtained using a calibrated digital scale (Taylor Precision Products, Model 7329B, Oakbrook, IL) set on a stable, level surface, and values were recorded in 0.1 lb increments and converted to kilograms. Bodyweight (including shirt, pants, and fire boots; approximate weight=4 kg) was subtracted from the total ensemble weight to calculate equipment weight. Equipment ensembles were independently verified throughout the workshift using photographs taken during direct observation of the participant and using aircraft manifest weight estimates of the equipment.[20] Participant

height was self-reported, and body surface area was calculated using the participant height and body weight in the following equation[23]:

$$BSA(m^2)=\sqrt{[(\text{height in cm}\times\text{weight in kg})/3600]}$$

CRITERIA FOR PHYSIOLOGICAL HEAT STRAIN

A T_c of 38.0°C was used as a threshold for increased risk of heat stress.[1] As an additional measure to evaluate conditions of heat stress, the physiological strain index (PSI) was used to quantify the heat strain experienced by each participant using the following equation[24]:

$$PSI=5\times(T_{c(t)}-T_{c(0)})\times(39.5-T_{c(0)})^{-1}+5\times(HR_{(t)}-HR_{(0)})\times(180-HR_{(0)})^{-1}$$

$T_{c(0)}$ and $HR_{(0)}$ were the initial T_c and HR measured at the start of the workshift. $T_{c(t)}$ and $HR_{(t)}$ were the T_c and HR values corresponding to a given time point. PSI[25,26] was based on T_c and HR values, a valid expression of thermoregulatory load. A PSI ≥6.0 is recognized as the onset of marginal risk of heat injury due to high heat stress levels from exercise and can be associated with a T_c of 38.3°C.[24-26]

ACTIVITY MONITORS

ActiCal activity monitors (MiniMitter, Bend, OR) were used to record activity counts (ACT). The monitors were initialized and distributed to participants to determine ACT during 1 d of firefighting. The activity monitor was placed in the left chest pocket of the Nomex fire shirt, similar to prior studies.[27] The pectoral location was chosen because of the amount of upper body movement associated with firefighting. For protection and stability, each monitor was secured in a cardboard square (~8×8 cm). ACT data (counts·min^{-1}) were divided into 3 intensity ranges: sedentary (0−99 counts·min^{-1}), light (100−1499 counts·min^{-1}), and moderate/vigorous (≥1500 counts·min^{-1}).[27,28]

AMBIENT TEMPERATURE AND RELATIVE HUMIDITY

An OMEGA temperature data logger (OMEGA Engineering, Inc., Stamford, CT) was attached to the shoulder strap on each participant's fireline pack to record ambient temperature and relative humidity (RH) at minute intervals throughout the workshift. Heat index (HI) values were calculated using temperature and RH inputs and adjustments using the method described in prior studies.[29] Temperature and RH values when the participant was riding in vehicles were removed from the data set, and the remaining data were then used to compute HI (n=141,785 min) and allocated into quartiles (Q1: 13.3−25.1°C; Q2: 25.2−26.4°C; Q3: 26.5−28.9°C; Q4: 29.0−49.1°C) to evaluate ambient conditions and their relationship with a measured physiological response.

STATISTICS

Data are presented as mean±SD or percentages to describe the distribution of time spent at various activity levels. Independent-sample t-tests were used to analyze differences in study variables between resource types and HI quartiles with SPSS statistics software (SPSS Inc., Chicago, IL). $P<0.05$ was considered significant. Levene's test for equality of variances was used to check assumptions of equal variance before inference procedures were used on the response variables.

Results

DESCRIPTIVE DATA

Male (n=193; 27±6 y; 179±9 cm; 86.0±11.8 kg) and female (n=28; 26±4 y; 167±9 cm; 68.6±8.4 kg) WLFFs participated in this study (see Table 1 for participant age, height, weight, body mass index, equipment weight, and body surface area [BSA][23]). Age, height, and BSA did not differ by resource type. IHCs carried more equipment weight than type II resources (difference of 2.3 kg; $P<0.05$), and females' equipment weight was a significantly larger percentage of body weight compared with males (females=27% of bodyweight; males=22%: $P<0.05$).

AMBIENT CONDITIONS

Observation days (n=167) consisted of wildfire management activities on 50 wildfire incidents. As such, the environmental conditions varied widely by geographic location and season. WLFFs in this study experienced ambient temperatures exceeding 30.4°C (≈87°F) in 20% of the data set. The highest observed ambient temperature was 50.3°C (≈123°F), and 30% of WLFFs in the study experienced a maximal ambient temperature higher than 37.7°C (≈100°F) while assigned to a wildfire incident. In WLFF communities, 1400 is a critical time point for changes in fire behavior. Wildland fire management activities thus occur in preparation for this time of day but can also change at this time of day as well, dictated by the fire's behavior. The distribution by state of mean ambient temperature and RH values at 1400 is reported in Figure 1. The mean ambient temperature and RH recorded at 1400 was 29.5±6.5°C (≈85°F) and 28±15%, respectively.

Table 1. Participant descriptive data (mean±SD)

Sex	*Resource type*	*n*	*Age (y)*	*Height (cm)*	*Weight*[a] *(kg)*	*BMI (kg·m^{-2})*	*Equipment weight*[b] *(kg)*	*BSA (m^2)*
All	All	221	27±6	177±10	84.8±12.5	26.7±3.9	18.7±3.7	2.03±0.19
	IHC	101	28±6	179±10	81.6±11.0	25.9±2.8	20.0±3.2	2.00±0.17
	Type II	120	27±6	177±10	85.6±14.0[c]	27.4±4.5	17.7±3.7[c]	2.05±0.21
Male	All	193	27±6	179±9	86.0±11.8	27.0±3.8	18.7±3.7	2.06±0.17
	IHC	90	28±6	179±9	83.5±9.8	26.2±2.8	20.1±3.1	2.03±0.15
	Type II	103	27±7	179±9	88.2±13.0	27.6±4.5	17.7±3.8	2.09±0.18
Female	All	28	26±4[d]	167±9[d]	68.6±8.4[d]	24.8±3.5[d]	18.2±3.6	1.78±0.13[d]
	IHC	11	26±4	167±10	66.0±6.4	23.8±1.7	19.8±4.0	1.75±0.13
	Type II	17	26±4	166±8	70.3±9.2	25.6±4.2	16.7±2.3	1.80±0.13

BMI, body mass index; BSA, body surface area; IHC, interagency hotshot crew.
[a]Dressed weight − body weight while wearing fire boots, Nomex pants, and a t-shirt.
[b]Dressed weight subtracted from total ensemble weight (dressed weight and all necessary fireline equipment including pack and tools).
[c]Indicates difference compared with IHC (P<0.05).
[d]Indicates difference compared with males (P<0.05).

ACTIVITY COUNTS

Participants in the study spent 67, 61, 62, and 64% of their time performing sedentary activities (<100 counts·min^{-1}) in Q1, Q2, Q3, and Q4, respectively. The percentage of time spent carrying out light-intensity activities increased slightly from Q1 to Q2, Q3, and Q4 (Figure 2). Time spent executing moderate and vigorous activities decreased by 67% from Q1 to Q4 (3% to 1%; P<0.05).

When considering overall activity (all levels of activity including sedentary; n=49,871 min), activity levels for IHCs were highest in Q2 and Q3, with lower activity counts observed in Q1 and Q4 (Figure 3). For type II resources, Q1, Q2, and Q3 activity values were higher than Q4 (P<0.05). When evaluating physical activity only (ACT>100 counts·min^{-1}; n=30,957 min), Q1, Q2, and Q3 activity values were all higher than Q4 (P<0.05). Overall activity for Type II resources (n=31,672 min) displayed a negative correlation between ACT and HI quartiles, although only Q1 and Q2 activity values were higher than Q4 (P<0.05). When evaluating physical activity only (ACT >100 counts·min^{-1}; n=19,580), only Q1 and Q2 activity values were higher than Q4 (P<0.05). Resource type differences existed only for Q1 and Q3 (P<0.05; Figure 3).

THERMOREGULATORY VARIABLES

Mean peak HR values for all subjects was 171±25 beats·min^{-1}. When considering physical activity alone, mean HR values for IHCs displayed a positive correlation with HI quartiles (108±24, 114±23, 115±23, and 119±23 beats·min^{-1} for Q1, Q2, Q3, and Q4, respectively.) Q1, Q2, and Q3 IHC mean HR values were significantly less than Q4 HR values (P<0.05). Conversely, mean HR values for type II resources for Q1, Q2, and Q3 were all significantly higher than Q4 HR values (115±26, 112±23, 111±22, 108±19 beats·min^{-1} for Q1, Q2, Q3, and Q4, respectively; P<0.05). Mean HR values were significantly different between resource types at all HI quartiles (P<0.05).

T_c and PSI values are reported in Figure 4. Briefly, T_c showed a positive correlation with HI quartiles for IHCs, with mean IHC T_c for Q4 being significantly greater than for all other quartiles (P<0.05). Type II resources displayed a weaker association between T_c and HI quartiles, where mean T_c for only Q1 and Q3 was different from Q4 values (P<0.05). IHC mean PSI values for Q1, Q2, and Q3 were less than those in Q4 (P<0.05), whereas only the mean PSI values for Q2 were different from Q4 values for Type II resources (P<0.05). All quartile values of T_c and PSI were significantly different between resource types except for the T_c values of Q2 (P<0.05).

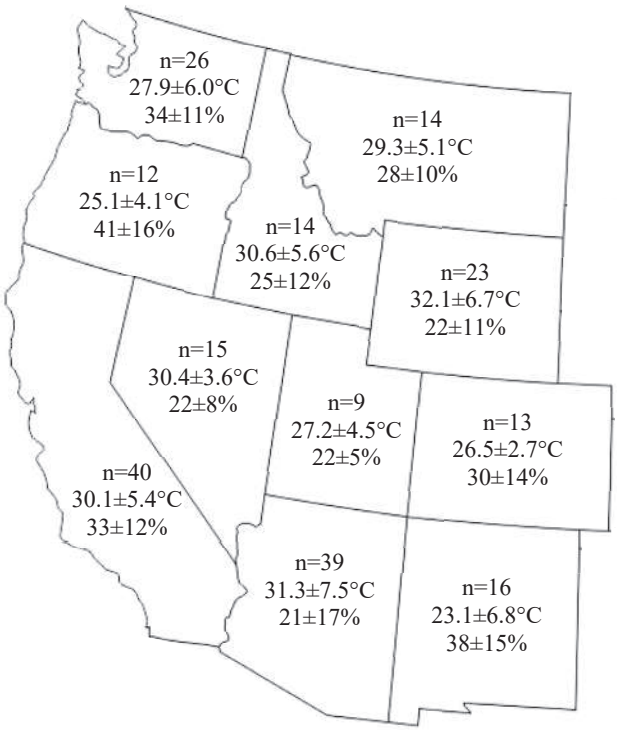

Figure 1. Map of the western continental United States with the number of study participants, mean ambient temperature, and relative humidity from data collection sites in each state at 1400. In wildland firefighting communities, a critical time point for changes in fire behavior is 1400. Wildland fire management activities thus occur in preparation for this time of day but can also change at this time of day as well, dictated by the fire's behavior.

Nearly 61% of WLFFs (n=134) experienced a T_c ≥38.0°C; 93 of these individuals experienced this elevated T_c for >30 min (average frequency=97 min). Comparatively, 50% of WLFFs (n=111) experienced a PSI ≥6.0; 37 participants experienced this elevated PSI for >30 min.

IHC T_{sk} in Q4 was significantly higher than in all other quartiles (31.7±1.9, 31.9±2.0, 32.2±2.1, and 33.1±2.0°C for Q1, Q2, Q3, and Q4, respectively; $P<0.05$). Type II resource T_{sk} in Q4 was also significantly higher than in all other quartiles (31.9±1.9, 32.3±2.0, 32.8±1.8, and 33.3±2.0°C for Q1, Q2, Q3, and Q4, respectively; $P<0.05$). Type II T_{sk} values were higher than IHC values across all HI quartiles ($P<0.05$).

HYDRATION STATUS

The range of first void USG values (mean=1.017±0.007; Figure 5) displays the prevalence of a wide variation in estimated hydration status among WLFFs before their respective workshifts. Similar post-shift values (1.015±0.008) were observed.

Discussion

This study establishes that WLFFs typically operate at T_c values elevated above resting values throughout the workshift in a wide array of ambient conditions. Twenty percent of the ambient conditions in this study correspond with moderate- to very high-risk conditions for heat stress, according to industrial and military guidelines.[30] Despite these conditions, mean T_c and T_{sk} values displayed only a mild association with ambient conditions. When accompanied by the abnormal performance of 1 or more organ systems, a T_c ≥38.0°C is defined by the National Institute for Occupational Safety and Health as heat exhaustion.[1] Of note, nearly two-thirds of participants (61%) experienced a T_c ≥38.0°C, whereas a lower percentage (50%) experienced a PSI ≥6.0, which can be associated with a T_c of 38.3°C.[24-26] This difference is likely due to the impact of low heart rates in the PSI calculation that may minimize the estimated heat strain. Although these individuals did not report symptoms or incidence of heat-related injury, hyperthermia prevalence and duration are significant. Furthermore, this study established that although moderate and arduous activity levels are tempered during elevated HI, activity is performed in all ambient conditions.

The occupation of wildland firefighting presents a unique and diverse set of challenges for individuals from a physiological perspective. As evidenced in Figure 1, WLFFs can expect summer campaigns of 14-d assignments, each with a unique environmental profile. Furthermore, WLFFs demonstrate a wide array of USG values, suggesting large variations in estimated pre-shift hydration status. Despite significant water turnover values documented in prior studies, USG values were comparable between pre-shift and post-shift collection points.[4,5,12,19] However, it is essential to acknowledge that 17% of WLFFs began and ended the workshift with a USG >1.025 (Figure 5). Although a single measure of USG, particularly a first void measurement, does not clearly demonstrate dehydration, this array of values supports the promotion of diligent hydration practices throughout the day in this population.

Accelerometry data demonstrated that the percentage of time spent in sedentary, light, and moderate/vigorous intensity activity remained relatively constant across all ambient conditions. Because no evidence of heat-related injuries was found in the study, these results suggest a propensity toward self-regulation of physical activity to

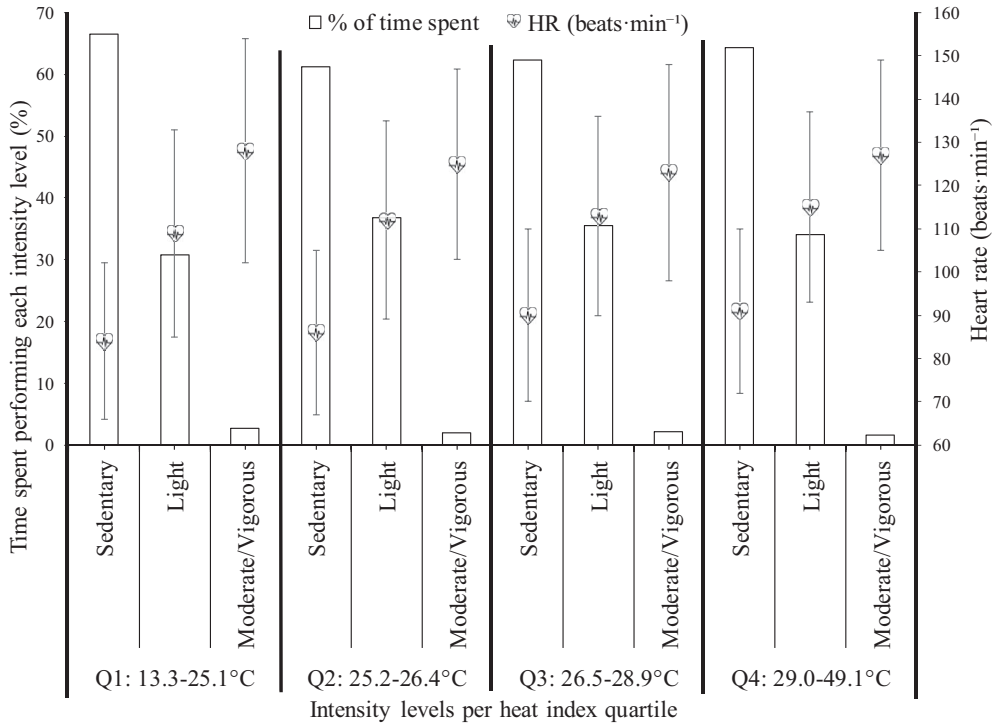

Figure 2. Combination bar and line graph displaying the distribution of time spent at each intensity level across all heat index quartiles and the associated heart rate at each intensity.

manage body temperature in most WLFF field operations, similar to conclusions in Australian WLFF populations.[18] Key principles that have been previously suggested to mitigate heat strain during fire suppression activities include avoiding unnecessary heat and encouraging self-pacing, drinking fluids, and allowing sweat to evaporate.[18]

The activity level and equipment weight both contributed to conditions of physiological strain that can be self-regulated. Of specific interest in this population, muscle work results in body heat, with more than 70% of metabolic energy converted to heat during physical activity.[31-35] The demand of job tasks performed on the fireline is likely further influenced by the weight of equipment carried (18.7±3.7 kg for all resource types; Table 1) and hot temperatures associated with wildfire conditions. Supported by the HR and T_c values observed in this study, regulating the duration of high-intensity activities, management of work:rest guidelines during operations, and promoting aerobic fitness in this population should be emphasized. Interestingly, HR displayed a negative correlation with HI for Type II resources in the study, whereas HR showed a positive correlation with HI for IHCs. Differences in WLFF resource utilization likely influence these results; the different resource types may receive various tasks in response to higher ambient conditions and fire activity. Although this study observed a decrease in the percentage of time spent performing moderate and vigorous activity from Q1 to Q4 (67% reduction in accelerometer data), it is essential to note that increased ambient conditions may result in increased fire activity, which can impede wildfire management activities. This concept and its effect on WLFF tactics may contribute to managing physiological strain in these individuals.

As in prior WLFF studies,[2] hiking was a critical metabolic contributor to heat loading in these tactical athletes (Figure 6). The initial hike into the fire or work unit generally raises an individual's body temperature significantly and may result in elevated body temperature for several hours afterward.[8,9,22] Because these hikes occur before the physical labor portion of the workshift, the crew transports their equipment for the entire shift during this hike. This initial hike (typically 20−60 min in duration) also often occurs during a part of the day (usually between 0730 and 1100) when ambient temperatures are rising, and RH may not have decreased to respective afternoon values (21.9±6.2°C and 41±15%, respectively).

Mitigation strategies for WLFFs should include reducing equipment weights for resources working in hot

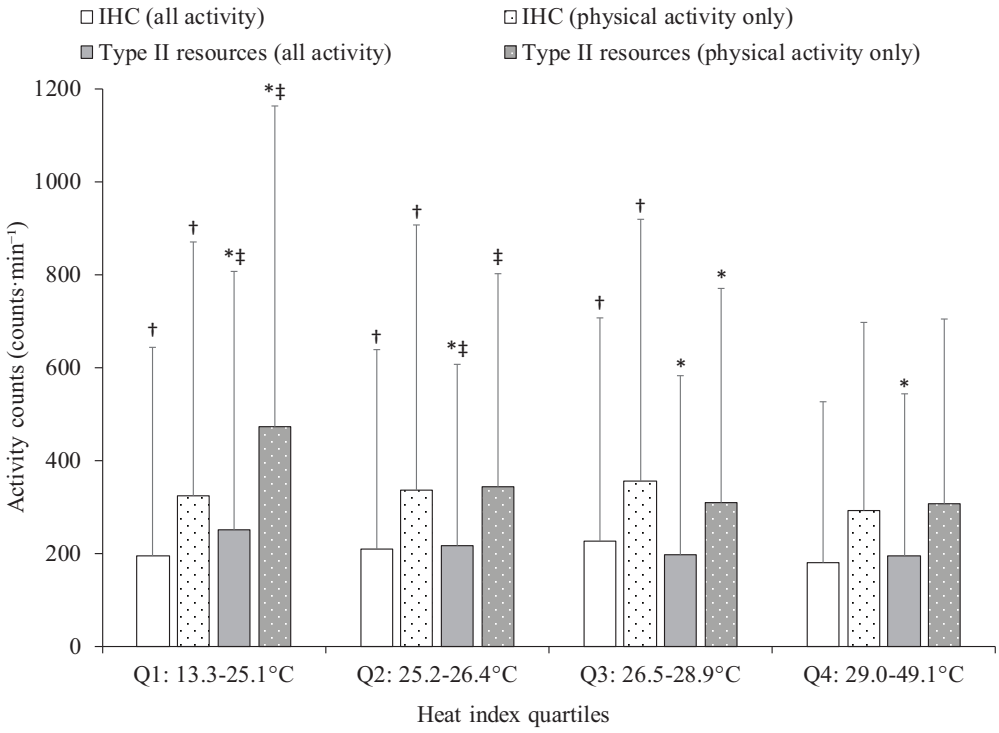

Figure 3. Bar graph displaying the mean activity counts for both resource types in each heat index quartile. Activity was divided into all activities and physical activity only, defined as activity counts exceeding 100 counts·min^{-1}. * - indicates a difference from IHC (P<0.05); † - indicates difference from Q4 of IHC data (P<0.05); ‡ - indicates difference from Q4 of type II data (P<0.05).

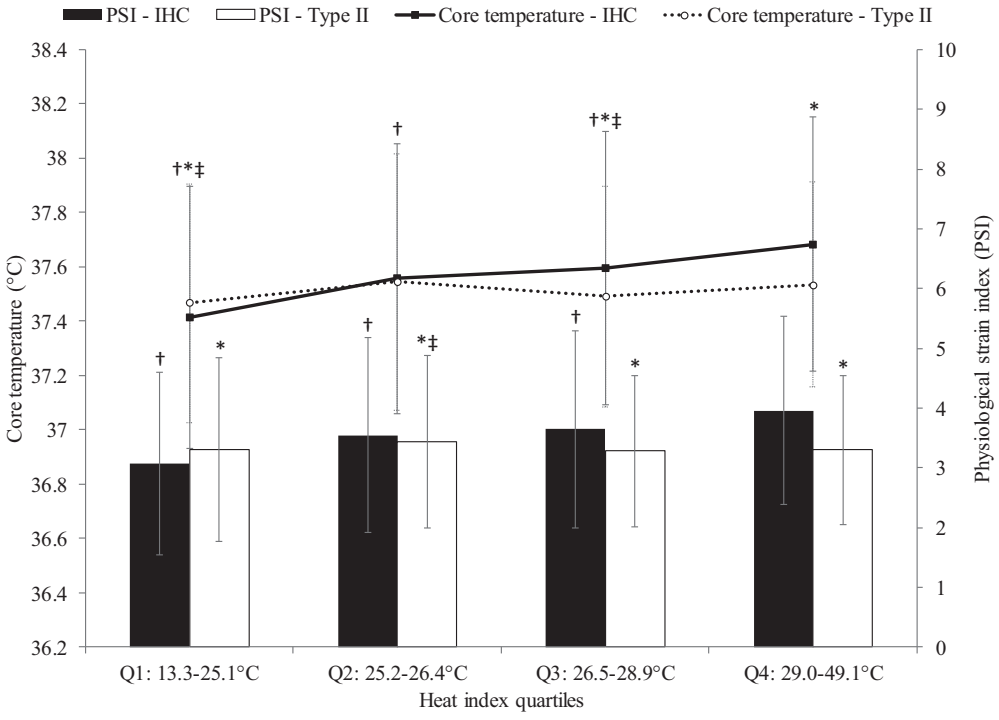

Figure 4. Combination bar and line graph displaying the core temperature and physiological strain index values for each resource type in each heat index quartile. * - indicates a difference from IHC (P<0.05); † - indicates difference from Q4 of IHC data (P<0.05); ‡ - indicates difference from Q4 of type II data (P<0.05).

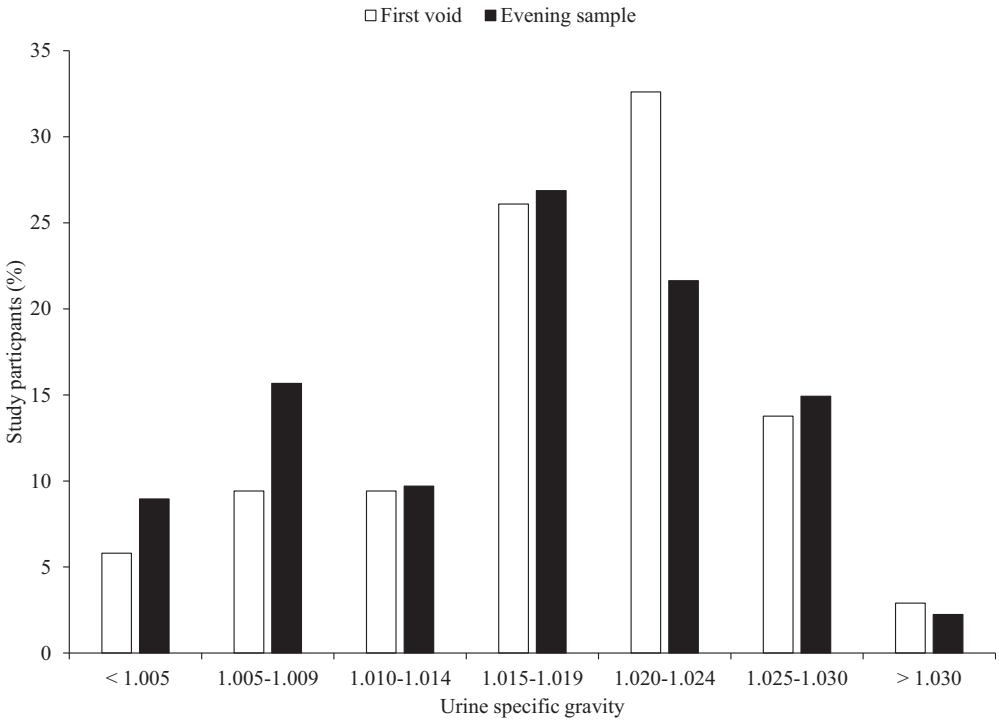

Figure 5. Graph showing the distribution of first void and post-shift urine specific gravity measurements collected in the study.

temperatures; equipment weights over 25% of an individual's bodyweight significantly increase the demand of all activities performed.[36] Fitter males have been shown to have a substantially lower risk for exertional heat illness during activity.[37,38] As such, to better prepare individuals for the demands of wildfire management and training activities, preparation should emphasize developing aerobic capacity through aerobic training and heat

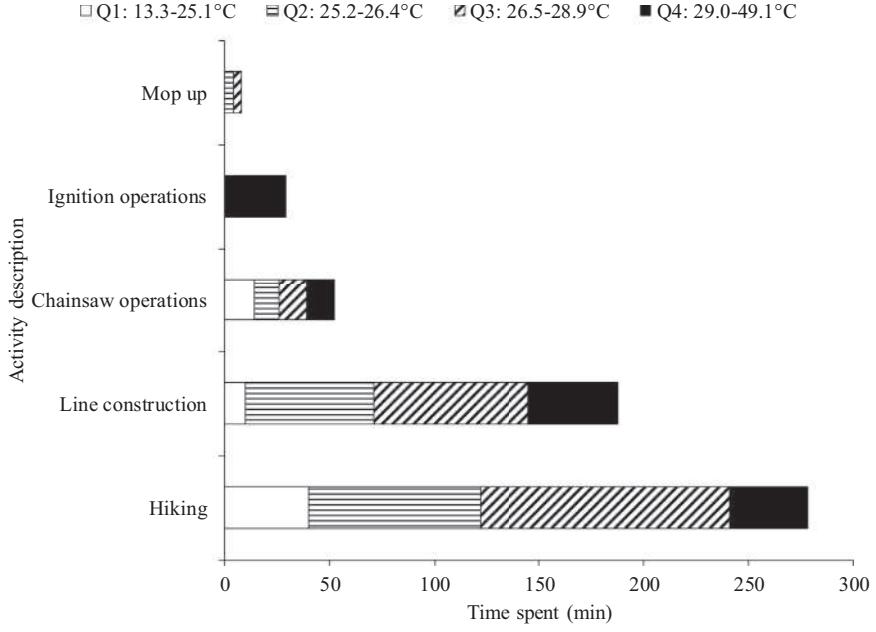

Figure 6. Stacked bar graph showing the frequency of everyday wildland firefighter job tasks that occurred in conditions in which physiological strain index exceeded 6.0. Each bar graph shows the frequency of each activity in each heat index quartile.

acclimation strategies, such as light to moderate exercise in hot climates, with adequate rest in colder conditions afterward.[39] These training programs should also implement increasing aerobic capacity and pacing strategies, such as work:rest, for the crew as a whole and individual members to prepare for the long, chronic effects of fire season activities.[8,9,22,37] Moreover, these factors should be emphasized at a higher priority than aggressive fluid delivery strategies; prior research has documented heat-related injury in a WLFF despite significant water consumption.[8] Recent research has also reported that the mean WLFF breakfast and shift provision intake provides adequate electrolyte replacement, without the use of supplements, for most workshifts.[40]

LIMITATIONS

Data collected for this study were from different WLFFs during different seasons. Participants were not selected at random; they were recruited from available resources in the fire camps and asked to volunteer for the study. There is a lack of female representation in this occupation,[2] and our results do not address sex differences for multiple variables. No preliminary testing of the participants was conducted because of each participating crew's location across the Western United States. As stated in the Methods, participants typically had at least 1 d of work before participation, and the number of days worked by WLFFs before participation varied. The nature of the field research did not allow us to control the number of days previously worked. As such, these data do not consider the effects of previously worked shifts on the observed physiological response.

WLFFs often have different daily assignments while on an incident for multiple weeks. Typical workdays can include 10 to 30 or more activities (eg, hiking, digging, briefing, rest) per workshift. After the morning ingress hike, which the crew performs together, tasks/activities are distributed among crewmembers to achieve the operational assignment for that workshift. Assignment distribution changes the total daily energy expenditure across crewmembers and may reduce some crewmembers' higher intensity workload relative to others.

Because food and water consumption was ad libitum and not recorded, caloric ingestion and hydration status variations may affect thermoregulation and activity attributes. Furthermore, food and water consumption may have affected T_c readings during the workshift. There is also the potential for error in the Hidalgo unit's infrared T_{sk} sensor because the shoulder straps and other equipment donned by WLFFs may interfere or interact with the placement and reading of the variable.

Conclusions

WLFFs performed job tasks of varying intensities in all ambient conditions, with significant portions of the workshift at elevated heat strain risk. Most of the activity occurring during a workshift can be characterized as sedentary or light intensity. However, spontaneous bouts of arduous labor, duration of work shifts, and other occupation characteristics present the possibility for a substantial duration of hyperthermia. Despite these demanding work conditions, no heat-related injuries occurred in this study. Variations in physiologic variables between resource types are likely due to latent variables associated with occupational assignments and leadership. Despite chronic exposure to rugged sloped terrain, load carriage, and environmental conditions, self-regulation and individual attention to managing work:rest appear to be the primary strategy in mitigating excessive accumulation of body heat in this occupation.

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ORIGINAL RESEARCH

Indoor Bouldering—A Prospective Injury Evaluation

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Introduction—Bouldering has become a sport of growing interest, but little prospective evidence exists about injury proportions and patterns. The purpose of this study was to prospectively evaluate the cause of injuries sustained during indoor bouldering, proportion of affected body location, and injury severity.

Methods—Proportions and patterns of injury among German-speaking indoor boulderers were evaluated prospectively in an explorative cohort study. Participants completed a baseline questionnaire assessing anthropometric data and sport-specific potential preventive and risk factors, followed by monthly injury questionnaires including injury location and injury severity over a period of 12 mo.

Results—Out of 507 boulderers, 222 (44%) sustained 305 injuries. Of those, 78% (n=238) were classified as Union Internationale de Associations d'Alpinisme (UIAA) 1, 19% (n=57) as UIAA 2, and 3% (n=10) as UIAA 3. Injuries of the upper extremities accounted for 63% (n=191) of all injuries. Injuries of the lower extremities accounted for 23% (n=71) but were more often classified as UIAA≥2 (P=0.0071; odds ratio [OR] 2.23; 95% CI 1.23–4.04) and were more often caused by falling (P=0.0005; OR 2.92; 95% CI 1.57–5.42) and jumping off the wall (P<0.0001; OR 4.39; 95% CI 2.25–8.56) than injuries of other body locations. There was no statistically significant protective effect of the evaluated potential preventive measures. Participants who used heavily downturned climbing shoes had a higher risk of sustaining a UIAA ≥2 injury (P=0.0034; OR 2.58; 95% CI 1.34–4.95).

Conclusions—Injuries in indoor bouldering are common. Lower extremity injuries are associated with higher injury severity. Preventive measures need to be established to reduce bouldering injuries, especially during falls and landings.

Keywords: rock climbing, sports injuries, epidemiology, risk factors, injury severity

Introduction

In its origins, bouldering was a method of training for alpine expeditions and summits. In the 20th century, bouldering evolved to an individual climbing subdiscipline, with written bouldering guidebooks and equipment. Regulation to conserve nature in popular bouldering areas became necessary, such as regulated parking, mandatory trails to the boulders, and protections zones where bouldering is prohibited. Around the turn of the millennium, a climbing boom started that led to the appearance of indoor climbing gyms, often equipped with small bouldering rooms, in almost every major city in Germany. In recent years, more and more standalone indoor bouldering gyms have appeared in a vast number of German cities, leading to a considerable increase in active sports boulderers.[1] With more people practicing bouldering, an increase in bouldering-related injuries is to be expected.[1-5]

Bouldering can be defined as rock climbing without the use of a rope and harness on rock or artificial climbing walls of lower height.[6] The goal in bouldering is to solve a boulder problem by a sequence of movements, which usually have to be practiced several times until the athlete is able to put them together. A boulder problem consists of predefined holds that help to reach a defined target hold.[2] The majority of boulder problems have a height of 2.5 to 5 m, and to reduce the risk of injury from

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falls from this height, it is common to use composite foam safety mats, either portable for outdoor utilization or permanently installed indoors.[2,7] Most boulderers use special climbing shoes to enable them to stand on small edges and use chalk to absorb moisture and prevent sweaty hands.[8-11] Climbing shoes have a special design and feature a varying degree of downturn (ie, a concavely shaped sole that places more pressure on the toes, which then are forced into a flexed position) and an asymmetric design that maximizes pressure on the hallux.[8,10,12]

There are many similarities between sport climbing and bouldering, but there are also some differences, which could affect injury pattern and prevalence.[2] Because of the low height of artificial indoor bouldering walls, bouldering problems tend to consist of fewer, but more difficult and strenuous, moves than comparable sport climbing routes.[2] Those moves are repeatedly practiced, which can lead to an increased number of overuse syndromes. In sport climbing, most falls are stopped by the rope and falls to the ground are rare. In bouldering, it is much more common to repeatedly fall to the ground or, in most cases, onto the safety mat on the ground; this is called a ground fall and can result in different injury patterns and possibly a different injury rate, especially of the lower extremities.[1,2,12] Additionally, injuries due to a collision with other falling athletes are also possible. Spotting, a boulder-specific safety measure, is practiced by bystanders, who try to redirect uncontrolled falls of the climber to enable safe landing, usually by rotating the falling climber into an upright position. This is typically done by grabbing the falling boulderer by the hips with both hands to slow their fall, enable landing on their feet, and direct them to a safer landing zone. This is to prevent injuries in general and in particular of the head, neck, and back. Because of these differences, the literature on rock climbing injuries cannot be unreservedly transferred to bouldering.[2,12]

The purpose of this study was to prospectively evaluate the cause of injuries sustained during indoor bouldering, the proportion of affected body location, and injury severity. Furthermore, the influence of potential preventive measures and risk factors on the injury rate was investigated.

Methods

STUDY DESIGN AND POPULATION

This was a prospective explorative cohort study in German-speaking boulderers. The study protocol was approved by the ethical review board of the Medical University of Würzburg.

Study participants were recruited between October 2017 and March 2018 via online media, print media, and in cooperation with bouldering gyms. The internet address to register for this study was published online and shared on Facebook by 11 German bouldering gyms and multiple private persons. In addition, the registration link was printed on flyers and distributed to 34 German bouldering gyms, and the survey was announced in a German climbing magazine.

Participants completed an online baseline questionnaire on anthropometric data, training habits, and potential preventive measures and risk factors. Follow-up questionnaires were sent monthly to the individual email address of each participant over the study period of 12 mo.

Inclusion criteria were an age of 18 y or older and activity in an indoor bouldering gym on a regular basis (ie, no one-time boulderers) during the study period. The exclusion criterion was failure to answer the questionnaire in the 12th month. Participants who did not answer emails in earlier months were not excluded, but we controlled for injuries sustained in the missing months in the following questionnaire.

QUESTIONNAIRE DEVELOPMENT

The questionnaire was adapted from existing literature on rock climbing and sports injuries.[2-4,8,13-17] The questionnaire was developed and refined by clinical physicians in cooperation with active bouldering athletes. It was tested by active boulderers and revised based on their suggestions received until a consensus was found. A great value was set on comprehensibility for medical nonprofessionals and low time investment to accomplish high study participation.

MEASUREMENTS AND OUTCOME MEASURES

The baseline questionnaire addressed anthropometric and sport-specific data, including sex, height, and weight; details on climbing shoes (size in comparison to the everyday shoe and degree of downturn, categorized as none, slight, medium, or heavy downturn); average weekly bouldering time; bouldering experience; and history of injury ("was there ever an injury that stopped you from training or doing your job for more than 24 h?", "was there ever an injury that stopped you from training or doing your job for more than 10 d?"). Boulder-specific data such as duration of warm up (0 min, 0–10 min, 10–20 min, 20–30 min, or >30 min), spotting (not used, just used during risky moves, or used most of the time), and average weekly time practicing outdoor bouldering, indoor sport climbing, and outdoor sport climbing were assessed. Ultimately, the use/practice of cardio sports,

taping of the fingers, taping of other locations, slacklining or balance training, yoga, stretching, training with body weight or weights, warming up by doing easy boulder projects, warming up of fingers, campus board, pegboard, fingerboard, or grip trainers were addressed (yes or no).[2,16]

The monthly follow-up questionnaires assessed injuries sustained during bouldering in the past month. There were 3 possibilities: 1) occurrence of an injury, 2) no occurrence of an injury, and 3) still injured. Injuries were only included in this study if they led to an absence from work or training for more than 24 h; this was done to avoid reports of minimal trauma such as small abrasions or aching muscles. Body location (19 categories: head/face, neck/cervical spine, shoulder/clavicle, upper arm, elbow, forearm, wrist, hand/finger/thumb, chest, thoracic spine, trunk/abdomen, lumbar spine, pelvis/buttock, hip/groin, thigh, knee, lower leg, ankle, and foot/toe), severity (Union Internationale de Associations d'Alpinisme [UIAA] 1: mild injury or illness, no medical intervention necessary; 2: moderately severe injury or illness, not life threatening, outpatient therapy, heals without permanent damage; 3: major injury, not life threatening, hospitalization, surgical intervention, heals with or without permanent damage; 4: acute mortal danger, polytrauma, alive with permanent damage; 5: acute mortal danger, polytrauma, death; 6: immediate death), and cause of injury (bouldering, falling, jumping, training/stretching, caused by another boulderer, no acute trauma) were evaluated and classified using the Medical Commission of the UIAA MedCom Score.[18]

The primary outcome measure was the number of injuries caused by indoor bouldering during the study period, and the secondary outcome measure was injury severity.

STATISTICAL ANALYSIS

Descriptive statistics are presented as mean±SD and categorical data as frequency counts (percentages). As statistical tools, the χ^2 test was used for nominally scaled data and Fisher's exact test to account for small sample size. The Kolmogorov-Smirnov test was used to test for standard distribution. The Mann-Whitney U test was used for ordinal or interval scaled data without standard distribution and logistic regression for categorical and continuous variables. Univariable logistic regression was used to calculate odds ratio (OR) and corresponding 95% CI. Multivariable logistic regression analysis was then performed on statistically significant independent variables to explore potential associations between potential preventive measures and risk factors and to calculate the adjusted OR and CI. $P<0.05$ was considered statistically significant. All analyses were performed using IBM SPSS statistics, version 22.0.

Table 1. Self-reported anthropometric data of indoor boulderers

	Male (n=285, 56%)	*Female (n=221, 44%)*	*Total (n=506, 100%)*
	Mean±SD	*Mean±SD*	*Mean±SD*
Age (y)	31±8	29±7	30±8
Height (cm)	181±7	169±6	175±9
Weight (kg)	75.2±10.7	61.0±7.2	69.0±10.8
BMI ($kg \cdot m^{-2}$)	23.1±2.9	21.4±2.2	22.4±2.7

Results

ENROLLMENT AND POPULATION CHARACTERISTICS

A total of 697 participants completed the initial questionnaire. Of those, 17 were excluded because of incorrect contact data (nonexistent email addresses, n=14) and withdrawal of consent (n=3). The study period was completed by 507 participants, resulting in a drop-out rate of 25%. All 12 questionnaires were completed by 214 participants; the number of questionnaires completed was 10.5±1.9.

The study population consisted of 285 (56%) males, 221 (44%) females, and 1 unknown sex (0.2%). Men were slightly older (P=0.0089; OR 1.03; 95% CI 1.01–1.06) and had a higher self-reported body mass index (P<0.0001; OR 1.46; 95% CI 1.32–1.61) than women (Table 1). The majority of all participants (59%; n=297) had sustained at least 1 injury before the study period that had caused more than 1 d absence from training or work; 55% (n=277) had an injury with a time loss of more than 10 d. A minority of 2% (n=8) practiced spotting most of the time, spotting during hard moves only was practiced by 53% (n=271), and 45% (n=228) never practiced spotting. Climbing shoes of a smaller size than regular shoes were used by 76% (n=385) of participants, 9% (n=46) used larger shoes, and 15% (n=73) used equally sized shoes. Heavily downturned climbing shoes were used by 12% (n=62), 63% (n=320) used moderately or slightly downturned shoes, and 25% (n=125) used climbing shoes without any downturn. The majority of participants (n=292, 58%) reported an average weekly boulder time of 2 to 5 h. In the study population, 22% (n=113) of boulderers had been bouldering regularly for more than 5 y and 23% (n=118) had been bouldering regularly for less than 1 y.

Table 2. Number of injuries during the study period by injury, location, and severity

Body location of injury	*Injury severity*			*Total injuries n (%)*
	UIAA 1 n (%)	*UIAA 2 n (%)*	*UIAA 3 n (%)*	
Head/Face	0	1 (0.3)	1 (0.3)	2 (0.7)
Neck/Cervical spine	5 (2)	4 (1)	0	9 (3)
Shoulder/Clavicle	36 (12)	10 (3)	2 (0.7)	48 (16)
Upper arm	12 (4)	0	0	12 (4)
Elbow	17 (6)	2 (0.7)	2 (0.7)	21 (7)
Forearm	7 (2)	2 (0.7)	0	9 (3)
Wrist	12 (4)	3 (1)	0	15 (5)
Hand/Finger/Thumb	77 (25)	9 (3)	0	86 (28)
Chest	7 (2)	0	0	7 (2)
Thoracic spine	1 (0.3)	1 (0.3)	0	2 (0.7)
Trunk/Abdomen	3 (1)	0	0	3 (1)
Lumbar spine	9 (3)	5 (2)	0	14 (5)
Pelvis/Buttock	2 (0.7)	1 (0.3)	0	3 (1)
Hip/Groin	1 (0.3)	0	0	1 (0.3)
Thigh	6 (2)	0	1 (0.3)	7 (2)
Knee	12 (4)	9 (3)	4 (1)	25 (8)
Lower leg	3 (1)	2 (0.7)	0	5 (2)
Ankle	15 (5)	7 (2)	0	22 (7)
Foot/Toe	10 (3)	1 (0.3)	0	11 (4)
Location unspecified	3 (1)	0	0	3 (1)
Total	238 (78)	57 (19)	10 (3)	305 (100)

INJURY PROPORTION AND LOCATION

During the study period, 222 (44%) of the 507 participants sustained a total of 305 injuries. The highest individual number of injuries sustained during the study period was 5. The majority of injuries were classified UIAA 1 (78%; n=238), 19% (n=57) were UIAA 2, and 3% (n=10) were UIAA 3 (Table 2). There was no injury graded higher than UIAA 3.

The majority of injuries affected the upper limbs (shoulder/clavicle, upper arm, elbow, forearm, wrist, and hand/finger/thumb: n=191; 63%). However, injuries in that location were less often classified UIAA ≥2 than injuries in the other main location groups (P=0.0004; OR 0.37; 95% CI 0.21–0.65). The most common injury location was fingers, hand, and thumb with 28% (n=86) of all injuries, followed by shoulder and clavicle with 16% (n=48). Injuries of the lower limbs (hip/groin, thigh, knee, lower leg, ankle, and foot/toe) accounted for 23% (n=71) of all injuries and were significantly more often classified UIAA ≥2 than injuries of other locations (P=0.0071; OR 2.23; 95% CI 1.23–4.04).

The most common cause of acute injury was bouldering itself, such as performing a strenuous move, followed by falling and jumping off the wall (Table 3). Lower limb injuries were more often sustained due to falling (P=0.0005; OR 2.92; 95% CI 1.57–5.42) and jumping off the wall (P<0.0001; OR 9.33; 95% CI 3.44–25.37) compared to injuries of other body locations. Injuries of the upper limbs happened more often without acute trauma (P<0.0001; OR 4.39; 95% CI 2.25–8.56), and injuries of the ankle were more often caused by jumping off the wall (P<0.0001; OR 17.12; 95% CI 6.04–48.53) than injuries of other locations. Nine of the 22 total injuries that occurred in the ankle region were caused by jumping. Just 1 (0.3%) injury was caused by another boulderer, who fell on the shoulder of a resting study participant.

PREVENTIVE MEASURES AND RISK FACTORS

According to our findings, there was no significant difference in age (P=0.48; OR 0.99; 95% CI 0.97–1.02), size (P=0.88; OR 1.00; 95% CI 0.98–1.02), body weight (P=0.85; OR 1.01; 95% CI 0.99–1.02), or body mass index (P=0.88; OR 1.01; 95% CI 0.93–1.08) between participants who were affected by an injury and those who were not.

There was no protective effect of preventive measures or specific training for climbing (Tables 4 and 5). Warming up fingers, taping locations other than the fingers, and using a fingerboard seemed to affect the risk of sustaining an injury in a negative way. To investigate a possible explanation for these results, logistic regression

Table 3. Number of injuries sustained during the study period by location (sorted by main group) and cause of injury

	Head and neck n (%)	*Upper limbs n (%)*	*Trunk n (%)*	*Lower limbs n (%)*	*Location unspecified n (%)*	*Total n (%)*
Bouldering	1 (0.3)	55 (18)	8 (3)	19 (6)	0	83 (27)
Falling	6 (2)	23 (8)	4 (1)	23 (8)	0	56 (18)
Caused by another boulderer	0	1 (0.3)	0	0	0	1 (0.3)
Training/Stretching	0	3 (1)	2 (0.7)	1 (0.3)	0	6 (2)
No acute trauma	1 (0.3)	65 (21)	3 (1)	8 (3)	0	77 (25)
Jumping	1 (0.3)	3 (1)	2 (0.7)	14 (5)	0	20 (7)
Other	0	3 (1)	2 (0.7)	1 (0.3)	0	6 (2)
No information	2 (0.7)	38 (12)	8 (3)	5 (2)	3 (1)	56 (18)
Total	11 (4)	191 (63)	29 (10)	71 (23)	3 (1)	305 (100)

adjusting for potential confounders was used. Adjusting for bouldering experience, history of injury, and age showed no influence on warming up the fingers, but taping locations other than the fingers and using a fingerboard was no longer significant. There was no significant difference in the risk of sustaining a UIAA ≥2 injury between users and nonusers of preventive measures and specific training.

Spotting behavior (P=0.96; OR 0.99; 95% CI 0.71–1.38), duration of a typical warm up (P=0.97; OR 1.00; 95% CI 0.90–1.12), activity in other climbing sports (P=0.67; OR 1.00; 95% CI 0.98–1.02), and size of climbing shoes used (P=0.28; OR 0.92; 95% CI 0.80–1.07) had no influence on injury rate. Intensity of downturn in climbing shoes did not affect the general risk of sustaining an injury (P=0.16; OR 1.14; 95% CI 0.95–1.36), but participants who used heavily downturned climbing shoes had a higher risk of sustaining an injury classified as UIAA ≥2 (n= 5071 P=0.0034; OR 2.58; 95% CI 1.34–4.95), despite adjusting for bouldering experience, history of injury, and age. Bouldering experience did not affect general rate of injury (P=0.40; OR 1.25; 95% CI 0.74–2.25) or rate of injury UIAA ≥2 (P=0.59; OR 1.04; 95% CI 0.90–1.22), but participants who had practiced bouldering on a regular basis for less than 1 y were more often affected by injuries of the lower extremities (n=507; P=0.027; OR 1.85; 95% CI 1.07–3.21). The risk of sustaining an injury was higher among those who had a history of injury (n=504; P=0.020; OR 1.54; 95% CI 1.07–2.21). Adjusting for bouldering experience and age had no influence on this result. Of the participants who had sustained at least 1 injury that caused more than 1 d absence from training or work prior to the study period, 48% (n=143) were injured during the study period, compared to 38% (n=78) of those without a history of injury. Examining only injuries of severity UIAA ≥2, no statistically significant difference was found.

Discussion

A considerable proportion of boulderers (44%) reported at least 1 injury during the assessed 12 mo. The majority of injuries were of minor severity. This extends findings from an prospective cohort study that found similar results, with an injury rate of 42% during a 12-mo period in a mixed population of indoor climbers and indoor boulderers; however, only injuries of the upper extremities were included, and an injury rate of 58% was found in the subgroup of boulderers.[14] Another prospective cohort study reported an alarming rate of injury, with 17 injuries caused by bouldering and 11 injuries caused by falling in 22 indoor boulderers within 12 mo, but these numbers could be biased by a high dropout rate and a small sample size.[2] In a retrospective study including a collection of mixed climbing styles, an injury rate of 50% within 12 mo was reported.[17]

Data on injury location are inconsistent in sport climbing. Some studies focus on upper extremity injury, excluding injury of the lower extremities, whereas others focus on a single injury mechanism.[8,14,19,20] A prospective single-center study in a German climbing area, including 836 patients with climbing injuries, reported a 6% rate of injuries of the lower extremities.[21] Three analyses of rock climbing injury treated in US emergency rooms reported that most acute climbing injuries concern the lower extremities.[5,13,22] It is worth mentioning that those 3 analyses included only acute injuries, no injuries due to overstrain, and were not limited to indoor bouldering. In our study population, the most common injury location was the upper extremity, especially hand, finger, and thumb, as well as shoulder and clavicle. Less than a quarter of all injuries affected the lower limbs, but those injuries were more severe and more likely to be caused by falling. These results suggest that further research is needed to determine methods for reducing the severity of lower limb injury and ways to improve falling and jumping safety.[1,8]

Table 4. Effect of preventive measures on injury rate

	Total sample size: Preventive measure used		*Injured participants (injury rate): Preventive measure used*		*Comparison of injury rate by usage of training method*	
	Yes n (%)	*No n (%)*	*Yes n (%)*	*No n (%)*	*P*	*OR (CI)*
Warming up of fingers	268 (53)	239 (47)	130 (49)	92 (39)	0.023	1.51 (1.06–2.14)
Warming up with easy boulder problems	478 (94)	29 (6)	211 (44)	11 (38)	0.51	1.29 (0.60–2.80)
Training with bodyweight or weights	269 (53)	238 (47)	125 (47)	97 (41)	0.20	1.26 (0.89–1.80)
Stretching	361 (71)	146 (29)	164 (45)	58 (40)	0.24	1.26 (0.85–1.87)
Yoga	122 (24)	385 (76)	54 (44)	168 (44)	0.90	1.03 (0.68–1.55)
Slacklining, balance training	96 (19)	411 (81)	41 (43)	181 (44)	0.81	0.95 (0.61–1.48)
Taping of the fingers	102 (20)	405 (80)	47 (46)	175 (43)	0.60	1.12 (0.73–1.74)
Taping of other locations	39 (8)	468 (92)	23 (59)	199 (43)	0.047	1.94 (1.00–3.77)
Cardio sports	257 (51)	250 (49)	105 (41)	117 (47)	0.18	0.79 (0.55–1.12)

Total sample size n=507; injured participants n=222; injury rate refers to ≥1 injury sustained during study period.

Most acute injuries were caused by bouldering itself, followed by falling. Injury caused by another boulderer is rare, which seems to be consistent with the literature.[2] Injury without acute trauma was sustained by 15% of our study population during the study period. Most of these injuries can be classified as overuse injuries, and most affected the upper extremities. The literature is inconsistent in terms of overuse injuries and rates differ greatly. A retrospective study reported a 44% career risk of sustaining an overuse injury, and another retrospective study investigating chronic climbing injuries reported a rate of 58% within 6 mo.[23,24]

In 1 previous study, a protective effect of weight training and wrist taping was found.[2] In our study population, use of the investigated preventive measures did not reduce the injury rate, a finding also supported by a study on youth rock climbers.[25] Warming up the fingers seemed to increase the risk of injury, but it is likely that this effect was caused by a confounder that we did not take into account within this study (eg, level of bouldering skill or compensation of risky behavior and frequency of use were not assessed).[25]

Spotting seems to be less common in German indoor bouldering facilities than in the indoor bouldering cohort of a North American prospective study.[2] Spotting behavior had no impact on injury rate in this indoor bouldering study population, but these results should definitively not be transferred to outdoor bouldering. There is literature on orthopedic changes and nerve compression syndromes caused by tight and unnaturally shaped climbing shoes that enable standing on small edges but force the foot and toes into an unnatural position.[8-10,26] Previous literature suggests a connection between choice of climbing shoe and the rate of injury in sports climbing.[8,9,26] Although there was no effect on the injury rate in our study population, there was an effect on injury severity. Thus, usage of heavily downturned climbing shoes might lead to a higher risk of severe injury. It may be beneficial to reduce usage of heavily downturned climbing shoes by using a second pair of loose shoes for training, as suggested by the literature on foot injuries in rock climbers.[8]

Beginners had a higher risk of injury of the lower extremities; thus, courses for beginners should teach safer falling techniques. The risk of injury was increased among those who had a history of injury, which is consistent with the literature on climbing injuries.[14,15] This finding is not limited to climbing-related sports but is common in other sports as well (eg, football and CrossFit).[27,28] The reason for a high reinjury rate in bouldering may be in the nature of the sport: hard moves at the athlete's maximum physical capacity, which are practiced repeatedly.

LIMITATIONS

Using an online questionnaire without any medical validation creates several limitations.

Because this study involves self-reported data, the results should be interpreted with caution. It is possible that participants provided false information, intentionally or unintentionally. Although the dropout rate was only about a quarter of the original participants, this may have influenced the results in either direction. There could be a reporting bias, meaning that those who experienced an injury could have been more likely to complete the follow-up, which could result in overreporting of the injury rate. Possible fatalities could not be detected in this study design, and fatalities would have been lost to follow-up. It is also possible that nonfatal injuries were lost to follow-up (eg, decreased willingness to participate in a study when bouldering

Table 5. Effect of training methods on injury rate

	Total sample size: Training method used		*Injured participants (injury rate): Training method used*		*Comparison of injury rate by usage of training method*	
	Yes n (%)	*No n (%)*	*Yes n (%)*	*No n (%)*	*P*	*OR (CI)*
Campus board	109 (22)	398 (78)	49 (45)	173 (44)	0.78	1.06 (0.69–1.63)
Pegboard	32 (6)	475 (94)	18 (56)	204 (43)	0.14	1.71 (0.83–3.52)
Fingerboard	105 (21)	402 (79)	56 (53)	166 (41)	0.027	1.63 (1.06–2.50)
Grip trainers	91 (18)	416 (82)	48 (53)	174 (42)	0.057	1.55 (0.99–2.45)
No use of a training method	262 (52)	245 (48)	107 (41)	115 (47)	0.17	0.78 (0.55–1.11)

Total sample size n=507; injured participants n=222; injury rate refers to ≥1 injury sustained during study period; training method refers to specific training method for climbing.

was no longer possible because of injury). This could lead to underreporting of the injury rate. Other results of this study, such as identified risk factors, may also have been influenced by participants lost to follow-up. There was no validation and examination of the sustained injury by a medical professional during the study period. The reported injuries were not always clear and easy to translate into medical terms, and the severity of injury was not always clearly assignable to 1 category of the UIAA Injury and Illness Severity Classification. This could result in both overreporting and underreporting of injury severity. Weekly bouldering time was assessed only in the baseline questionnaire and not in the monthly follow-up questionnaires. It is likely that during the duration of the study the real weekly bouldering time differs considerably from the initially stated time. This is why we did not state an injury rate per time. Future studies should survey the actual time spent bouldering to calculate injury risk per 1000 hours of bouldering, as the UIAA recommends.[18] Usage of preventive measures, specific training, and so on was surveyed only at the beginning of the study, and frequency of use was not assessed. There was no possibility to adapt changed training habits. This study did not examine any possible comorbidities that might act as confounders. We also did not investigate the difficulty of bouldering problems that caused an injury or the most difficult climbing problem that a participant was able to perform. We refrained from investigating these data because there is no comparable common grading system in most German indoor bouldering facilities. We used "bouldering experience" as a surrogate measure of bouldering skill to calculate an adjusted OR, but this variable probably does not fully correlate with bouldering skill. Due to the study design, injuries of 1-time boulderers and athletes who do not regularly boulder were not investigated. The size of climbing shoes differs between brands and is not always comparable. Furthermore, the perception of downturn and asymmetry is highly subjective. The height of fall was not consistently specified; therefore, we cannot state a correlation between injury and height of fall. The geographic limitation of this study to German-speaking participants could influence the injury rate because of different safety standards of indoor bouldering gyms.

Conclusions

Injuries in indoor bouldering are common. Lower extremity injuries are associated with higher injury severity. Commonly used preventive measures seem to be ineffective in reducing the rate and severity of injury. The choice of climbing shoes seems to affect severity of lower limb injury. Preventive measures need to be established to reduce bouldering injuries, especially during falls and landings.

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ORIGINAL RESEARCH

Coaches' Attitudes Toward Campus Board Training in Adolescent Climbers in the United States

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Introduction—Campus board training in adolescent climbers is controversial. Evidence, albeit limited, suggests this type of training may lead to the development of finger epiphyseal stress fractures. The purpose of the present study was to investigate coaches' attitudes toward campus board training in the United States.

Methods—Surveys were sent to 116 coaches at gyms across the United States with affiliated adolescent climbing teams. Outcomes collected included demographic information, training time, campus board use prevalence, coaches' attitudes toward campus board use, and willingness to participate in future research.

Results—Seventy-three coaches representing 3090 adolescent climbers completed the survey. Forty-six coaches (63%) reported using full weight (no foot contact) campus board training in some or all of their climbers. A variety of factors were cited by coaches when deciding which climbers should or should not use the campus board, including physical maturity, climbing ability, and age, among others.

Conclusions—Climbing coaches in the United States disagree on the use of campus board training in adolescent climbers. Some coaches avoid using this training modality entirely, whereas others believe certain climbers can use it safely. There is lack of consensus in determining who those certain climbers are.

Keywords: rock climbing, sport climbing, campus board, finger stress fracture, growth plate injury

Introduction

Rock climbing is a popular sport around the world with notable growth among adolescents.[1] Approximately 5 million athletes under the age of 18 y participate in rock climbing in the United States.[2] Furthermore, indoor competitive climbing, or sport climbing, is becoming increasingly popular, evidenced by its addition to the 2020 Olympic Games,[3] now set to debut in 2021.

Sport climbing is not without risk. Acutely, it is not uncommon to see injuries such as fractures, sprains, pulley ruptures, and ligamentous injuries.[4,5] The incidence of chronic climbing injuries is more difficult to ascertain, but overuse injuries occur commonly. These injuries generally involve the upper extremity and include muscle injuries, tendinopathies, and stress fractures.[5,6] Most climbing injury research to date has focused on adults. As youth participation grows, knowledge of injuries and risk factors specific to the adolescent population is critically needed. One of the most consequential injuries unique to the adolescent climber is an epiphyseal stress fracture of the proximal interphalangeal (PIP) joint of the finger.[7,8] The injury can occur after a clear, sudden event or insidiously, and climbers typically describe pain located at the dorsal PIP joint with point tenderness.[9-11] Left untreated, this injury can result in long-term disability, deformity, and pain.[6,9] The true incidence of stress fractures in the fingers of adolescent climbers is unknown owing to a scarcity of research investigating the subject. An estimated 100 cases have been reported in the literature.[9] Type and quantity of training are likely critical factors in the development of this injury, yet specific training recommendations to guide climbers, climbing organizations, parents of climbers, and climbing coaches are essentially nonexistent.

Campus board training, or campusing, is a commonly used training modality among climbers. A traditional campus board consists of a series of thin wooden slats in

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a ladder-like configuration, generally secured with a slight overhang of 0 to 20 degrees off vertical (Figure 1). Campusing typically involves ascending and descending these holds without foot contact. Campus board training in adolescent climbers is controversial. Evidence, albeit limited, suggests campusing may lead to the development of finger epiphyseal stress fractures and may even result in the development of finger osteoarthritis.[6,9,10] Although many athletes and coaches see it as a useful and safe training modality when used correctly, some clinicians and climbing organizations advise entirely avoiding use in athletes aged 18 y and younger.[5,8,9]

A recent study assessed adolescent climbers' knowledge of safe training practices, and slightly more than half (51%) of the 267 climbers involved were previously uninformed about growth plate injuries.[8] Among this group, almost two-thirds performed potentially unsafe training practices, including campus board training. Climbing coaches likely play a vital role in guiding training practices in this population, but studies assessing coaches' practices and attitudes toward this type of training are lacking. It is unclear from the current literature whether adolescent climbing coaches believe that campus board training is a safe or useful training modality, nor is it clear whether coaches have a protocol for deciding who should or should not campus. The purpose of the present study was to investigate coaches' attitudes toward campus board training in the United States.

Methods

This study was approved by the University of Washington institutional review board. Climbing gyms were identified across the United States using Google Maps with the search term "climbing gym" state by state. Gym websites and contact information were logged in a spreadsheet. The gym's website was then used to determine whether the gym likely had an affiliated youth climbing team. Whenever listed, a direct email contact for the climbing coach was documented. All other climbing gyms with a publicized climbing team were contacted via email requesting a direct email contact for the climbing coach. For gyms that did not respond, a follow-up email was sent 1 wk later. A final request was sent to gyms 1 mo after the initial email. In this manner, a list of climbing coaches' direct emails was compiled. In cases in which multiple coaches managed the same team, the head coach or most senior coach was identified as the primary contact.

Research electronic data capture (REDCap) software tools hosted at the University of Washington were used to create the survey and collect and manage responses. REDCap is a secure, web-based software platform designed to support data capture for research studies.[12,13] The survey design was edited and condensed in several phases with input from a local climbing coach, a physician and parent of an adolescent climber, and several independent musculoskeletal researchers before the creation of the final survey.

Survey questions were designed to assess the following: amount of time climbers spend training, coaches' attitudes toward campus board participation, and interest in future research participation. Questions were divided into 4 main categories: team information, training time, campus board training, and general monitoring. No questions asked for personal or team identifiers; however, respondents were asked how many climbers they personally coach to help ensure that they were in fact a team coach. Specific credentials required to be a climbing coach are established by the individual gyms at which the coaches work. Although USA Climbing does offer a coach membership and certification, this was not an explicit requirement for our survey respondents. Depending on the question, respondents were allowed to enter an integer, select 1 or many from a list of multiple choices, or answer with free text. When assessing the importance of certain factors coaches use to assess climbers' readiness for campus board training, a sliding scale was used with values of 0 to 100 (0 being "not something I care about" and 100 being "the most important factor I consider").

The survey was distributed to the aforementioned list of climbing coaches via email through the REDCap system. Links generated were unique to each survey recipient and could not be forwarded. Surveys were only sent to 1 coach per team in cases in which multiple coaches coached the same climbers. Additionally, respondents were explicitly instructed not to have multiple coaches of the same team complete the survey. Instruction was also included for the survey recipients not to complete the survey and instead to email our team directly if they were not coaches but had been mistakenly sent the survey. Survey recipients who did not complete the survey within 1 wk were sent 1 reminder email. Within the REDCap system, respondents were able to edit their survey answers until survey closure but were not allowed to submit more than 1 survey. All participants who completed the survey were entered into a lottery to win a $100 gift card as an incentive for survey participation. Survey responses were received anonymously through the REDCap system, and the survey was closed approximately 6 wk after the initial invitation to participate.

All responses were then exported to a spreadsheet for analysis. Totals, percentages, medians with interquartile range (IQR), and distribution were calculated using Microsoft Excel 2009 software. The data collected were nonnormally distributed as determined by the Shapiro-Wilk test. Therefore, data were reported with median and IQR.

Figure 1. Traditional wooden campus board.

Results

Requests for a coach's email address were sent to 214 climbing gyms. A list of 116 coaches' email addresses was created from the gyms that responded. Seventy-three of these 116 coaches completed the survey, for a response rate of 63%. Thirty-one states were represented in survey responses. A complete list of survey questions and responses is shown in Table 1.

TEAM INFORMATION

The median age of climber coached was 12 y (IQR 12–13 y), with the youngest climber coached reported as 4 y old. The number of climbers coached ranged from 5 to 160 with a median of 30 climbers per coach. The total number of climbers represented by coaches in this study was 3090.

TRAINING TIME

For their most competitive climbers during peak training/competition season, the respondent coaches reported that their climbers train for a median of 12 $h \cdot wk^{-1}$ (IQR 10–15 $h \cdot wk^{-1}$), with responses ranging from 5 to 25 $h \cdot wk^{-1}$. Coaches reported that these climbers train most of the year, with 33 of 73 (45%) reporting their most competitive climbers train all 12 mo. Seven coaches (10%) reported these climbers train 8 $mo \cdot y^{-1}$ or less.

Campus Board Training

Forty-six coaches (63%) reported using full-weight (no foot contact) campus board training in some or all of their climbers. In their most competitive climbers, the number of weeks per year campusing is used varied substantially, ranging from 2 to 50 $wk \cdot y^{-1}$. Of the remaining 27 coaches who reported they do not use campus board training in their climbers, 19 (70%) cited a concern that it could lead to injury.

A variety of training techniques, defined in Table 2, were used with the campus board. Half of the coaches reported other techniques in addition to those listed in the table. Many coaches reported using "bumping," a technique in which one hand remains on the bottom rung as the other hand is advanced sequentially, or "bumped," up the other rungs.

In general, coaches believed that age, climbing experience, climbing ability, the climber's comfort, and the coach's personal intuition are important factors when considering which climbers should use the campus board. Forty-two of 46 coaches who use campus board training responded in short-answer format to a question asking the coach to describe specific policies regarding campus board use in detail. Several themes emerged from these comments.

PHYSICAL MATURITY

Fifteen coaches referenced physical maturity as a measure used to evaluate readiness for campus use. Several coaches commented that a climber should be "past puberty" or "physically mature" as a prerequisite. One coach reported monitoring and recording growth rates in climbers. Another cited using x-ray and medical professional assurance that finger growth plates have indeed closed before allowing campusing.

CLIMBING FORM

Thirteen coaches referenced climbing or hanging form as a critical factor when evaluating a climber for campus use. One coach mentioned that climbers should keep shoulders "back and down" while also maintaining good body awareness. Another cited shoulder and scapular engagement as being important.

TYPES OF CAMPUS BOARD HOLDS

Nine coaches made some reference to the types of holds allowed while campusing. Some reported using only sloper

Table 1. Complete survey questions and answers

Questions and answer choices	*Respondents (%)*	*Median (IQR)*	*Range*
Team demographics			
How old is the youngest climber you coach?	73 (100)	8 (7–9)	4–12
What is the average age of youth climbers you coach (approximately)?	73 (100)	12 (12–13)	8–15
How many youth climbers do you coach (approximately)?	73 (100)	30 (18–60)	5–160
Training			
During peak training or competition season, about how many hours of formal (supervised) practice per week do your most competitive climbers perform?	73 (100)	7 (6–9)	4–12
During peak training or competition season, about how many hours of informal (not supervised, on their own time) practice/training per week do your most competitive climbers perform?	73 (100)	4 (3–6)	0–13
Total training calculated by adding 2 previous fields		12 (10–15)	5–25
During peak training or competition season, about how many hours per week are your most competitive climbers literally on the wall or hanging?	73 (100)	5 (2–6)	1–12
How many months out of the year are your most competitive climbers climbing?	73 (100)	11 (10–12)	6–12
Approximately what percentage of your climbers participate competitively in a sport other than climbing?	73 (100)	30 (11–50)	0–100
Campus board training			
Do you utilize full-weight (no foot contact) campus board training with your climbers?			
Yes, for all climbers	1 (1)		
Yes, for some climbers	45 (62)		
No	27 (37)		
How many weeks out of the year do you utilize campus board training in your most competitive climbers?	46 (100)	10 (6–20)	2–50
During peak training or competition season, how many hours per week do you utilize campus board training in your most competitive climbers?	46 (100)	1 (1–1)	0–2
What types of campus board training do you advise in your most competitive climbers? (select all that apply)	46 (100)		
Laddering	45 (98)		
Skip laddering	37 (80)		
Switch hands	16 (35)		
Double dyno	19 (41)		
Other	23 (50)		
For the following questions a sliding scale was used. (0="not something I care about." 50="a moderately important factor." 100="the most important factor I consider.")			
When determining who should train on the campus board, age is	46 (100)	79 (69–98)	40–100
When determining who should train on the campus board, climbing experience is	46 (100)	84 (72–96)	35–100
When determining who should train on the campus board, climbing ability is	46 (100)	80 (71–93)	49–100
When determining who should train on the campus board, the climber's comfort level is	46 (100)	70 (55–95)	31–100
When determining who should train on the campus board, my intuition is	46 (100)	68 (50–87)	9–100
How do you determine which of your climbers will utilize campus board training? Do you have any concrete rules? Please be as specific as possible.	42 (91)		
If you do not utilize campus board training for some or all of your climbers, why not? (select all that apply)	72 (100)		
It is not readily available	0 (0)		

(continued on next page)

Table 1 *(continued)*

Questions and answer choices	*Respondents (%)*	*Median (IQR)*	*Range*
I do not think it is an effective training modality	11 (15)		
I am concerned it could lead to injury	56 (78)		
It takes time and focus away from other more important aspects of training	36 (50)		
My climbers are physically unable to utilize a campus board	31 (43)		
Other	11 (15)		
You indicated that you have a concern campus board training could lead to injury. What led to this concern?	56 (100)		
Intuition (doesn't feel right)	12 (21)		
Personally experienced an injury this way	8 (14)		
Seen or coached climbers injured this way	26 (46)		
Physician or nurse's advice	6 (11)		
Physical therapist or athletic trainer's advice	23 (41)		
Scientific journal article	24 (43)		
Book, magazine, website, or news article	33 (59)		
Other	10 (18)		
If you would like to make any additional comments or clarify any of your answers regarding campus board training, please do so here:	29 (40)		
General Monitoring			
Do you keep a record of injuries that occur with your climbers?	73 (100)		
Yes	44 (60)		
No	29 (40)		
Would you be willing to utilize an online injury recording system to track injuries in your climbers for research purposes? (climber personal information would remain anonymous except to you, the coach)	73 (100)		
Yes, happily	24 (33)		
Yes, if it's easy	40 (55)		
No	9 (12)		
You answered "No." Why not?	7 (78)		

Categorical variables are presented as n (%). Continuous variables are presented as medians (interquartile ranges). Ranges are presented as minimum-maximum value. This survey used nested questions such that additional questions would appear to the survey taker only if applicable; thus, not every respondent saw every question. Percentages are based on the total number of respondents who were given that particular question.

or jug holds in younger climbers. One coach noted that only certain climbers could campus using a 30 mm edge or lower.

STRENGTH/ABILITY

Several coaches referenced specific strength targets they set for climbers before allowing campus use. For example, 1 coach noted that campus climbers should be able to perform 5 unassisted pullups. Five coaches cited that they require their climbers to boulder at least V5, an intermediate difficulty as measured by the V-scale, a grading scale used by climbers and climbing organizations to measure the difficulty of a bouldering problem.

AGE

Seven coaches noted they have a specific minimum age cutoff for campus board training. One coach cited using age 14 y as a cutoff, 1 coach cited age 15 y, 3 coaches cited age 16 y, and 1 coach cited age 17 y.

GENERAL MONITORING

A majority of coaches (60%) reported keeping a record of injuries in their climbers. Sixty-four (88%) expressed a willingness to use a central database to record injuries if it were available. Those who expressed they would be unwilling cited a lack of time to do so and/or concern about parental permission and anonymity.

Discussion

There is a paucity of published data regarding training habits among competitive adolescent climbers. The purpose of the present study was to investigate coaches' attitudes toward campus board training in the United States. We received survey responses from 73 coaches, representing over 3000 climbers. These responses provide insight into current training practices among adolescent climbing teams in the United States and warrant further investigation.

Table 2. Definitions of campus board training modalities

Training modality	*Definition*
Laddering	Ascending hand-over-hand to the top of the board
Skip laddering	Ascending hand-over-hand, skipping every other rung or more, to the top of the board
Switch hands	One hand positioned on 1 rung, the other positioned 1 rung higher, followed by switching hands mid-air simultaneously to the opposite rungs, and repeating
Double dyno	Simultaneously removing both hands from 1 rung and moving to the next rung or higher and then either dropping back to the original rung simultaneously with both hands and repeating or ascending until reaching the top of the board

TRAINING TIME

According to survey responses, nearly half of coaches' most competitive climbers train year-round. The American Orthopaedic Society for Sports Medicine recommends that athletes play a single sport no more than 8 $mo \cdot y^{-1}$.[14] Ninety percent of coaches in the present study estimated their most competitive climbers are climbing more than this. Consequences of overtraining have been documented in numerous sports and include overuse injuries, extremity pain, and burnout.[15-17] The present study was not specifically designed to assess for overtraining among climbers, but findings indicate a need for future studies that prospectively quantify how much time this cohort spends training.

CAMPUS BOARD TRAINING

More than half of coaches surveyed use full-weight (no foot contact) campus board training in some or all of their climbers. There are several potential mechanisms by which this type of training might put adolescent climbers at risk of developing epiphyseal stress fractures in the fingers. First, the thin ledge of a campus board generally necessitates the use of a crimp grip (Figure 2). This grip results in stress directed to the PIP joints, especially in the ring and middle fingers.[10,18] In adolescent climbers, it is thought that those forces can lead to fracture either acutely or with repetitive stress.[5,9,10] Salter-Harris type 3 fractures are seen most commonly, potentially because forceful flexion of the PIP joint during a crimp grip pushes the epiphysis dorsally and compresses it.[10,19] Another mechanism by which campusing may lead to injury is via the double dyno manuever.[5,6,10,20] A double dyno involves simultaneously removing both hands from one rung and jumping to the next rung or higher without use of the feet. This is often performed repetitively on the campus board. In the present study, 26% of coaches recommend double dyno training for at least some of their climbers.

A recent study suggests that speed climbing may be a risk factor for the development of growth plate injuries of the fingers as well.[20] Speed climbing is a climbing discipline that involves ascending a wall with uniform holds and hold placements as fast as possible. Campusing and speed climbing are similar in that they often involve repetitive dynamic movements such as the double dyno maneuver.[20] Because these maneuvers are done without foot contact, force through the upper extremities must overcome the climber's entire body weight. Repetitious force on the fingers could conceivably lead to growth plate fractures.

Lastly, adolescent climbers may be particularly vulnerable to growth plate injuries during growth spurts as bony maturation lags increasing musculotendinous use and force generation.[21] A combination of factors likely play a role in the development of this injury.

The majority of coaches surveyed believed campus board training to be an appropriate training modality for selected individuals. Approximately three-quarters of coaches expressed concern over the potential risk of injury and therefore exert some degree of caution. However, there was a lack of consensus regarding what restrictions should be in place and for whom. Twenty-one percent of coaches specifically referenced physical maturity as a prerequisite for campus use. This aligns with evidence suggesting athletes are particularly susceptible to epiphyseal injury during growth spurts.[9,10,21] However, it is unclear how coaches are measuring physical maturity. One coach reported monitoring growth curves. Another reported obtaining x-rays to confirm growth plate closure. Although x-rays can indeed confirm growth plate closure,[22] this exposes the athlete to radiation that may be unwarranted. Ten percent of coaches reported a strict minimum age required for campus board use. However, if the goal is to wait for growth plate closure, an age cutoff of at least 17 y is probably required.[9] This contradicts climbers' beliefs concerning campus board training—a majority believe it is safe to start double dyno campusing younger than age 18 y.[8] Eighteen percent of coaches specifically referenced an attention to climbing or hanging form as being critical to determining who should be allowed to campus. Often, they mentioned shoulder and scapular engagement as

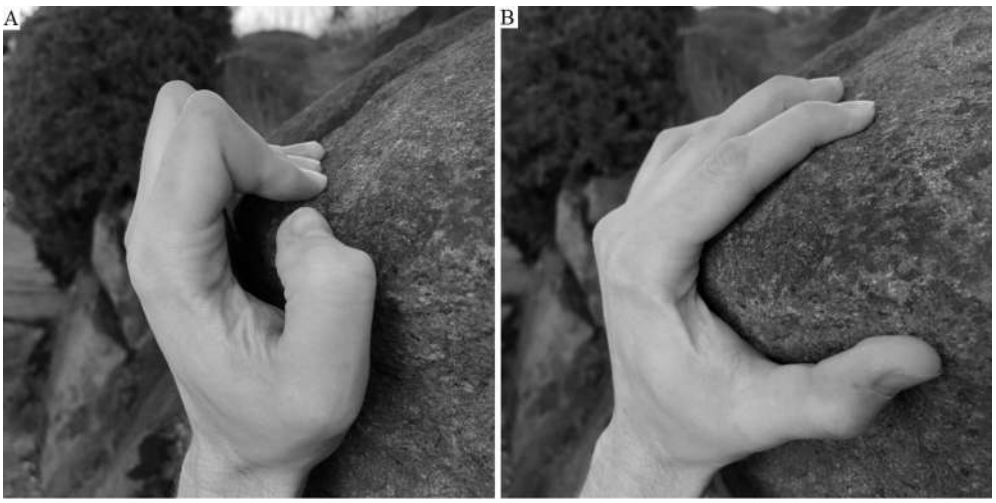

Figure 2. (A) Crimp grip. (B) Open hand grip.

being critical. Strength and proper activation of the shoulder and periscapular stabilizers might decrease load through the finger flexors during dynamic movements, but this is unproven and requires further investigation. As discussed, an open grip exerts less stress through the PIP joints than a crimp grip (Figure 2).[11] A traditional campus board consists of wooden slats; however, boards can be modified, and larger jug or sloper holds could be used as an alternative to thin ledges. These types of holds are more likely to encourage an open hand grip. Twelve percent of coaches made specific reference to the types of holds used in their training regimens, generally commenting that they promote larger holds in younger or less experienced athletes.

Overall, many coaches using the campus board with their athletes claim to do so in a thoughtful manner; however, prospective studies are needed to help determine the type and quantity of campus board use that is appropriate in this cohort.

LIMITATIONS

Our study is limited by its design as a survey and may suffer from recall bias. Our findings, particularly surrounding training time, should be interpreted with caution. Questions regarding how much climbers train relied on 1 group, coaches, recalling activity on the part of a second group, climbers. Although coaches may have been able to provide a reasonably accurate account of training time they formally supervised, they may have been unable to reliably estimate time spent training outside of supervised practice. This indirect assessment of training time decreases the overall reliability of the data. Additionally, many questions in our survey asked coaches for approximations or averages. These terms may have been interpreted differently among respondents. Coaches were asked to estimate how much their most competitive climbers train. "Most competitive" is subjective, and some coaches may have included more climbers than others when considering this term. Answers may have differed had we asked coaches to recall information regarding their "average climber." Coaches may have differing opinions of what defines campus board training, and that may have affected their response.

A novel survey was used for this study. A random sample was not compared to the overall sample, and this may reduce generalizability.

Coaches' responses potentially suffered from selection bias. Coaches in this study were identified by the climbing gyms for which they work and the teams they coach. They were not required to be formally credentialled or provide proof of license. Efforts were made to contact as many US climbing gyms as possible to create a comprehensive list of climbing coaches, but it is possible that some gyms were missed and never contacted and that some coaches work independently of climbing gyms. Additionally, it may be that those coaches more willing to participate in a survey are more likely to be interested in climbing research and more likely to consider how training may relate to injury.

Conclusions

Climbing coaches in the United States disagree on the use of campus board training in adolescent climbers. Some coaches avoid using this training modality entirely whereas others believe certain climbers can use it safely.

There is lack of consensus in determining who those certain climbers are. Physical maturity, age, ability level, and climbing form were cited as key factors, but specific criteria varied. This likely stems in part from a lack of evidence-based consensus guidelines in the United States and underscores the opportunity for further research. Coaches can play a helpful role in this endeavor and should continue to be recruited for future studies. We feel an obligation to help develop and substantiate training guidelines to protect the adolescent climbing community, and we enthusiastically welcome others into this effort.

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BRIEF REPORT

Fatal Events Related to Running Competitions in the Mountains

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Introduction—The few epidemiologic studies published previously about different forms of mountain running (ie, fell running, sky running, and ultratrail running) have not reported on fatal events. This report aims to contribute to the literature on mountain running fatalities, recording and classifying fatal events related to mountain running competitions found in online literature searches over a 12-y period.

Methods—From 2008 to 2019, searches on mountain sport, mountain races, and newspaper websites were periodically performed, looking for fatal events related to running competitions in the mountains. Data are presented as mean±SD or percentage, as appropriate.

Results—Fifty-one fatal events, involving 45 men (88%) and 6 women (12%), aged 50±11 and 46±6 y, respectively, were recorded in the 2008 to 2019 period in Western Europe. These events occurred during races (n=35; 69%), during training (n=13; 25%), and after races (n=3; 6%); 43% were sudden cardiac death, 32% due to falls (blunt trauma), 16% due to hypothermia, 4% due to both blunt trauma and hypothermia, 4% due to lightning strike injuries, and 2% due to animal attack (injuries from deer).

Conclusions—Understanding all of the causes of fatal events is necessary to institute preventative efforts and to organize rescues. Preventative efforts should be implemented by race organizers and by athletes themselves, and rescue teams can be trained and equipped to address all of these possible events. The relatively high percentage of sudden cardiac deaths stresses the need for preparticipation cardiovascular screenings. Further longitudinal studies are necessary to better understand the real impact of fatal events on the mountain running population.

Keywords: hypothermia, mountain running, blunt trauma, sudden cardiac death

Introduction

Since the 1990s, participation in running competitions in the mountains has notably grown owing to increased interest in off-road competitions[1] and an increase in the number of races along mountain trails of any type, distance, difficulty, and elevation.

Currently, considering the number of mountain races (200–220) and the average number of participants (150–180), it can be estimated that there are approximately 30,000 to 40,000 mountain runners in western Europe. Indeed, this is an overestimation because some runners participate in more than one event. It is difficult to calculate the true number of runners in mountain running competitions; however, by organizing events into the following categories, the number can be estimated:

1) Races in mountain trails characterized by a total length that exceeds the 42 km of the classical marathon, with more than 1000 to 1500 m total uphill and/or downhill elevation (named ultratrail-running);
2) Races on mountain trails ranging from 3.5 to ~80 km, with technical difficulties up to the second degree of the Union International des Associations d'Alpinisme climbing classification and with most technical parts of the route secured by mountain guides, reaching a minimum altitude of 2000 m above sea level, where acute mountain sickness can be possible[2] (named sky-running);
3) Races with different or some of the aforementioned characteristics, including fell running (a term popular in the United Kingdom, roughly meaning running on moor-covered hills).

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There is little epidemiologic data published on mountain running. A longitudinal screening of 1442 sky-runners participating in 36 competitions at between 2000 and 5200 m of altitude, with race times between 34 min and 10 h, showed that only 16 athletes (1%) withdrew for minor trauma (injury incidence of 9.6/1000 participants or 3.2/1000 h).[3] Another study on a 65-km mountain race reported 1.9 injuries/1000 participants (13.1/1000 h),[4] a result not so different from that reported for beginners (17.8/1000 h) and for recreational runners (7.7/1000 h).[5] None of these studies reported fatal events.

From these published data, running in the mountains is a sport with typically benign injuries and few to no fatalities, with only minor acute trauma such as contusions and ankle sprains, mainly affecting fell-runners and sky-runners during descents[6]; ultratrail runners are mainly affected by blisters, tendinopathies, cramps, and other overload injuries.[7]

Although fatalities in mountain running are infrequent, instances have occurred. Whereas there are studies on fatalities during running sports,[8] we did not find mountain running fatalities in our literature review, so it is difficult to understand their incidence and whether it may be possible to prevent them. For this reason, data reported in web-based and news sources were analyzed with the aim to contribute to the literature on mountain running fatalities.

Methods

In this brief report, we present a summary of fatal events related to mountain running competitions recorded in news published about mountain sports and mountain races and on newspaper websites from 2008 to 2019.

The study did not require ethics approval because the search and the analysis were performed on publicly available data, in accordance with the 1964 Helsinki Declaration involving human participants and its later amendments or comparable ethical standards.

This study considered some of the western European countries (Andorra, Austria, France, Italy, Spain, Switzerland, and the United Kingdom) where the aforementioned forms of mountain running are most popular. Consultation of websites was done periodically, every 1 to 2 mo, so this is a retrospective study. Searches were carried out using Google and Yahoo as web search engines, with several keywords: fall, fell-running, heart attack, hypothermia, mountain, running, sky-running, sudden death, trail running, training, ultratrail running, and the names of several races, variously associated with "death" and/or "fatal." When a fatal event related to running competitions on the mountains was found, the information was checked, and the web search was deepened for the place, date, and name of the race.

Table 1. Injury and illness classification—Union International des Associations d'Alpinisme MedCom Score

Score	*outcome*
0	No injury or illness
1	Slight injury or illness; no medical intervention necessary, self-therapy (eg, bruises, contusions, strains)
2	Middle severe injury or illness, not life-threatening; prolonged conservative or surgical therapy, outpatient therapy, doctor's attendance within a short time frame (days), injury-related work absence; heals without permanent damage (eg, undisplaced fractures, tendon ruptures, pulley ruptures, dislocations)
3	Major injury or illness, not life-threatening; residential hospital therapy, surgical intervention necessary, immediate doctor's attendance necessary, injury-related work absence; heals with or without permanent damage (eg, dislocated joint, fractures, vertebral fractures, cerebral injuries)
4	Acute mortal danger, polytrauma; immediate prehospital doctor or experienced trauma paramedic attendance if possible, acute surgical intervention; outcome: alive, permanent damage
5	Acute mortal danger, polytrauma; immediate prehospital doctor or experienced trauma paramedic attendance if possible, acute surgical intervention; outcome: dead
6	Immediate death

This is a retrospective classification that considers outcome and mortality, giving exact guidelines for classification.[9]

Events related to running competitions were defined as occurring 1) to participants during races, 2) within 1 h after crossing the finish line or to returning participants after suspension of the race (post-race); and 3) to runners in training for mountain competitions.

Fatal events related to injuries were classified as being fifth and sixth grade in the injury and illness classification Union International des Associations d'Alpinisme Medco Score, with a fatality risk calculation between I and III (Tables 1 and 2).[9]

The results were analyzed by descriptive statistics with frequency, percentage, and mean±SD. Linear regression was performed to assess any trend in the annual number of injuries. Significance was set at $P<0.05$.

Results

In the 12 y of the considered period, 51 fatal events (Table 3) affecting 45 men (88%) and 6 women (12%),

Table 2. Union International des Associations d'Alpinisme fatality risk classification

Grade	*outcome*
I	Fatalities technically possible but very rare, no objective danger (eg, indoor climbing)
II	Few objective dangers, fatalities rare, falls are not very dangerous, risk is mostly calculable (eg, sport climbing, mid-range Himalayan peaks)
III	High objective danger, risk difficult to calculate, falls lead frequently to injuries, fatalities more frequent (eg, traditional climbing, high Himalayan [7000–8000 m] or difficult peaks)
IV	Extremely dangerous, falls have a high fatality rate, totally unjustified to normal individuals

The classification grades the fatality risk of mountaineering and climbing sports.[9]

aged 50±11 (range 22–75) and 46±5 (range 38–50) y, respectively, were recorded. These events occurred during races (n=35; 69%), during training (n=13; 25%), and after races (n=3; 6%). There were 4.3±2.5 fatal events per year: 3.2±2.0 during races, 1.1±2.5 during training, and 0.3±0.5 after races.

Sudden cardiac death affected only men, aged 51±9 y (range 37–70), mainly during the races (91%), without any relationship to the distance traveled or the hour of the day. Information regarding the use of automated external defibrillators was reported for 3 cases only, preventing us from analyzing this aspect.

Two cases of hypothermia affected sweat-covered runners after nonfatal injuries reported during training. One case of lightning strike injury affected a female participant after suspension of an ultratrail race. One case of death during training was due to hemorrhage after a probable deer attack, as from the conclusion of the coroner.

During the study period, multiple fatal events during the same race were recorded only twice, both due to hypothermia. In 2008, there were 2 fatal events in the Zugspitze race (Germany), and in 2009, there were 3 fatal events during the Mercantour Raid (France). Two more fatal events were recorded in Spain, 1 during the Cavalls del Vent race (2012) due to hypothermia and 1 during training on the same path some weeks before the race (2015) due to leg trauma and subsequent hypothermia after getting lost. Finally, in the Sentiero 4 Luglio races (Italy), 1 fatal event due to sudden cardiac death occurred in 2013 and 1 fatal event due to a fall occurred in 2016.

Considering an estimated 30,000 to 40,000 participants per year in 2016–2019, with 4.5±0.6 fatal in-race events in each year, an incidence of 0.1/1,000 participants/y can be calculated.

Table 3. Fatal events related to mountain running competitions recorded in the 2008–2019 period

Fatal events	*Training*	*Race*	*Post-race*	*Sum*
Sudden cardiac death				
n (%)[a]	1 (2)	20 (39)	1 (2)	22 (43)
f/m	0/1	0/20	0/1	0/22
Blunt trauma (fall)				
n (%)	8 (16)	7 (14)	1 (2)	16 (31)
f/m	1/7	1/6	0/1	2/14
Hypothermia				
n (%)	1 (2)	7 (14)	0 (0)	8 (16)
f/m	0/1	2/5	0/0	2/6
Blunt trauma + Hypothermia				
n (%)	2 (4)	0 (0)	0 (0)	2 (4)
f/m	1/1	0/0	0/0	1/1
Lightning strike injury				
n (%)	0 (0)	1 (2)	1 (2)	2 (4)
f/m	0/0	1/0	0/1	1/1
Hemorrhage from deer attack				
n (%)	1 (2)	0 (0)	0 (0)	1 (2)
f/m	0/1	0/0	0/0	1/0
Totals				
n (%)	**13 (25)**	**35 (69)**	**3 (6)**	**51 (100)**
f/m	**2/11**	**4/31**	**0/3**	**6/45**

f, females; m, males.

[a]Percentages were calculated from the sum of all the events (n=51).

Discussion

The limited epidemiologic data on mountain running have reported only minor injuries. Nonetheless, a certain number of fatal events occurred yearly during the study period.

Comparing the entire period (2008–2019) to the last 4 y (2016–2019), a trend toward an increasing yearly incidence of fatal events was found (R^2=0.259). Considering this trend and the trend toward greater participation, a rise in mountain runner deaths would be expected to follow. However, the relatively low number of events recorded from soft sources requires caution in drawing conclusions. Further investigation is required.

Contrary to what one may think regarding mountain races, fatal blunt trauma subsequent to falls occurred much less frequently than sudden deaths. These events were almost always severe head injuries, occurring during the races and during training. In addition, a sweat-covered, injured runner can develop hypothermia in a cold environment. In most cases, falls were due to technical mistakes on descent or inadequate equipment.

Fatal events caused by hypothermia can occur when there is a sudden change in weather, such as snow or rain, owing to a decrease in temperature. If a participant is not adequately equipped or trained, or becomes lost, hypothermia can develop. The risk of hypothermia may be mitigated by using suitable compulsory equipment recommended by race organizers, who may also consider using alternative trails or suspending/canceling the race. In all of the races in which fatal hypothermia occurred, there were also many cases of nonfatal hypothermia.

Lightning strike injury is very rare, but possible, during violent thunderstorms, which currently seem to be more common owing to climate change. Usually, in the event of thunderstorms the race is canceled or suspended, but it is not always possible to communicate with all participants of an ultratrail race. Lightning strikes can cause sudden death owing to simultaneous cardiac and respiratory arrest or from blunt injury due to the shock wave that hits the athlete.

Finally, there is the possibility of encountering wild animals, especially when training alone.

Preventive efforts should be implemented by race organizers and athletes themselves. Both must know all of the risks of training and competing in the mountains and adopt the necessary measures. Rescue teams should be organized, trained, and equipped to face all of these possible events.[10] Tracking runners with global positioning systems during races and training appears to be a good solution and is currently adopted by many organizers. Tracking allows organizers to know exactly where participants are at all times and lets participants call for aid if they require assistance.

LIMITATIONS

The reliance on web and media sources of information posed a challenge to data collection and understanding the circumstances of these fatal events. These reports allow for the collection of limited data regarding the event, so it is challenging to obtain detailed information about the casualty and precisely analyze it. A further limitation is the difficulty in verifying questionable information that can be found in news reports.

Although consultation of the various websites was done periodically, it is probable that some events were missed (ie, those occurred during training). Furthermore, an absence of data identified in a search does not necessarily equate to an absence of cases.

Another limitation is that it is not possible to calculate the real incidence because of the impossibility of counting the total number of participants, which should include participants in races in which there were no fatal events and runners in training outside competitions.

However, this report has tried to collect and classify fatal events related to mountain running, giving the reader a more realistic idea of the safety of this sport and the causes of fatalities. Further investigation is necessary.

Conclusions

In western Europe, fatal events related to mountain running competitions (ie, fell running, sky running, and ultratrail running) were collected over the past 12 y. Understanding the most frequent causes of these events, and all of their causes, is necessary to institute preventive efforts and organize rescues. The relatively high percentage of sudden cardiac deaths stresses the need for preparticipation cardiovascular screening. Further longitudinal studies are necessary to better understand the real impact of fatal events on the mountain running population.

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BRIEF REPORT

Epidemiology of Ice Climbing Injuries Among Recreational Ice Climbers at the 2019 Ouray Ice Climbing Festival

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Introduction—As a close relative to the sports of mountaineering and rock climbing, water ice climbing has been traditionally considered "high risk." There is little data to support or refute this assertion. Prior estimates of the injury rate range from 4.8 to 248 injuries per 1000 participation hours. In this study, we characterized the types of injuries and determined an injury incidence among water ice climbing participants at the 2019 Ouray Ice Festival climbing manmade ice walls.

Methods—A survey was distributed during the 2019 Ouray Ice Festival in Ouray, Colorado. Respondents were asked to include festival-related injuries and estimate their participation time at the 4-d festival. Individuals who reported an injury were asked to provide additional details with regard to injury type, location, and timing. The injury incidence rate was calculated as the total number of injuries reported divided by the total number of participation hours and is reported as injuries per 1000 participation hours.

Results—A total of 75 surveys were analyzed. A total of 16 injuries, all which were minor, and 912 participation hours were reported, resulting in an injury incidence of 17.6 injuries per 1000 participation hours.

Conclusions—This study reports an injury incidence based on an unselected sample of water ice climbers at an ice climbing festival. Water ice climbing at festivals appears to result in injuries of minor severity with no major accidents or fatalities. This study could be repeated to confirm its validity with a larger sample size.

Keywords: mountaineering, athletic injuries, event medicine, sport festival, ice park

Introduction

Water ice (WI) climbing consists of ascending vertical or overhanging ice formations such as a frozen waterfall or an ice wall using ice tools and crampons (Figure 1).[1] Once limited to elite athletes and mountaineers, the sport is rapidly becoming more accessible to the public. There are now 16 annual ice climbing festivals held in North America that allow enthusiasts to easily obtain the equipment needed to trial the sport. The Ouray Ice Festival, now in its 26th year, is one of these festivals and is held at the Ouray Ice Park in Ouray, Colorado. The Ouray Ice Park is a manmade ice climbing venue created in the Uncompahgre Gorge (Figure 2). Using an intricate plumbing system, water is sprayed down the walls of the gorge nightly to form the ice necessary for climbing. The Ouray Ice Festival is the largest in North America and hosts approximately 2000 ice climbers of all experience levels.

WI climbing has been traditionally considered "high risk." However, there is little data to support or refute this assertion. Prior studies of ice climbers have reached no consensus on participant safety, with injury rates ranging from 4.8 to 248 injuries per 1000 participation hours.[1-3] For comparison, the injury rates of various activities

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Figure 1. Ice formation.

among Finnish adults was found to range from 0.3 per 1000 participation hours for golf to 18.3 per 1000 participation hours for squash.[4] A prospective study of indoor rock climbers found an injury rate of 0.02 per 1000 participation hours,[5] and a post hoc analysis of data from traditional rock climbers in Yosemite yielded an injury rate of 37.5 per 1000 participation hours.[6]

The lowest ice climbing injury rate (4.8 per 1000 participation hours) is from a retrospective study of predominantly advanced climbers, 20% of whom were competition athletes.[1] Climbers reported injuries and climbing time over the past 3 y in a single survey, introducing significant recall bias and possibility for underreporting of injuries. A single-season, prospective survey of intermediate to advanced climbers found an injury rate of 9.8 injuries per 1000 participation hours.[3] Both studies used selected sampling methods in which the authors recruited participants via personal communication with known contacts. The highest injury rate reported is from a study of non-Olympian visitors to the 2014 Sochi Winter Olympics (248 injuries per 1000 participation hours).[2] This estimate is based on recreational speed ice climbing on the competition climbing wall by visiting spectators. No prior ice climbing experience was required to participate, and no data on the skill level of participants were collected. The authors enrolled all climbers as part of the waiver process. However, this estimate cannot be generalized to WI climbing because participants were limited to 2 min of climbing on a speed climbing wall.

In this study, we characterize the types of injuries and report an injury incidence among WI climbing participants at the 2019 Ouray Ice Festival.

Figure 2. View of Uncompahgre Gorge from bridge.

Methods

This study was reviewed and approved by the institutional review board of the University of California San Francisco Fresno medical education program.

SURVEY

A survey was designed using terminology and methods in accordance with Union Internationale des Associations d'Alpinisme (UIAA) Medical Commission recommendations.[6] Respondents were asked to report ice climbing–related injuries and estimate their participation time (number of climbing days and average hours of climbing per climbing day) at the 4-d festival. The UIAA Medical Commission recommends that injuries and illnesses that occur during belaying, rappelling, approach to climbing, or descent from climbing be reported for epidemiologic analysis of climbing sports and that time spent on these "non-climbing" activities be included in incidence calculations.[7] As such, we specifically asked participants to report all injuries occurring during these activities. In the event that respondents did not provide an estimated average of hours of climbing per day, 6 h per

climbing day was used based on UIAA Medical Commission recommendations.[7] Individuals reporting an injury were asked to provide additional details with regard to injury type, location, and timing.

Demographics on the survey included prior ice climbing experience. Participants self-assessed their ice climbing experience by selecting the highest WI grade they could climb consistently. The difficulty of a WI climb is dependent on length, vertical overhang, and ice quality; the difficulty scale ranges from WI 1 to WI 7.[8] To align with prior literature, climbers were characterized as beginner (WI 1), intermediate (WI 2–WI 4), or advanced (≥WI 5).[3]

SURVEY DISTRIBUTION

The 2019 Ouray Ice Festival was held between January 23 and January 26, 2019, in Ouray, Colorado. Paper surveys were distributed by volunteers from the University of Colorado wilderness medicine section and Event Medical Specialists who were staffing the medical tent. Volunteers were present 8 h per day during 3 d of the festival. Multiple volunteers were available, with teams stationed at the medical tent and patrolling the park. Reasons for visiting the medical tent aside from medical care included asking questions about the medical team's role at the festival and available medical courses.

DATA MANAGEMENT AND ANALYSIS

All data were imported into Google Sheets (Google LLC, Mountain View, CA) for analysis. Responses were excluded if the respondent reported an age <18 y or reported not climbing during the festival. Only injuries reported to have occurred during climbing, belaying, rappelling, approach to climbing, or descent from climbing were included in calculations.

The injury incidence rate was calculated as the total number of injuries reported divided by the total number of participation hours and is reported as injuries per 1000 participation hours. When categorical responses or ranges of numerical responses were given instead of a single number, the following methods were used to standardize answers to a numerical format for data analysis: 1) If participants wrote "all" when asked how many days they climbed during the festival, we inputted the median number of days climbed in the remainder of our data, which was 2 d; 2) if participants failed to give an answer or wrote "all day" when asked how many hours climbed per day, we inputted the UIAA recommended estimate of 6 h per day; 3) if participants wrote a number range when asked how many hours climbed per day, we inputted the average of the given range as the value (eg, 3–4 became 3.5); and 4) if participants wrote an answer in the "X+ hours" format when asked how many hours climbed per day, we inputted the number provided (eg, 8+ became 8). Medical volunteers from Event Medical Specialists and the University of Colorado wilderness medical section were responsible for medical care during any major incidents at the festival (Figure 3). Their reported incidents rates and EMS transports were queried to catch all major injuries.

The incidence rate ratio was calculated to compare injury rates among experience levels. Results are reported as median and interquartile range (IQR) as appropriate.

Results

A total of 81 surveys were completed. Seventy-five surveys were analyzed after 6 were excluded due to age <18 y (n=1), not climbing at the festival (n=4), and being deemed an outlier for reporting climbing 30 d at a 4-d festival (n=1).

Respondent age ranged from 18 to 70 y (n=72, median 30.5, IQR 27–36) and 28% (n=20) of respondents were female. The median number of seasons of ice climbing experience was 3 (n=74, IQR 1–6). The median number of days climbed per season was 5.5 (n=72, IQR 3–20). The median highest WI grade climbed consistently was WI 4 (n=72, IQR WI 3–WI 4). Safety equipment utilization while belaying and climbing is shown in Table 1.

Fourteen respondents reported a total of 16 injuries. One injury occurring during an unrelated social activity was excluded. An injury occurring during skiing, not climbing, was assumed to be during approach or return and was included in injury calculations. Eleven injuries occurred during climbing or rappelling, and 5 occurred during the approach or descent. Of the 16 injuries, 3 were self-reported as "overuse injuries." The location and type of injuries are shown in Tables 2 and 3.

Climbing time estimates that were not a numerical response were addressed as per the Methods section. A numerical substitution of the median number of days climbed for a categorical response ("all") was performed for 2 entries. An estimate of 6 h per climbing day was used for 3 entries. A numerical substitution of the average of an hour range for climbing time was performed for 4 entries. A numerical substitution of the base number of hours being defined as "X+ hours" was performed once. A total of 912 participation hours were reported, resulting in an injury incidence of 17.6 injuries per 1000 participation hours. No medical incidents were reported at the medical tent. No patients required EMS transport. An online search for reported fatalities or major incidents at the 2019 Ouray Ice Festival did not yield any relevant results.

Figure 3. Volunteers providing medical care.

A total of 3 beginner (4%), 52 intermediate (72%), and 17 advanced (24%) climbers responded (n=72). Injury rates for the intermediate and advanced climbers were 16.5 and 5.2 per 1000 participation hours, respectively. One injury was reported in the beginner cohort; however, no injury rate was calculated owing to the small sample size of 3 persons. The odds ratio of injuries among intermediate climbers as compared to advanced climbers was 4.3 (95% CI 0.5–36.0).

Discussion

In this survey of ice climbers at the 2019 Ouray Ice Festival, we found an injury incidence of 17.6 per 1000 participation hours. This incidence is comparable to that found in a prospective study (9.8 per 1000 participation hours) and is considerably lower than that found among amateurs with no ice climbing experience at the 2014 Sochi Olympics (248 per 1000 participation hours). The latter study calculated an injury rate using an unselected sample; however, the population studied was one with minimal climbing experience. In contrast, our study reports an injury incidence based on an unselected population of actual WI climbers.

Although ice climbing may be colloquially viewed as a single sport, using this assumption for epidemiologic purposes is akin to considering "track and field" a single sport. Just as there are differences in the types and number of injuries between those competing in various track and field disciplines,[9] we must consider differences in risk profiles among the various subdisciplines of ice climbing. This is relevant with regard to discussion of the 2014 Sochi study. The participants of this study climbed on a speed ice climbing wall and were limited to 2 min of climbing for each attempt. Speed ice climbing differs from traditional WI climbing by occurring in standardized, easily accessible venues to ensure fair competition. Because participants are not required to spend time belaying, rappelling, and approaching to or descending from a climb, the time spent speed ice climbing is significantly shorter than the time spent WI climbing. As such, the estimate of 248 per 1000 participation hours is based on a total of 18 injuries that occurred during 73 h of climbing. This injury incidence cannot be generalized to WI climbing as a whole owing to the unique circumstances and risk profile of this study population.

With regard to climbing experience and injury rates, a prior prospective study found a statistically significant difference in the injury rate between intermediate and advanced climbers (16.2 vs 6.3 per 1000 participation hours; OR 2.55, 95% CI 1.17–5.54).[3] Our incidence rates are similar for the 2 experience groups, at 16.5 and 5.2 per 1000 participation hours for intermediate and advanced climbers, respectively; however, there was no statistical difference between these 2 groups within our study, which may be related to our small sample size.

We evaluated the safety equipment use, injury locations, and injury description among our participants in a fashion similar to a prior prospective study.[3] Our data are consistent in showing that 100% of participants used a helmet while climbing. Of note, 41% of participants reporting using an assisted braking belay device (GriGri), which, if operated correctly, provides an additional safety margin while belaying. We noted that 53% of

Table 1. Reported use of safety equipment

Equipment	*Belaying % (n)*	*Climbing % (n)*
Backup belay	27 (20)	–
Crampons	91 (68)	–
Eye protection	49 (37)	53 (40)
Gaiters	–	27 (20)
Assisted braking belay device (GriGri)	41 (31)	–
Helmet	97 (73)	100 (75)
Leashes	–	17 (13)

Table 2. Injury locations

Location	*% (n)*
Head/Face	31 (5)
Shoulder/Clavicle	13 (2)
Elbow	6 (1)
Lumbar spine	6 (1)
Knee	6 (1)
Lower leg	19 (3)
Ankle	19 (3)

Table 3. Injury descriptions

Description	*% (n)*
Abrasion	9 (2)
Bone fracture	5 (1)
Contusion	5 (1)
Joint sprain	23 (5)
Laceration	27 (6)
Muscle strain	18 (4)
Tendonitis/Tendon strain	9 (2)
Other	5 (1)

participants used eye protection while climbing, which is higher than the prior value of 31%. This is important given that both our study and this prior study show the most frequent injury location to be the head/face (31%). Unlike this prior study, however, our data revealed the most common injury types were lacerations (n=6, 38%), joint sprains (n=5, 31%), and muscle strains (n=4, 25%). The prior study noted 74% of injuries described as abrasions or contusions, whereas ours only reported 19%. The higher proportion of sprains and strains could be related to the festival setting, where participants might be doing more climbing in a short period of time than they would otherwise. Overall, the severity of reported injuries appears to be minor, with only one reported fracture.

LIMITATIONS

This study has notable limitations. The retrospective nature introduces recall bias. We attempted to limit this by asking participants to only report recent climbing and injuries, rather than those occurring over the course of the past climbing season. Our response rate and sample size are both low (75 of approximately 2000 participants). As with any survey, our study had incomplete and missing data. When ranges, incomplete responses, or categorical responses were given in lieu of a single numerical value, we used median values and/or conservative estimates that would err on the side of inflating the injury rate rather than minimizing it. This was performed in 10 instances. Specifically, 2 participants did not provide an answer to the number of hours climbed in a day and 1 participant responded "all day." Our calculations used the UIAA recommendations of estimating 6 h per day climbed for these 3 instances. If we imputed the median number of hours climbed per day instead, the calculated injury incidence is 17.7 based on a total of 903 participations hours, which is essentially unchanged.

A single survey did not allow us to collect data on overuse injuries with delayed presentations. This may lead to incorrect estimation of the true injury rate. Participants were asked to self-report data such as climbing experience and safety equipment use, which can result in recall and social desirability biases. Injury description information is also based on the climber report. It is possible that injuries reported as minor (eg, joint strain, contusion) were later diagnosed as more significant by a medical provider (eg, tendon rupture or fracture). Injury severity was inferred by authors. It is possible that this was underestimated (eg, complex laceration or subsequent infection).

An important caveat to note is the unique climbing setting at ice festivals in general and specifically at the Ouray Ice Park. Festivals tend to be crowded, resulting in decreased spacing among climbers and poor ice quality, both of which have potential for increasing injuries. Questions on alcohol or recreational drug use before climbing were not included in the survey. Some climbers may opt to avoid festivals owing to their crowded nature, leading to selection bias in our study and decreased generalizability to ice climbing outside of this setting. The Ouray Ice Park is unique in its lack of technical approaches, readily available top rope anchors, and predominantly manmade ice. The climbs within the Ouray Ice Park climbs are graded at a maximum of WI 5. A number of mixed (rock and ice) climbs are also available. We did not differentiate between climbers climbing only on WI vs mixed climbs. Any of these factors can conceivably increase or decrease the observed injury rate. Given the differences between ice climbing at a festival and ice climbing outside of this setting, our estimate cannot be readily generalized to WI climbing as a whole.

Conclusions

We calculated an injury incidence rate based on an unselected sample of WI climbers at an ice climbing festival. WI climbing at festivals appears to result in injuries of minor severity with no major accidents or fatalities. This study could be repeated to confirm its validity in a larger sample and would ideally include participant follow-up to further characterize injury severity. Additional studies eliciting injury rates among WI climbers while avoiding the limitations of this and other prior studies are warranted.

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BRIEF REPORT

A National Survey of Wilderness Medicine Curricula in United States Emergency Medicine Residencies

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Introduction—Wilderness medicine (WM) is a growing subspecialty of emergency medicine. In 2018, we surveyed all 240 emergency medicine residencies in the United States to assess the scope of WM education in emergency medicine training programs in light of the nearly 30% increase in the number of residencies since 2015.

Methods—A survey was e-mailed to the Council of Residency Directors in Emergency Medicine listserv and individual program directors of each of the 240 residencies. The survey included questions on educational content, format, number of hours taught, availability of conference credit, offering of an elective or fellowship, and several predefined WM curricula. We evaluated differences between 3-y and 4-y residencies using the χ^2 test, where $P<0.05$ was considered significant.

Results—We had a response rate of 57% for completed surveys. Analysis showed 63% of respondent programs teach WM material. The majority (86%) partially or completely developed their curriculum, with 33% offering at least 1 of the predefined curricula. Thirteen percent taught with lecture only, 2% taught by hands-on only, and 85% used a combination of the 2. WM electives were significantly more likely to be offered by 4-y than 3-y residencies ($P=0.009$).

Conclusions—Almost two-thirds of respondent residency programs teach WM material. Of these, only one-third teach any of the predefined curricula. Four-year residencies are more likely to offer WM electives but are otherwise comparable to 3-y programs.

Keywords: education, residents, didactics, fellowships

Introduction

Wilderness medicine (WM) is a subspecialty with a growing body of literature and expanding educational opportunities. As of June 2019, there were 17 WM fellowships in the United States (14 at the time of our survey).[1] A number of curricula for WM have been published, including models focused on US Board of Emergency Medicine core content.[2-5]

A 2015 assessment of WM content in residency training reported that 45 programs taught WM as part of their curriculum, with almost all (96%) of the programs using lecture as their primary format and 51% using hands-on training.[6] Since that time, there has been a 28% increase in the number of emergency medicine residencies in the United States, from 187 to 240.

In this study, we update and expand the knowledge of the current state of WM education in the United States on a national level, including detailing the inclusion of formally available WM curricula into residency training.

Methods

We designed an 8-question survey to explore the level of WM training, availability of conference credit, and overall hours in the didactic curriculum dedicated to WM content in each residency program. Yale University's institutional review board approved the study (protocol ID #2000021632).

We developed and refined the survey instrument through discussion among the education faculty in our department. Additionally, we evaluated the use of several predefined curricula, including wilderness first aid/wilderness life

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support,[7] basic wilderness life support/wilderness first responder,[8] and advanced wilderness life support (AWLS).[9] Survey questions consisted of multiple-response or fill-in-the-blank formats and were tested among a group of residency educators for clarity.

The questionnaire employed Qualtrics software and was distributed to all emergency medicine residency program directors and assistant/associate program directors using the council of residency directors in emergency medicine listserv. All 240 of the Accreditation Council for Graduate Medical Education–accredited emergency medicine residencies were invited to participate. Programs that did not complete the survey received a second invitation within 2 mo. Finally, we directly e-mailed program directors of nonresponding programs with an invitation to complete the survey. No incentives were offered.

Survey responses were anonymously reviewed, and names of residency programs were elicited separately as identifiers only to ensure recognition of duplicate survey responses. If respondents answered "no" to Q1 ("Does your program teach wilderness medicine?"), the survey was considered complete. If respondents answered "yes" but did not answer any of the subsequent questions, the survey was considered incomplete. When specific residency programs submitted more than 1 survey response, these were checked for conflicting data and merged as follows. For questions on course format (Q3), hours spent (Q4), and type of teaching (Q6), we consolidated responses and included all answers. When programs submitted multiple, conflicting answers for either the conference credit question (Q5) or the elective offerings question (Q7), the merged answer defaulted to "yes." Here, we assumed that an affirmative response was more likely to be accurate and based on actual knowledge of the residency's WM offerings, whereas a respondent might answer in the negative simply due to lack of awareness of available options. Omitting these conflicting observations did not change result significance. One program had a conflicting entry on WM fellowship availability (Q8), which we cross-checked with the Society for Academic Emergency Medicine website on fellowships for accuracy.[1]

Data were exported to Microsoft Excel and R for quantitative analysis. We evaluated differences between 3-y and 4-y programs using the χ^2 test, where $P<0.05$ was considered significant. The 95% CIs were calculated using the Wilson interval.[10] Proportions were reported as percentages, where the denominator was the number of programs responding to each question rather than the total number of programs answering the survey. For the formal curricula (Q3), percentages do not sum to 100% because certain residencies teach multiple curricula.

Results

The survey was sent to 240 programs and received 189 responses, 16 of which did not identify their program by name and were therefore excluded from the analysis. Of the remaining 173 responses, we excluded 16 as incomplete. There were 38 duplicate responses attributable to 17 programs, which we resolved using the rules outlined earlier. This left a total of 136 complete surveys, giving a response rate of 57%. Table 1 summarizes the results.

Analysis of the data showed that 85 (63%) respondent programs have some form of WM training as part of their residency curriculum. Subsequent results analyze these 85 programs. Sixteen (19%, 95% CI 12–28) taught basic wilderness life support/wilderness first responder, 16 (19%, 95% CI 12–28) taught wilderness first aid/wilderness life support, 9 (11%, 95% CI 6–19) taught AWLS, and 73 (86%, 95% CI 77–92) at least partly developed their own curriculum, with many programs offering a combination of these curricula. Twenty-eight (33%, 95% CI 24–43) programs offered some formalized curricula as part of their teaching.

Among programs specifying hours spent teaching the subject matter (n=82, 60%), 40 (49%) taught ≤5 h, 22 (27%) taught between 6 and 10 h, and 20 (24%) taught >10 h cumulatively over the course of an academic year. Forty-nine (58%, 95% CI 47–68) offered conference credit for WM material.

Eleven (13%) taught with lecture only, 2 (2%) taught by hands-on only, and 72 (85%) used some combination of the 2 modalities. Thirty-one (36%, 95% CI 27–47) programs offered a WM elective, and 10 (12%, 95% CI 7–20) offered a WM fellowship.

Comparing 3-y and 4-y programs, there was no significant difference in teaching WM ($P=0.547$), the inclusion of at least 1 of the formal curricula versus own curriculum only ($P=0.962$), or offering of a fellowship ($P=0.104$). Four-year programs were significantly more likely to offer an elective in WM ($P=0.009$).

Discussion

The majority (63%) of respondent residencies offer some level of WM training as part of their curriculum, predominantly as a combination of lecture and hands-on teaching. In comparison to 2015,[6] residency program numbers overall have increased from 187 to 240 (+28%), and programs offering WM education have increased from 45 to 85 (+89%). Although WM content has always been part of the model of the clinical practice of emergency medicine[11] and thereby an expected component of the educational curriculum of any residency, the increasing amount of WM education reported

Table 1. Survey questions and compiled responses

	Total		*3-y programs*		*4-y programs*		*P*
	n	%	*n*	%	*n*	%	
Q1 Does your program teach WM?							
Yes	85	63	61	61	24	67	0.547
No	51	38	39	39	12	33	
Q2 What type of residency is your program?							
3 y	100	74		n/a		n/a	n/a
4 y	36	27					
Q3 What level of WM is being taught?							
Own curriculum only	57	67	41	67	16	67	0.962
Some formal curriculum[a]	28	33	20	33	8	33	
BWLS/WFR[b]	16	19	10	16	6	25	n/a[a]
WFA/WLS[c]	16	19	11	18	5	21	
AWLS[d]	9	11	8	13	1	4	
Q4 How much time (in cumulative hours) is spent over the academic year on teaching WM?							
≤5 h	40	49	27	45	13	59	0.148
6–10 h	22	27	15	25	7	32	
>10 h	20	24	18	30	2	9	
Q5 Does your program offer conference credit for any WM courses or teaching?							
Yes	49	57	33	54	16	67	0.291
No	36	42	28	46	8	33	
Q6 What format does your program teach by?							
Lecture only	11	13	7	12	4	17	0.562
Hands-on only	2	2	2	3	0	0	
Both	72	85	52	85	20	83	
Q7 Does your program offer a WM elective?							
Yes	31	36	17	28	14	58	0.009
No	54	64	44	72	10	42	
Q8 Does your program offer a WM fellowship?							
Yes	10	12	5	8	5	21	0.104
No	75	88	56	92	19	79	

[a]Subcategories showing number of programs teaching each curriculum do not sum to 100%, and a *P* value is not calculated because multiple options were possible.
[b]basic wilderness life support/wilderness first responder.
[c]wilderness first aid/wilderness life support.
[d]advanced wilderness life support.

by respondents may suggest a growing interest in, and recognition of, unique WM didactic content.

Approximately one-third of programs teaching WM used some predefined course format, such as AWLS, whereas the majority (86%) at least partially developed their own teaching content. Almost half (49%) taught ≤5 h of content over the course of an academic year, an increment chosen to reflect the equivalent of 1 full conference day or less for most emergency medicine residency programs. There was no significant difference in the hours taught, format taught, or fellowships offered by 3-y versus 4-y programs. Four-year programs were significantly more likely to offer a WM elective, as would be expected considering they generally incorporate more elective months into their residency format.[12]

It should be noted that only 4 of the 14 programs offering WM fellowships[1] at the time were nonresponders, for a response rate of 71% in this subgroup. This could suggest a response bias favoring programs that are more invested in WM education and a possible overestimation of the percentage of programs teaching WM.

Published curricula for WM medical student electives, resident tracks, and fellowships are available,[2-5] but no specific residency curriculum other than the model of the clinical practice of emergency medicine[11] exists in the United States. We queried the use of 3 widely available and commonly known teaching curricula,[7-9] but others exist and could be included in future studies. Although the queried curricula are intentionally accessible to a nonprovider audience, they are familiar

to most WM-interested emergency physicians and, in the absence of a national curriculum, are a source of structure in teaching WM content.

Our study shows that there is a range of WM education formatting, in terms of hours, didactic style, and use of formalized courses, with no singular format predominating. Although independently customizing their curricular content allows programs to adjust to resident needs and available resources, a more widely accepted national consensus and educational practice guidance may result in a more consistent educational experience. One barrier may be the cost of acquiring educational materials, along with the lack of local expertise or familiarity with formal curricular options. The number of hours taught is rather low, but it reflects the amount of WM content included in the 2016 emergency medicine model[11] and therefore its representation in the US board of emergency medicine in-training and qualifying (written) board examinations.

LIMITATIONS

We did not define the term "wilderness medicine" in our survey introduction. Our intention was to capture data on what respondents believed was included under the heading. This may have led to under- or overreporting of content, especially because the emergency medicine model includes this under the heading of "environmental disorders."[11] E-mailing residency program directors was not a perfect proxy for responses directly from WM education faculty; program directors may not necessarily have been aware of the exact details of the WM educational format at their institution. Unfortunately, no national listing of all residency WM educational leaders exists.

Another limitation was a possible response bias: Programs that are invested in WM content may have been more likely to complete the survey. Increased survey participation by programs enthusiastic about WM could inflate our estimate of programs teaching WM content. If none of the nonresponding programs taught WM, then only 35% of the total 240 residencies would be teaching WM. Although it is difficult to increase survey response participation, it is worth mentioning the limitation of our response rate of approximately 50%. We excluded 16 survey responses that did not identify their program by name because we could not otherwise ensure avoiding the duplication of program information.

We chose not to include several potentially interesting questions in the survey, in the hopes of increasing our response rate by limiting survey length. Future work could allow respondents to detail specific topics covered in their curriculum so as to further identify areas of strengths and weaknesses in the national WM education landscape and could ascertain the availability of a WM "track."[2] Categorizing programs geographically would also have helped determine whether residency programs in more rural or isolated areas are more likely to teach WM content, given the relevance to their practice location.

Additionally, an element of our survey design may have led to underreporting of WM curricular content: The instructions for completion of the survey did not specifically clarify that the questions allowed respondents to select multiple answers, possibly leading to underreporting by programs using multiple curricula. When asking about curricular content, 1 option was "development of own curriculum." An additional free text box to describe the curriculum would have allowed for enhanced data collection.

Conclusions

WM education is rapidly growing as a regular part of emergency medicine residency training. Almost two-thirds of respondent residencies offer WM training as part of their curriculum. Four-year programs are more likely to offer WM electives but are otherwise comparable to 3-y programs. Only one-third of residencies teaching WM use one of the predefined courses as part of their teaching, suggesting there may be an opportunity for greater standardization of WM curricula.

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CASE REPORT

Medium Cut-Off Membrane Can Be a New Treatment Tool in *Amanita phalloides* Poisoning

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Mushroom poisoning is a common health problem that can be seen seasonally and geographically. Most mushroom poisoning requiring treatment worldwide is due to *Amanita phalloides*. Although liver failure and kidney injury are frequent, poisoning can also lead to more serious clinical situations, such as shock, pancreatitis, encephalopathic coma, cardiac failure, disseminated intravascular coagulation, and multiple organ dysfunction syndrome, and may cause death. In addition, when standard treatment approaches fail, extracorporeal treatment methods are often used. We report 2 cases in which hemodialysis with medium cut-off membrane was performed. We observed an improvement in liver and kidney function in both of our cases. The first case recovered, but the second case proved fatal owing to *Acinetobacter* sepsis, despite an improvement in renal function. Medium cut-off membrane hemodialysis may be an alternative option in the treatment of *Amanita phalloides* poisoning.

Keywords: mushroom poisoning, hemodialysis, dialyser, extracorporeal treatment, acute kidney injury, intoxication

Introduction

Amanita phalloides (AP) is responsible for more than 90% of mushroom poisonings resulting in death.[1] The presentation of AP poisoning can vary from subclinical status to fulminant hepatitis and death. Typically, the presentation follows a 4-stage clinical course: a symptomatic period (symptoms occur 6-8 h after oral intake); severe gastrointestinal symptoms, with nausea, vomiting and watery diarrhea, dehydration, renal injury, and electrolyte disturbances (up to the first 24 h); the development of hepatorenal syndrome and liver damage (24-48 h); and, finally, the terminal period, in which fulminant liver failure, hemorrhage, coma, and death can occur.

In the standard treatment approach, oral decontamination with activated charcoal, intravenous hydration, N-acetyl cysteine (NAC), silibinin, and penicillin are recommended. Numerous extracorporeal methods have been described in the literature, with varying results. Initiating these treatments in the first 48 h is extremely important in terms of clinical prognosis.[2] Medium cut-off (MCO) membranes are a new type of hemodialysis membranes that have been used more frequently in past decade. They have the advantage of clearing higher-molecular-weight toxins and offer beneficial effects in the management of sepsis and inflammation. It has been shown that the mortality rate may decrease by up to 9% in patients who have received intervention in the first 48 to 72 h in mushroom poisonings.[3] Despite standard treatments, most patients require liver transplantation.

Case 1

Mugla is situated in the southwest of Turkey and is a province dominated by the Mediterranean climate (37°12'54.8"N, 28°21'49.4"E). It is estimated that about 50 species of mushrooms are present in this region.

An 81-y-old local woman presented to the emergency department with nausea, vomiting, and diarrhea. She had

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Table 1. Laboratory values on admission

Values	*Case 1*	*Case 2*
Glucose (74–109 mg·dL^{-1})	172	115
Urea (16.6–48.5 mg·dL^{-1})	76.3	19.9
Na (136–145 mmol·L^{-1})	134	142
K (3.5–5.1 mmol·L^{-1})	3.8	4.43
Calcium (8.6–10 mg·dL^{-1})	7.9	7.9
Chloride (98–107 mmol·L^{-1})	92	105.6
Albumin (35–52 g·L^{-1})	31	32
GGT (6–42 U·L^{-1})	72	55
ALP (35–105 U·L^{-1})	129	86
LDH (135–214 U·L^{-1})	687	472
d-dimer (0–500 ng·mL^{-1})	882	-
Hemoglobin (11.2–19.9 g·dL^{-1})	10.2	14.2
APTT (22–34.5)	75	25.5

ALP, alkaline phosphatase; APTT, activated partial thromboplastin time; GGT, gamma-glutamyl transferase; LDH, lactate dehydrogenase.

a history of eating mushrooms she collected from the Dikmen mountain (near the village of Fadıl, at an altitude of 660 m [2165 ft] above sea level) 4 d prior. On physical examination, her blood pressure was 110/70 mm Hg, heart rate was 105 beats·min^{-1}, respiratory rate was 17 breaths·min^{-1}, and body temperature was 36.9°C. She was lethargic, and auscultation of the lungs revealed bilateral rales in the lower lung fields and bilateral 1+ pretibial edema.

On admission, laboratory findings revealed elevated hepatic enzymes and acute kidney injury (Table 1, Figures 1 and 2). When we examined the mushrooms that were collected, we found that they were an AP type. During follow-up, progression was observed in some laboratory values: aspartate aminotransferase (AST) 10564 IU·L^{-1}, alanine transaminase (ALT) 4922 IU·L^{-1}, total bilirubin 2.5 mg·dL^{-1}, direct bilirubin 1.8 mg·dL^{-1}, international normalized ratio (INR) 3.99, and activated partial thromboplastin time (APTT) 44.2 s. According to the progression and physical examination findings, the patient, who had an oliguric course, underwent hemodialysis (HD) with MCO membrane (Theranova, Baxter Healthcare, Deerfield, IL, USA) and other standard treatments (oral activated charcoal, intravenous hydration, NAC, penicillin, silymarin). The patient received a total of 17 sessions of HD, and significant improvement in clinical status and laboratory values was seen. The patient's urine output increased, and she was followed up for 22 d without additional HD. She was later removed from HD totally and discharged. On discharge, laboratory values were as follows: urea 100 mg·dL^{-1}, creatinine 2.0 mg·dL^{-1}, AST 11 IU·L^{-1}, ALT 5 IU·L^{-1}, total bilirubin 1.2 mg·dL^{-1}, direct bilirubin 0.79 mg·dL^{-1}, INR 1.12, and APTT 32 s.

Case 2

A 57-y-old woman presented to the emergency department with nausea and vomiting. The patient, who was the daughter-in-law of the first case and had been living in the same house, had consumed the same mushrooms. She presented to our hospital 2 d (5 d after eating the mushroom) after her mother-in-law's hospital admission. She had no history of chronic disease.

On admission, her blood pressure was 120/80 mm Hg, heart rate was 85 beats·min^{-1}, body temperature was 36.5°C, and respiratory rate was 13 breaths·min^{-1}. Laboratory values revealed high serum transaminase levels, but renal function tests were within normal limits (Table 1, Figures 3 and 4). On the second day of follow-up, her renal function deteriorated and she became anuric. In addition to other standard treatments (oral activated charcoal, intravenous hydration, NAC, penicillin, silymarin), HD was initiated with MCO membrane (Theranova, Baxter Healthcare). The patient was intubated on the third day.

A total of 4 sessions of HD were performed. From the fourth day of treatment, the patient's transaminases and renal function tests started to improve, and urine output started to increase. Although recovery in liver and kidney function was observed, the patient's acute-phase reactants progressed and fever developed. *Acinetobacter baumani* reproduction occurred in the tracheal aspirate culture, and antibiotherapy was modified. During follow-up, septic conditions could not be controlled, and on the seventh day of treatment she died of cardiac arrest related to septic shock.

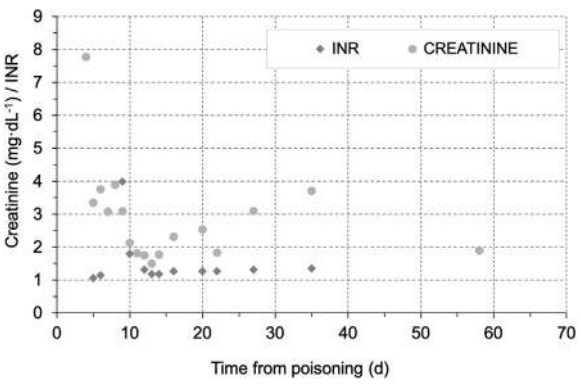

Figure 1. The course of creatinine and INR levels in case 1.

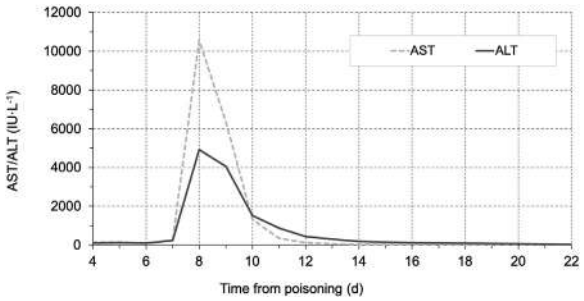

Figure 2. The course of hepatic enzymes in case 1.

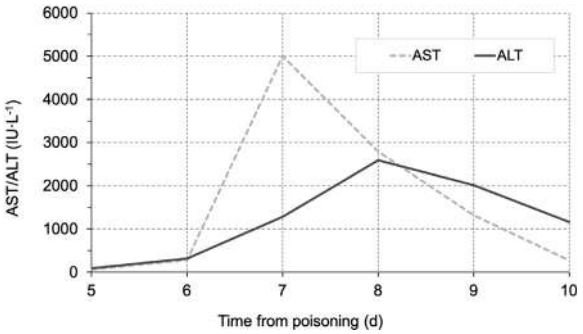

Figure 4. The course of hepatic enzymes in case 2.

Discussion

Mushroom consumption is increasing in both rural and urban areas secondary to developments in gastronomy. In particular, mushrooms consumed in rural areas can lead to fatal poisonings owing to careless and misinformed approaches to collection.

Amanitin group mushrooms are the most poisonous mushroom species and are assumed to comprise 90% of the species that cause human death globally. AP constitutes the majority of these. In a retrospective study analyzing 93 mushroom poisonings between 1990 and 2008 in Portugal, 63% were dependent on the amatoxin type and 12% ended in mortality.[4] AP contains 3 toxin groups: phallotoxins, virotoxins, and amatoxins. Amatoxins, which are cyclic octapeptides, consist of 9 different components. In the main mechanism of action of the main toxin, α amatoxin is to bound covalently to RNA polymerase II, causing transcription inhibition and leading to protein synthesis disorders. The lethal human dose of amatoxins is 0.1 to 0.3 $mg \cdot kg^{-1}$. This is a very low concentration and can often be found in a single mushroom. Amatoxin is absorbed through the gastrointestinal system; it is rapidly eliminated from the blood and disperses to the liver and kidneys within 48 h.

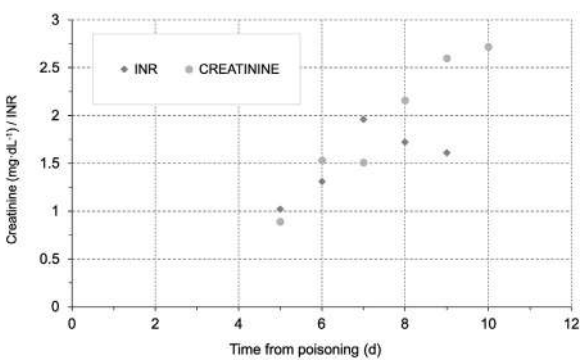

Figure 3. The course of creatinine and INR levels in case 2.

Because of this rapid absorption and distribution, it may be difficult to detect in plasma after 36 to 48 h. Approximately 60% of circulating amatoxins pass into bile and enterohepatic circulation, and the remaining 40% are cleared by the renal route. This metabolism explains the renal and hepatotoxicity commonly observed with these types of amitoxin mushroom ingestions.[5] Both of our patients consumed this type of mushroom by cooking it in boiling water, which was then drunk. The mushrooms were collected by the patients' relatives, brought to the emergency department, and later identified as AP by an expert mycologist (Figure 5).

Survival is primarily related to the degree of liver damage, but other potential complications are also very important. The major effect is due to liver necrosis (centrilobular and periportal, hemorrhage), which rapidly develops into both renal and liver dysfunction (hepatorenal syndrome). An increase in ammonia levels can lead to coma and convulsions. This may be followed by respiratory failure and hemorrhage.

In rat studies, it has been shown that concentration in renal tissue is much higher than that in liver tissue. In autopsy studies, macroscopically, cortical extravasation in the kidneys, diffuse hemorrhagic stasis, acute tubular necrosis (ATN), and tubular dense hyaline cylinders were observed histopathologically. Degenerative findings such as necrosis, vacuolization and edema, atrophy in the distal tubule, intratubular protein-rich cylinders, and thickening in the Bowman's outer capsule were found in renal biopsy studies. Experimentally, polymyxin B, by inhibiting the effect of α a-amanatin, significantly prevented the occurrence of these findings in rats and decreased mortality.[5] There were various levels of inflammatory and oxidative changes in rat studies (lymphocytic infiltrations, hydropic degeneration) and histopathologically brushy border loss as the applied dose increased, and necrotic areas have been shown to increase even more.[6]

Figure 5. *Amanita phalloides.*

In the standard treatment of mushroom poisoning, activated charcoal, penicillin-G, corticosteroid, NAC, and silibine treatments are advised to reduce absorption from the gastrointestinal tract in the acute period.[2] Because excretion is mainly through urine, force diuresis has been recommended by some studies, especially to provide renal clearance in the first 48 h. However, considering the clinical conditions that reduced renal perfusion (hepatorenal syndrome, ATN), in these particular cases, we advised against this intervention.

Regarding extracorporeal treatment options for patients with acute liver failure in AP ingestions, successful treatment with the molecular adsorbent recirculation system (MARS) and fractional plasma separation and adsorption system (FPSA) has been reported.[7,8] It was shown that MARS treatment can decrease mortality and decrease urea-creatinine levels in this patient group.[9] Positive results with FPSA also were reported.[8] A retrospective analysis of 81 patients with both liver and renal injuries secondary to AP intoxication who underwent different dialysis methods (HD, hemoperfusion [HP], plasmapheresis [PF]) found that although 16 fatal cases had ATN, none died of renal causes.[10] Again, in a study comparing patients with fatal and nonfatal intoxication, no significant difference in creatinine levels was observed between the groups.[11]

In recent decades, a new-generation membrane type called an MCO membrane has been popular owing to its new potential utilities in HD.[12] These membranes, by diffusion and convection, increase the permeability of medium-large toxins (up to 45 kDa molecular weight) owing to their higher pore sizes, allowing more and different uremic toxins to be removed. It has been reported that MCO membranes are more advantageous than high-flux HD in removal of some medium-molecular-weight toxins.[13] In recent studies of the use of MCO dialyzer in HD patients, molecules such as β2-microglobulin, light chains without kappa/lambda, complement factor D, and α1-microglobulin have been effectively removed, as well as expression of sepsis/inflammation-associated inflammatory markers such as TNF-α, mRNA-α, and IL-6 mRNA; levels of STNFR1 also have been shown to be reduced.

We intervened with emergent HD with an MCO membrane in both of our cases and successfully treated the first patient. In our second case, improvement in renal and liver function was achieved, but the patient died of sudden cardiac arrest related to septic shock. Because of the late clinical findings (at least 6–8 h) in AP poisonings, there is often a delay in hospital admission. Both of our patients delayed their presentation to our emergency department after consumption of the mushroom. However, in our first case, full resolution was achieved 48 h

after the first case, and the second case proved fatal owing to the late admission.

In a report of 2 patients with acute intoxication, 1 was treated for 72 h with continuous veno-venous hemofiltration (CVVH) and 3 plasma perfusions and the other with 100 h of CVVH and 5 doses of activated charcoal HP, and both cases fully healed.[14] Again, 2 cases who had combined therapy (HP + HD) completely recovered.[15] In a study in which 21 patients were evaluated, PF was applied to all, and mortality was nearly 5%, lower than the average in the literature.[16] In another report, beneficial effects of the combined use of extracorporeal therapies were emphasized. In this report, the effectiveness of FPSA and PF was demonstrated; continuous renal replacement therapy was used only to treat uremic status and elevated ammonia.[2] Many extracorporeal toxin removal treatments (HD, HP, PF) appear to be effective, especially in the first 48 h of exposure. The effectiveness of systems such as MARS and FPSA has been demonstrated in AP poisoning, especially in liver failure. However, an important disadvantage is that these types of interventions can only be performed at major tertiary care centers, and as the highest clinical efficacy is within the first hour after ingestion, this level of care cannot be applied in every center.

Mushroom poisoning is a common and possibly fatal health problem. It is extremely important to diagnose early and begin treatment as soon as possible, especially because early HD has been reported to significantly reduce mortality resulting from liver and kidney failure.[17] There are currently no specific antidotes or guidelines for treatment, so starting extracorporeal treatment methods as early as possible in addition to standard treatments can be considered as a method that reduces mortality and morbidity. Since the 1960s and 1970s, dialysis modalities have been tried in AP poisoning. Although the treatment of AP ingestion with extracorporeal methods seems rational, the most effective modality has not yet been clarified. Considering that special extracorporeal detoxification methods can be performed in a tertiary or quaternary care center, it is very important to start treatment in the first 24 to 48 h, until transport is provided. Additionally, we recommend that the HD MCO membrane be considered as a life-saving treatment modality for these types of mushroom poisonings. To our knowledge, these 2 cases represent the first documentation in the medical literature describing HD with MCO membrane in acute AP mushroom poisoning.

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CASE REPORT

Skin Necrosis, Diffuse Urticaria, and Cellulitis Due to Presumed *Loxosceles* Spider Bite

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The clinical manifestations of a recluse spider bite range from local erythema to necrotic skin reactions; bites rarely lead to a systemic disease known as viscerocutaneous loxoscelism. A 29-y-old female patient was admitted to the emergency department with a wound, swelling, and pain on her left leg and a rash on her whole body as a result of a spider bite. On physical examination, a round, hard, black, irregularly shaped necrotic area was found in the bite zone on the lower posterior part of the left thigh, as were lesions in the form of erythematous papules around the area. There was a color change around the lesion, extending from posterior to medial of the thigh, and a papule on a diffuse erythematous surface on the trunk and arms. At follow-up, the necrotic area had become more apparent. After approximately 1 mo, the necrotic area was surgically debrided and a flap was formed on the necrotic tissue area. In this article, a case that was thought to be caused by a *Loxosceles* spider bite and which started with erythema, progressed to lymphangitis, cellulitis, and severe necrosis, and was surgically debrided, was evaluated in light of the clinical findings and previously reported cases of verified loxoscelism. In patients with a history suggestive of a bite and with these clinical findings, the diagnosis of a bite by *Loxosceles* spp. should be carefully considered based on clinical and epidemiologic findings.

Keywords: presumed spider bite, emergency medicine, dermonecrosis, surgical intervention

Introduction

Although various *Loxosceles* species are common in the United States, very few recluse spider bite cases have been reported in the literature in Turkey and Europe.[1-6] *Loxosceles reclusa*, known as the brown recluse spider, is the most common and medically significant species in the United States.[7] The Mediterranean recluse spider *Loxosceles rufescens* is common throughout the Mediterranean region and has been accidentally introduced in numerous countries outside of its natural range (eg, Australia).[1-4] The color of these spiders ranges from gray to orange-red, pale brown, and dusk. There is usually a violin-shaped marking on the dorsum of the cephalothorax.[5,7,8] The clinical manifestations of the bites range from local erythema to necrotic skin reactions (known as necrotic arachnidism), and bites rarely lead to a systemic disease known as loxoscelism (causing myalgia, arthralgia, hemolysis, hemoglobinuria, acute renal failure, amputation, and death).[2,3,5,9,10]

Although a possible diagnosis of spider bite can be made based on the patient's medical history and physical examination findings, a definitive diagnosis can only be made by qualified verification of the species. Otherwise, the diagnosis of *Loxosceles* spider bite should be considered after other causes have been ruled out. History, clinical findings, and diagnostic tools can be used to rule out conditions that may cause skin necrosis. Bites and stings can cause skin necrosis.[9,10] As a result of usually not being able to recognize a spider at the time of the bite, the diagnosis can often be overlooked by clinicians and occasionally mislabeled as a skin infection or cellulitis.[11]

Cosmetic disorders resulting from presumed spider bite and dermonecrosis requiring surgical intervention are not commonly encountered, especially in young female patients. This study is presented to report a

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presumed loxoscelism case, which is extremely rare in Turkey, and to highlight dermonecrosis that started with erythema, leading to cellulitis, lymphangitis, and diffuse urticaria and which was treated surgically (flap).

CASE REPORT

A 29-y-old female patient who had no history of illness was admitted to the emergency department (ED) with a wound, swelling, and pain in the leg and a rash on the whole body as a result of a spider bite. In her medical history, the patient reported that she had been bitten by a brown spider on the upper part of her left knee 6 d earlier (Figure 1); 1 d after the bite, she had developed pain, blistering, and numbness on the back of the left thigh and a rash on her skin. The patient tried to treat the lesion with an application of cold compresses, analgesia (dexketoprofen tablet as a nonsteroidal anti-inflammatory drug), topical antibiotic cream, and oral antibiotics (cefuroxime 500 mg tablet) for a few days; however, she indicated that the lesion became gradually larger, the middle of it became black, the redness around the wound started to expand in the form of a ring, and the rash spread to her whole body.

The patient presented to our ED upon the progression of her symptoms. At the time of presentation, the patient's general condition was good; she was conscious, oriented, and cooperative, and her Glasgow Coma Score was 15. Vital signs (blood pressure 104/85 mm Hg, pulse 86 beats·min^{-1}, body temperature 37.2°C, and respiratory rate 22 breaths·min^{-1}) and oxygen saturation (95%) were normal.

On physical examination, in the bite zone on the lower posterior part of the left thigh, there was a round, hard, black, and irregularly shaped necrotic area with a diameter of 3 cm that was painful when touched, as well as lesions in the form of erythematous papules/plaques around the area. There was a color change (possibly lymphangitis) to a lighter color (brown) around the lesion, extending from the posterior to medial aspect of the thigh and a papule on a diffuse erythematous surface on the trunk and arms. Papule, plaques, and redness were detected around the lesion as well as on the arms and trunk. We interpreted them as urticaria (Figure 2A, B, C). The examination of other systems was unremarkable.

In the analysis of laboratory findings, white blood cell count was $6.25\times10^{3}\cdot\mu L^{-1}$, hemoglobin (Hb) was 14.1 g·dL^{-1}, platelets $207\times103\cdot\mu L^{-1}$, glucose was 131 mg·dL^{-1}, C-reactive protein was 8 mg·dL^{-1}, sedimentation rate was 4 mm·h^{-1}, and electrocardiography was evaluated as normal. By these findings, necrotic arachnidism, lymphangitis, urticaria, and cellulitis caused by spider bite were considered.

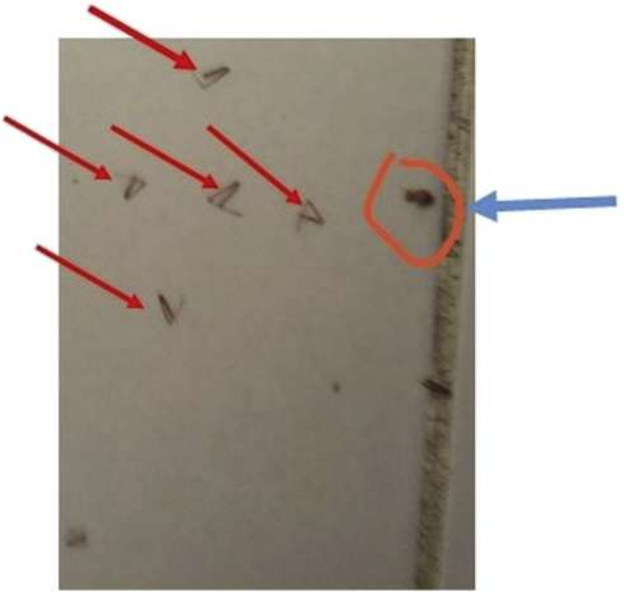

Figure 1. Body and feet of the spider that was killed. (Figure displayed is 6 cm wide; 1000% magnification.)

Hydration, tetanus vaccine, 45 mg pheniramine hydrogen maleate ampoule, and 60 mg methylprednisolone ampoule (intravenous) were administered in the ED, and the patient was referred to the dermatology service for follow-up. In the dermatology department, where she was hospitalized, pheniramine hydrogen maleate 45 mg (for 2 d), intravenous methylprednisolone 40 mg (given for 8 d), and, with the recommendation of the infectious diseases department, ampicillin/sulbactam 1.5 g (intravenous, given every 6 h for 10 d) were started. On soft tissue ultrasonography, thickening in the subcutaneous soft tissue planes in the area lateral to the left thigh and an increase in heterogeneity and echogenicity were observed, and an image that may be compatible with cellulitis in the posterior-posterolateral subcutaneous tissue on the level of the middle third of the diaphysis of the left femur was detected on magnetic resonance imaging.

The patient was hospitalized and followed for 10 d. On the 10th day, a decrease in the diameter of the bite zone and regression of erythema was observed, and the necrotic lesion in the bite zone was persistent. The patient was discharged with a recommendation for outpatient follow-up. During the follow-up, at 19 d after the bite, skin necrosis and ring-shaped erythema continued in the same area; necrosis became more apparent on the 33rd day (Figure 3A and B). The necrotic area was debrided through plastic surgery and a flap was implanted in that

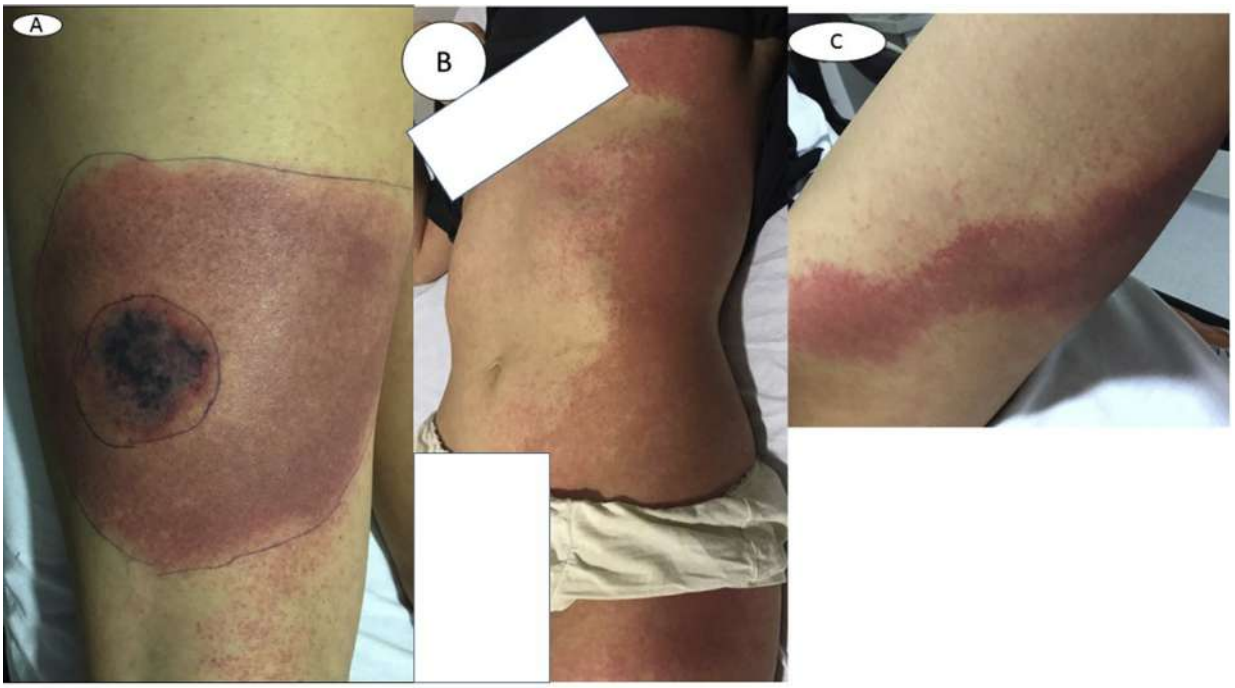

Figure 2. Physical examination findings are associated with a bite at the time of admission to the emergency department. (A) Skin necrosis in the bite zone and surrounding hyperemia, rash. (B) Skin eruption that spreads upward from the bite zone, cellulitis. (C) Hyperemia extending from the bite to a certain area (possible lymphangitis).

area. Follow-up and treatment of the patient were continued.

Discussion

In recent years, few cases of *Loxosceles* bite and associated clinical toxicity have been reported from Turkey. *L rufescens* was recorded in several regions in Turkey.[2,3,12,13] However, in Turkey, in 2 cases of reported spider bites, *Loxosceles* venom was detected in the patient's skin with enzyme immunosorbent assay and supported the diagnosis of *Loxosceles* bite.[6]

The verified identity of the spider is a key issue in suspected spider bites, and presumed identity is a common feature in reported loxoscelism. Identifying the spider itself is considered the only certain way to diagnose a *Loxosceles* bite. The characteristic appearance of many recluse spiders is an important clue.[9] The spider assigned responsibility for the bite was not precisely identified by an expert, but the spider's color and appearance (Figure 1), the clinical history of the bite, and its progression (as evidenced by the provided photographic timeline) are typical for the recluse spider. In our case, the lesion gradually enlarged, the patient had severe pain in the leg and difficulty in walking, and the wound on the skin ulcerated and turned into a necrotic lesion with large halo-shaped hyperemia around it and a black center (Figures 2 and 3). The patient herself could define the spider clearly and because she killed the spider, the remaining parts also supported this recollection.

However, there are important clues in the differential diagnosis to rule out other conditions that may cause dermonecrosis. Other factors accordant with the clinical presentation of a recluse spider bite are seasonality and location of the bite, as well as accompanying cutaneous conditions. Diagnostic criteria for brown recluse spider bites have undergone many changes. For that reason, revised diagnostic criteria have been defined.[14,15] The original diagnostic criteria[14] have been revised as a 4-level classification (putative, presumptive, probable, documented) of the probability of brown recluse spider bite, taking into account characteristics of the geographic location, physical examination features, and seasonality.[15,16] A "documented" diagnosis includes ≥1 positive examination finding and a brown recluse spider identified by an arachnologist. Geographical location of the putative bite being in the known habitat of the brown recluse spider, the lesion having ≥1 positive examination characteristic, and no characteristics indicating an alternative diagnosis (negative examination characteristics) are defined as probable. Using the revised Sams criteria, it

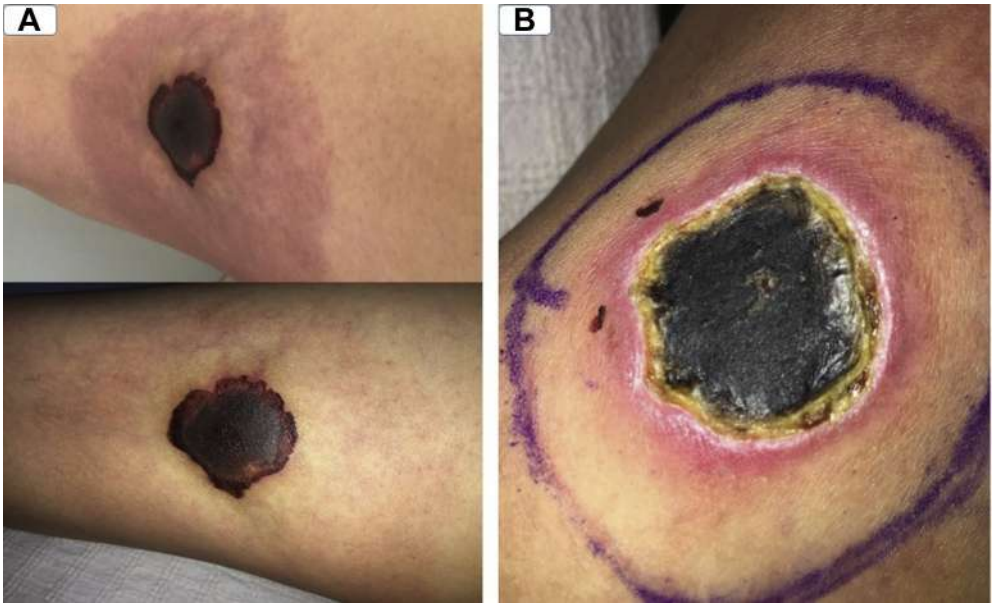

Figure 3. (A) Nineteen days after the bite and (B) on the 33rd day, physical examination findings of the patient and the appearance of the lesion.

was determined that 43 of 45 "probable and documented" brown recluse spider bites occurred between April and October.[15,16] Our case may conform to the definition of probable brown recluse spider bite according to the previously described 4-level classification of the probability of spider bite.[15] Furthermore, the patient lacked negative examination findings to suggest an alternative diagnosis, and the bite occurred in May, which increased the probability of a diagnosis.

Recluse spider bite is characterized by systemic symptoms known as dermonecrosis and loxoscelism. Sphingomyelinase-D activity in *Loxosceles* venom is responsible for hemorrhagic and necrotic lesions.[17] The bite of *L rufescens* can cause a clinical picture known as cutaneous loxoscelism. A typical bite exhibits a characteristic pattern with itching, pain, and erythema within 6 h and an irregular, erythematous ring that limits the bite in 24 h. Typical bite wound progression begins with blistering followed by color changes, ulceration, and necrotic eschar. These skin findings were observed in our patient.[5,9,12,18] Within 48 h, central necrosis may occur, starting as an area of central cyanosis that may form a blister and ulcerate. These findings give a typical blue-white-red concentric pattern to the lesion: erythema (red) around a pale area (white) of peripheral vasoconstriction with central necrosis or ecchymosis (blue). This describes the red, white, and blue color change sign associated with *L rufescens* bites in Europe,[3-6] which is generally observed in the early period (2–3 d). However, in our study, the patient presented after 6 d. Therefore, the red and white color can be seen clearly and the blue discoloration mark is not clear (more dark purple) (Figures 2 and 3).

Although skin lesions and necrosis due to brown recluse spider bite have been reported, diffuse skin lesions along with necrotic lesions, cellulitis, and lymphangitis are rarely defined. In the case series, cellulitis and skin necrosis were the most common indications of local loxoscelism.[11] Our patient did not have neutrophils and elevation in white blood cell count and C-reactive protein. This may be due to the use of antibiotics by the patient. In the literature, the progression of skin necrosis due to *Loxosceles* spider bites are usually similar. In the previously reported case,[3] the progression stages of erythema, plaque, and necrosis after the bite seem similar to our case, although the temporal progression may not be exactly matched. We think that there may be some differences depending on the case and the medical history that was taken. As mentioned previously, systemic envenoming occasionally occurs after *Loxosceles* bites; however, it did not develop in our patient, and only local effects develop in most envenomed patients.

The wound typically heals within 3 wk without leaving a permanent scar; however, 20% of bites may develop a necrotic ulcer that will remain symptomatic for a long time.[10,19] It is also stated that only 10 to 15% of bites cause "major problems," defined as unacceptable scarring, hospitalization, or chronic lesions.[18] In our case, the wound was surgically debrided, and a flap was performed on the necrotic area.

There are many causes of dermonecrotic lesions. Bacterial and fungal infections, drug reactions, ecthyma gangrenosum, pyoderma gangrenosum, necrotizing fasciitis, thromboembolic phenomenon, focal vasculitis, Lyme disease, neoplasms, and chemical burns should be considered in the differential diagnosis. Other arthropods can inflict dermonecrotic lesions that are accompanied by significant color changes, edema, surrounding plaques, and cellulitis, especially centipedes and millipedes, and some infections due to ticks and mites.[9,12,14,20-22] A study in Chile suggested that only 45% of presumed spider bites were inflicted by arthropods (17% linked to *Loxosceles*), with 44% of cases resulting from infection and 11% caused by physical or chemical agents.[23]

History, clinical findings, and diagnostic tools can be used to exclude conditions that may cause skin necrosis. The medical history of the patient is the most important determinant, and physical examination findings and clinicians' experience may help eliminate more common causes of necrotic skin lesions, such as bacterial and fungal infections and diabetic and pressure ulcers. It has been suggested that, in circumstances in which a spider is not captured close to the site of injury at the time that the envenomation occurred, the term "probable arthropod envenomation" be used if other medical causes of "necrotic arachnidism" have been excluded.[24] In our case, the fact that the patient clearly described the spider bite (Figure 1), even if not fully, confirms that it was a spider bite. The patient did not describe any other possible arthropods (ticks, millipedes, or mites). Confirmatory diagnostic testing techniques for *Loxosceles* envenomation have been developed[25] but are not widely available for clinical use.

Management of recluse spider bites is initially elevation, immobilization, tetanus prophylaxis, local wound care with antiseptic dressing, and supportive treatment with painkillers. Mild bites and itchy urticaria lesions can be treated symptomatically with antihistamines. It has been suggested that infected bites with tissue destruction, such as cellulitis, should be treated prophylactically with antibiotics.[9,26,27] Other alternatives in the treatment of *Loxosceles* spider bite include conservative wound management, hyperbaric oxygen, dapsone, glucocorticoids, vasodilators, and antivenin. These alternatives can be costly, painful, and/or toxic.[3,5,10] None of these treatments have been proven to be effective in patients' recovery. Most bites are self-limiting and heal without intervention.[3,5,10] Analgesic, antihistaminic, steroid, and antibiotic treatments were given to our patient because of itching, diffuse urticaria lesions, cellulitis, and skin necrosis. The clinical findings disappeared in 3 wk, except for skin necrosis. Skin necrosis was surgically debrided and the flap was performed on the necrotic area.

LIMITATIONS

The fact that the spider type was not precisely identified by an expert is an important limitation in this case. This can be described as a presumed brown recluse spider case. *Loxosceles* spider bite was considered the most likely diagnosis based on the patient's precise description of being bitten by a spider and on the seasonality of the spider bite, the geographic region where the bite occurred, clinical and physical examination findings, and diagnostic criteria, without the capture and identification of the spider by an entomologist. Poisoning by another arthropod or member of the family *Sicariidae* would be less likely. The patient did not describe any other possible arthropods (ticks, millipedes, or mites).

Conclusions

We report a case of envenoming due to a presumed *Loxosceles* spider bite, which is rarely seen in Turkey. We present this case to describe the typical features of the presumed *Loxosceles* spider bite and to highlight the unusually early and rapid progression of the disease in the patient. Symptoms can start with erythema and cause complications such as cellulitis, lymphangitis, and diffuse urticaria. Especially in a young patient, it may result in cosmetic defects and dermonecrosis treated surgically (flap). Such patients may need long-term follow-up in terms of dermonecrosis, and surgical intervention may be required. In patients with a history suggestive of a bite and with these clinical findings, the diagnosis of a bite by *Loxosceles* spp. should be carefully considered based on clinical and epidemiologic findings. Nevertheless, physicians should consider the far more common causes of skin ulcers and dermonecrosis before considering loxoscelism.

Author Contributions: Acquisition of case data and analysis of case data (AK, EA); photo credits (AK, EA); study concept and design (AK, EA, SBB); drafting the manuscript (AK, SBB); critical revision of manuscript (AK, SBB, AY); approval of final manuscript (AK, EA, SBB, AY).

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CASE REPORT

Point-of-Care Ultrasound Diagnosis of Acute High Altitude Illness: A Case Report

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With the advent of high-quality portable ultrasound machines, point-of-care ultrasound (POCUS) has gained interest as a promising diagnostic tool for patients with high altitude illness. Although POCUS is used successfully in hospital environments to detect interstitial pulmonary edema and increased intracranial pressure, the relationship between specific sonographic criteria and high altitude illness is still unclear. We report the case of a healthy 32-y-old male who developed acute respiratory distress and neurologic impairment at 4321 m while participating in a high altitude medical research expedition. We discuss the potential of POCUS to diagnose acute high altitude illness by lung ultrasound, optic nerve sheath diameter measurement, and echocardiography. Ultrasound in combination with clinical findings helped us to exclude relevant differential diagnoses, start on-site treatment, and organize an evacuation. We used serial clinical and ultrasound examinations to assess the patient over time. Although its role in high altitude medicine needs further investigation, we believe that POCUS can be a valuable tool to aid clinical decision-making in remote, high altitude environments.

Keywords: high altitude pulmonary edema, high altitude cerebral edema, acclimatization, ataxia

Introduction

Acute high altitude illness includes acute mountain sickness (AMS), high altitude pulmonary edema (HAPE), and high altitude cerebral edema (HACE). These illnesses are caused by hypobaric hypoxia. They can develop within a few days after ascent to altitudes above 2500 m. In rare cases, individuals can develop symptoms at elevations as low as 2000 m.[1] Although AMS and HACE usually develop relatively quickly after arrival at altitude, with the onset of symptoms within 4 to 12 h for AMS and within 24 h for HACE, HAPE usually develops within 1 to 5 d. The risk of developing AMS and HACE increases with altitude, rate of ascent, and degree of acclimatization. HACE rarely occurs at altitudes below 4000 m. The prevalence of HACE between 4200 and 5500 m is estimated to be 0.5 to 1%.[2] It can occur at lower altitudes, however, especially if concomitant with HAPE.[3] The risk of HAPE increases with rapid ascent, higher altitude, respiratory infection, history of HAPE, male sex, lower temperature, and physical exertion.[4]

Field diagnosis of high altitude illness is traditionally made with clinical signs and symptoms, field tests such as tandem gait, and the use of basic tools such as lung auscultation and pulse oximetry. With the development of high-quality portable ultrasound machines, point-of-care ultrasound (POCUS) has gained interest as a promising diagnostic tool for patients with high altitude illness. POCUS is ultrasonography used in combination with physical examination to search for specific pathologic findings and diagnose problems wherever a patient is being treated. It is easily repeatable if the patient's condition changes and is therefore useful for monitoring disease progression and treatment efficacy.[5]

In a hospital, point-of-care lung ultrasound (LUS) can be used to diagnose interstitial pulmonary edema by the presence of 3 or more B-lines in 2 or more bilateral

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intercostal spaces.[6] The relationship between the presence of B-lines and HAPE is not yet clearly understood. Although an increased number of B-lines is also a common finding in healthy individuals ascending to high altitude, patients clinically diagnosed with HAPE have a significantly greater number of B-lines than healthy controls.[7,8] The number of B-lines in healthy individuals has an inverse relationship with arterial oxygen saturation. The presence of B-lines in these individuals might be a manifestation of subclinical pulmonary edema.[8-11]

Serial optic nerve sheath diameter (ONSD) measurements have shown promise in sedated patients in intensive care units when invasive measurement of intracranial pressure (ICP) is not indicated or is unavailable. There is no universally accepted cutoff value above which ONSD can be considered abnormal. In intensive care medicine, measurements above 5 mm correspond to elevations in ICP above 20 mm Hg.[12] A meta-analysis showed a high degree of variation among studies, in which sensitivity and specificity ranged from 88 to 95% and 74 to 91%, respectively. The ONSD threshold values that optimized sensitivity and specificity ranged from 4.8 mm to 6.3 mm.[13] Although early reports indicated a positive correlation between AMS and increasing ONSD values,[14,15] later studies failed to reproduce these results. Instead, they showed an altitude-related increase in ONSD that seems to be independent of AMS symptoms and disappears after descent to lower altitudes.[16-19]

There is limited data available on the use of point-of-care echocardiography at high altitude. HAPE is a noncardiogenic type of pulmonary edema. Pulmonary artery pressure (PAP) increases significantly in healthy individuals ascending to high altitude. Although there is no correlation between PAP and the number of B-lines in healthy individuals, patients with HAPE have both elevated PAPs and high numbers of B-lines.[9] In healthy individuals at high altitude, left ventricular (LV) function remains normal and cardiac output increases with faster heart rates. With increased PAP and increased afterload, there is increased right ventricular (RV) strain and a decreased myocardial performance index. It is not known whether this is an adaptive mechanism to hypoxia or a pathologic response in susceptible individuals.[10]

We describe the case of a healthy 32-y-old male who developed acute respiratory distress and neurologic impairment while participating in a high altitude medical research expedition to the Khumbu valley of Nepal. We discuss the use of POCUS to diagnose acute high altitude illness using LUS, ONSD measurement, and echocardiography. We believe this case illustrates the possibilities and limitations of POCUS in the evaluation of acute high altitude illness. We hope to inspire further research.

Case Report

The patient was a member of a trekking group that had started in Lukla (2860 m) and was following a carefully planned ascent profile to Island Peak (Figure 1). Every evening, each participant completed a medical questionnaire and underwent clinical examination by a physician. During the examination, participants were evaluated for sonographic signs of high altitude illness using a handheld ultrasound machine (Sonosite iViz) to measure B-lines and ONSD. LUS was performed using a modified protocol with 4 chest areas per side instead of the usual 8, to limit examination time in a cold, high altitude environment.[6] LUS findings were recorded for the upper anterior and basal lateral chest areas of each side.

The patient's symptoms began on the evening of Day 6 at Phortse (3840 m) with a minor headache. His vital signs and the POCUS examination were normal. He had no B-lines on LUS and had an ONSD of 6 mm, comparable to his previous ONSD, starting at 2800 m. He was feeling better on Day 7. He continued trekking at a high pace and was also seen running up to different viewpoints along the trail to take pictures. That evening in Dingboche (4321 m), his blood pressure was 142/92 mm Hg, his heart rate was 72 beats·min^{-1}, his respiratory rate was 22 breaths·min^{-1}, and his SpO_2 was 89%. The normal range of SpO_2 at 4300 m is 85 to 95%.[20] On examination, he was alert, oriented, and showed no signs of ataxia with tandem gait. Lung auscultation was normal, with absence of inspiratory crackles. LUS, however, showed 1 to 2 B-lines in 4 of the 4 lung regions (Figure 2). The ONSD measurement remained unchanged at 6 mm (Figure 3).

The patient became increasingly dyspneic during the night and developed severe orthopnea in the early morning hours of Day 8. At breakfast, blood pressure was 144/104 mm Hg, heart rate was 90 beats·min^{-1}, respiratory rate was 34 breaths·min^{-1}, and SpO_2 was 78%. Lung auscultation was normal. He did not have a cough or a fever. He was mildly ataxic with tandem gait but had normal mental status without other neurologic findings. On LUS, the number of B-lines in all regions had increased compared to the previous evening. There were still no more than 2 B-lines in any region. There were no sonographic signs of pneumothorax or pneumonia. The ONSD had increased to 7 mm. This abrupt increase in ONSD coincided with the development of the patient's symptoms and was higher than the reported normal values. We interpreted it as a possible sign of early HACE (Figure 4).

To investigate pulmonary embolism (PE) as a potential cause of the clinical deterioration, we performed point-of-care echocardiography (parasternal long axis,

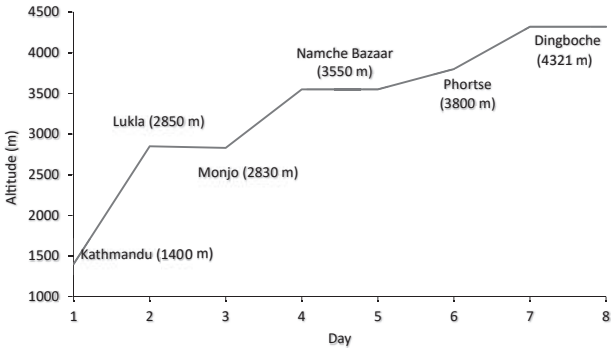

Figure 1. Ascent profile of the patient.

Figure 2. Lung ultrasound on the evening of Day 7.

parasternal short axis, and apical 4-chamber view). These views showed a slightly enlarged RV with an RV/LV ratio greater than 0.6, which allowed us to exclude a large, central PE. Although we could not exclude peripheral PE, the patient did not have typical symptoms such chest pain or a cough, which made us question peripheral PE as a differential diagnosis. We interpreted the RV enlargement as an indirect sign of pulmonary hypertension that was most consistent with HAPE (Figure 5).

The patient's rapid clinical deterioration together with the dynamic changes on POCUS prompted us to evacuate him by helicopter to a hospital in Kathmandu (1400 m). We gave the patient 20 mg of nifedipine by mouth. The patient remained in a seated position. His condition did not deteriorate while he was waiting for the helicopter to arrive. He did not receive oxygen because it was not available at our lodge or any of the neighboring lodges. Because the evacuation took place within 3 h of diagnosis, we did not contact the Himalayan Rescue Association aid post in Pheriche for treatment with a portable hyperbaric chamber. A dose of oral dexamethasone was discussed by the expedition leaders but was not administered.

The availability of real-time ultrasound images helped the patient, a physician himself, to understand and accept the decision to evacuate. Although shared decision-making is not always possible or appropriate in emergency situations, showing the dynamic changes on ultrasound was helpful in convincing our patient that descent was the appropriate course of action. Shortly after admission to the hospital in Kathmandu, a chest x-ray and transthoracic echocardiography were performed. The chest x-ray showed normal lungs with no signs of consolidation, interstitial pulmonary edema, pneumothorax, or pleural effusion (Figure 6). According to the hospital report, the transthoracic echocardiography showed no signs of pulmonary artery hypertension. The patient was discharged on the evening of the same Day. Three days after the evacuation, the patient developed a cough, muscle aches, and fever, most likely due to a respiratory infection. These symptoms subsided within 5 d with symptomatic treatment and without antibiotics. One month later, back home in Switzerland, the patient was mountaineering at altitudes up to 3000 m without any issues.

Discussion

The patient developed symptoms of HAPE and HACE during the first night after ascending to a new higher altitude and after performing a high level of physical exertion. Considering the severity of his respiratory symptoms, the number of B-lines seen was lower than expected and not enough to diagnose HAPE using the established sonographic criterion for interstitial pulmonary edema of 3 or more B-lines per intercostal space.[6] The same observation was reported in another study, in which B-lines were seen in healthy individuals ascending to high altitude. In 1 clinically diagnosed case of HAPE, the LUS findings did not meet the sonographic criteria for interstitial pulmonary edema.[11] As in our patient, the researchers noted an increase in B-lines that coincided with the onset of the patient's symptoms.

The relationship between the presence of B-lines and HAPE is not yet clear. When performing POCUS, it is recommended to perform repeated ultrasound assessments.[5,21] In this way, the patient's own images can serve as a control and help physicians to correlate dynamic changes on ultrasound with the development of new clinical symptoms.[22] For example, a sudden increase in B-lines in a patient with exertional dyspnea could prompt

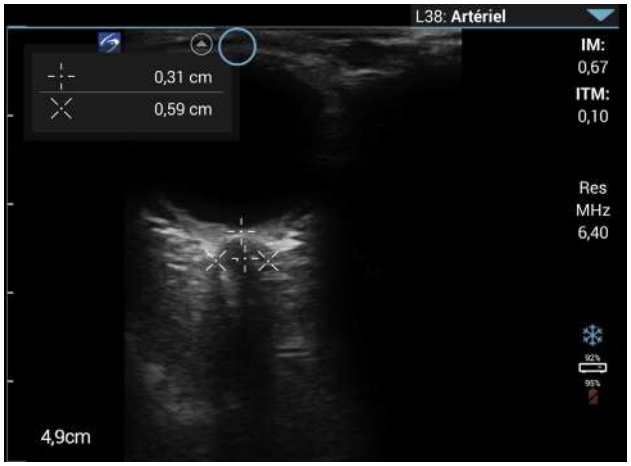

Figure 3. Optic nerve sheath diameter measurement on the evening of Day 7.

Figure 4. Optic nerve sheath diameter measurement on the morning of Day 8.

medical providers to recommend an extra layover day, followed by a slower ascent profile. If the patient develops clinical HAPE, ultrasound might be helpful in monitoring the effectiveness of treatment,[21] especially if the patient cannot be evacuated. A decreasing number of B-lines after treatment with nifedipine can provide additional information to show that the patient's condition is improving. Ultrasound monitoring of the course of HAPE has not been studied. It should be used to complement but not replace other clinical parameters, such as S_pO_2, to support clinical decision-making.

Because the exact ONSD cutoff value for predicting increased ICP and for the diagnosis of AMS and HACE is currently unknown, we believe that ONSD measurements should also be compared over time and not interpreted based on single absolute values. The sonographic images should always be correlated with clinical findings.[22] Because our patient developed mild ataxia in conjunction with an abrupt increase in ONSD, we interpreted these changes as a possible sonographic sign of early HACE.

We did not administer a dose of oral dexamethasone because we were able to rapidly evacuate the patient by helicopter. However, helicopter rescue services can be unreliable, and it is potentially dangerous to delay emergency treatment even if evacuation is imminent. Especially in remote, high altitude areas, helicopters can be subject to mechanical issues and delay due to changing weather conditions. In our case, care for the patient should have been improved by starting emergency treatment for HACE in the field. It would also have been prudent to obtain a portable hyperbaric chamber or oxygen while waiting for the helicopter. Patients with early signs and symptoms of HACE can deteriorate rapidly and develop a life-threatening illness.

A limitation of our examination protocol is that we only examined 4 of the usual 8 chest areas on each side.[6] It is therefore possible that we missed intercostal spaces with 3 or more B-lines, which could have facilitated the diagnosis of HAPE. Another limitation is that echocardiography was not part of the standard patient evaluation protocol. We only performed this examination on the morning before the evacuation. It therefore represents a single point value that we could not compare to previous measurements. We cannot say whether RV enlargement was a new development. It might have developed earlier when the patient initially arrived at high altitude.

A general limitation of ultrasound is that special precautions need to be taken to ensure machines work in cold, high altitude environments. Batteries can discharge quickly under these circumstances and can be difficult to recharge, even in highly developed trekking areas such as the Khumbu valley. Batteries should be protected from cold temperatures, and the need for alternative charging methods should be anticipated. We found that power was available in most guesthouses up to Namche Bazaar. At higher altitudes, we successfully charged our equipment with a combination of solar panels and external batteries.

Despite the limitations, we found that our ultrasound findings helped us to rule out several relevant differential diagnoses in the field. The absence of sonographic signs of pneumothorax and pneumonia, for example, made them unlikely causes of the patient's respiratory symptoms. In case of central PE, we would have expected a significantly higher RV/LV ratio (>1:1). Even if the rapid

Figure 5. Apical 4-chamber view on the morning of Day 8.

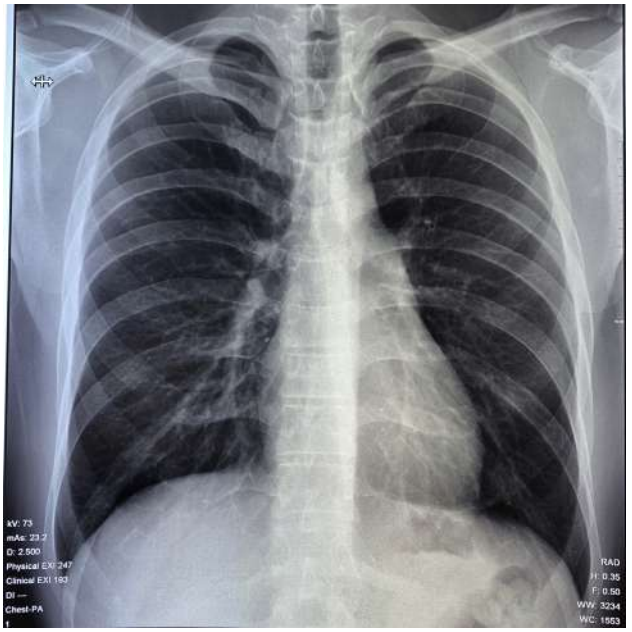

Figure 6. Chest x-ray after evacuation on Day 8.

resolution of symptoms and normalization of clinical findings at lower altitudes is most consistent with HAPE, we cannot rule out peripheral PE based on our findings and the further investigations done by the hospital in Kathmandu. With the reduction of pulmonary hypertension by nifedipine and the increase in partial pressure of alveolar oxygen by evacuation to a lower altitude, the symptoms caused by peripheral PE could also have improved.

The normal chest x-ray in Kathmandu could be explained by chest radiographic abnormalities that can lag behind the clinical signs of HAPE. Through timely treatment with nifedipine and descent to a lower altitude, it is also possible that sufficient interstitial fluid was reabsorbed by the time the patient arrived in Kathmandu and had the x-ray taken. Because the patient developed symptoms of an upper respiratory infection shortly after evacuation, it is possible that the respiratory infection had begun at high altitude. Nasal congestion may worsen hypoxemia, especially at night when excessive oxygen desaturation is common during episodes of periodic breathing.[23]

Conclusion

The development of high-quality, portable ultrasound machines has increased interest in the use of POCUS in high altitude medicine. The association between B-lines on LUS and the presence of HAPE is still unclear and needs further investigation. The correlation of increased ONSD with AMS and HACE is also still uncertain. Considering these limitations, there remains a need for clinical decision-making based on history and physical examination using basic tools such as auscultation and pulse oximetry. POCUS can, however, provide additional information to exclude relevant differential diagnoses and complement clinical findings with dynamic changes on ultrasound. When using POCUS, it is recommended to perform repeated ultrasound assessments. This allows medical providers to interpret sonographic changes over time and integrate these changes with clinical findings. We believe that POCUS can be a valuable tool for clinical decision-making in remote, high altitude environments.

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Author Contributions: Acquisition of data (FW, NH, JL, AB, MBM, DM); analysis of the data (FW, NH, DM); drafting of the manuscript (FW, NH); critical revision of the manuscript (FW, NH, JL, MBM, DM); approval of the final manuscript (FW, NH, JL, AB, MBM, DM).

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Disclosures: None.

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CASE REPORT

Kounis Syndrome Following *Hypnale zara* (Hump-Nosed Pit Viper) Bite in Sri Lanka

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A 47-y-old man was bitten by a hump-nosed viper *(Hypnale zara)* and gradually developed retrosternal chest pain associated with ST segment elevation on electrocardiogram. He had normal troponin I levels and no evidence of coagulopathy. Initially, he was managed as having anterior ST elevation myocardial infarction with thrombolysis. Later, because troponin levels were normal, he was suggested to have the type I variant of Kounis syndrome (allergic coronary artery spasm). This was supported by high eosinophil counts in peripheral blood. He was successfully managed with supportive treatment and discharged 6 d after the snakebite. Cardiac complications are rarely reported after hump-nosed viper bites, and clinical reports of coronary vasospasm after snakebites are extremely rare in the literature. This is the first known report of Kounis syndrome after a hump-nosed viper bite.

Keywords: snakebites, coronary vasospasm, electrocardiogram, cardiotoxicity, myocardial infarction

Introduction

Kounis syndrome is hypersensitivity of coronary vessels induced by drugs, snake venom, environmental exposures, foods, and coronary stents. Vasospastic allergic angina, allergic myocardial infarction, and stent thrombosis with occluding thrombus infiltrated by eosinophils are the 3 reported variants of this syndrome.[1] The type I variant includes either coronary artery spasm without increased cardiac enzymes and troponins or coronary artery spasm progressing to acute myocardial infarction with raised cardiac enzymes and troponins. Vasospastic angina is a form of angina pectoris caused by vasospasm of coronary arteries, in which chest pain is accompanied by a transient ST segment elevation on electrocardiogram (ECG).[2] It is sometimes misdiagnosed as ST elevation myocardial infarction (STEMI), and it is well known that snake venom causes allergic coronary artery vasospasm.[3,4]

Hump-nosed vipers are the most common cause of venomous snakebites in Sri Lanka, causing 27 to 77% of all snakebites.[5] They mostly cause local envenoming only[6,7] and less frequently cause significant morbidities such as acute kidney injury, chronic kidney disease,[8] venom-induced consumption coagulopathy,[9] thrombotic microangiopathy,[10] chronic wounds, and amputations of digits.[6] The genus *Hypnale* comprises 3 species: *H hypnale*, *H zara,* and *H nepa*. The former is found in both Sri Lanka and Southwestern India, and the latter 2 are endemic to Sri Lanka. Of these 3 species, *H hypnale* is responsible for the most bites (80%) because it is frequently encountered all over the island except in the peninsula of Jaffna in the north. Cardiac complications such as myocardial infarction, atrial fibrillation, ischemic changes of myocardium, and cardiac arrest after *Hypnale* bites are rarely reported,[11,12] although a fatal case of cardiac arrest and atrial fibrillation after *H hypnale* has been reported.[12] Such manifestations of *H zara* envenoming have not been described. An extensive literature search confirms that there is no previous report of

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Figure 1. Necrosis of left fourth finger (circled) on day 2 after snakebite.

coronary vasospasm caused by hump-nosed viper bites. Therefore, we report the first case of coronary vasospasm after *H zara* bite in Sri Lanka.

Case Report

A 47-y-old previously healthy tea estate worker was bitten by a hump-nosed viper on his left fourth finger at about 1400 while he was working in a tea estate. He lives in an estate situated Udaniriella, a remote village in Ratnapura district. He is a nonsmoker and does not consume alcohol. His family history is not significant for heart disease. He was not given any native treatments and was admitted to the hospital at 1545. The snake was identified by the admitting medical officer as a hump-nosed viper.

On admission, he had severe local pain, mild swelling over the finger, and necrosis at the site of bite (Figure 1), but there was no local bleeding. His blood pressure was 110/80 mm Hg and his pulse rate was 80 beats·min^{-1}. Respiratory rate was 18 breaths·min^{-1}, and oxygen saturation on room air was 99% detected using pulse oximetry. Examination of the respiratory system, nervous system, and abdomen revealed no abnormality. His 20-min whole blood clotting test (WBCT20) on admission and then at 6-hour intervals for 24 h was negative (<20 min). The other laboratory findings were white blood cells (WBC) 9.8×10^{3}·μL^{-1}, neutrophils 4280 μL^{-1} (44% of whole WBC count), eosinophils 3000 μL^{-1} (31% of whole WBC count), lymphocytes 2080 μL^{-1} (21% of

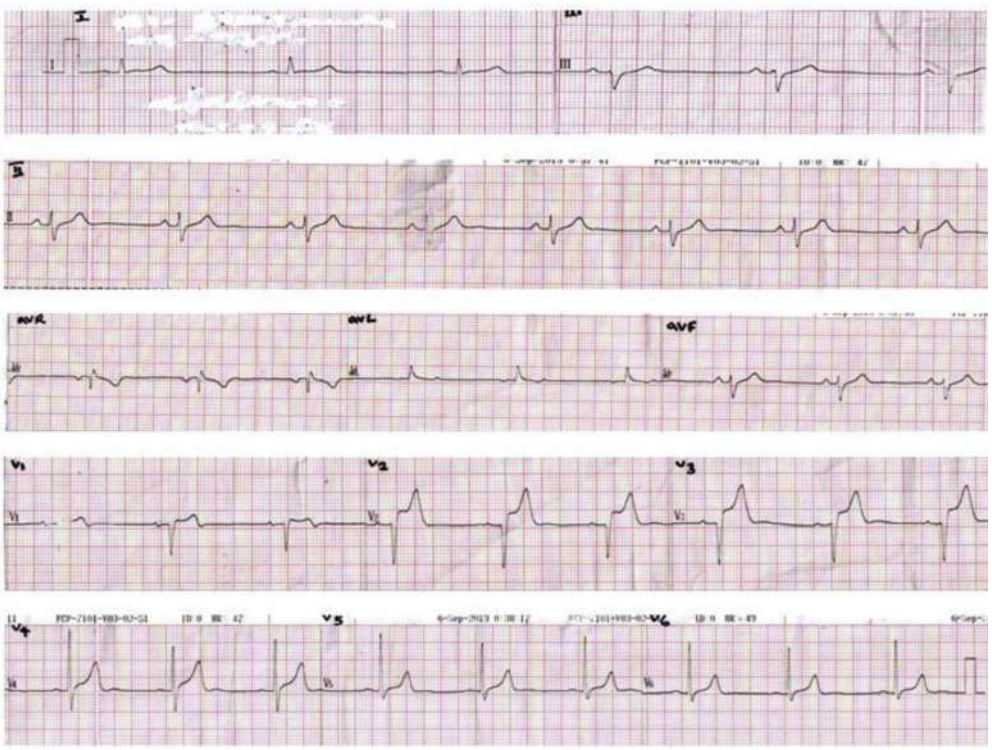

Figure 2. Electrocardiogram changes 10 h after snakebite: ST segment elevation in chest leads (V_1-V_5).

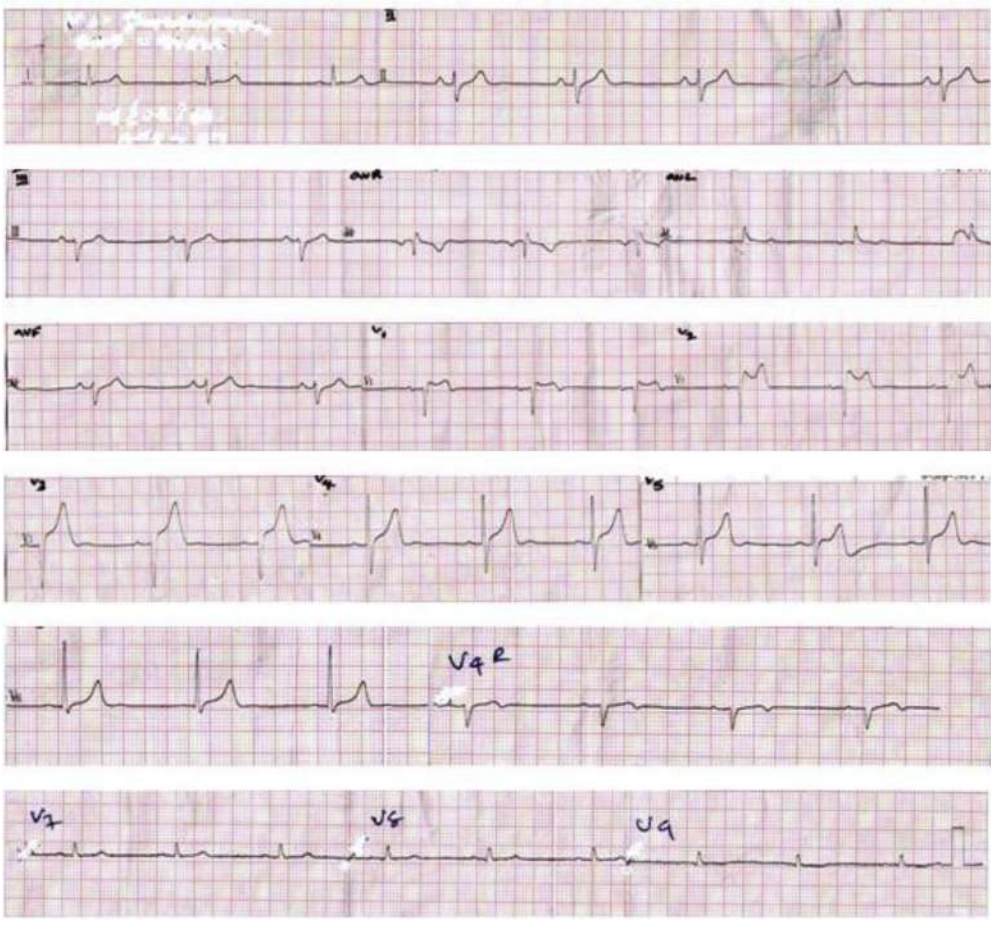

Figure 3. Progressive changes on electrocardiogram 12 h after snakebite: ST segment elevation in chest leads (V_1-V_5).

whole WBC count), platelets $280\times10^3\cdot\mu L^{-1}$, Hb 13.1 $g\cdot dL^{-1}$, international normal ratio 1.12, and activated partial thromboplastin time 38/32 sec. The biochemical laboratory findings were normal.

Around 10 h after the snakebite, the patient reported central retrosternal, nonradiating chest pain, and ECG showed ST segment elevation on V_1-V_5 (Figure 2). Repeat ECG of 1 h also had progressive changes (Figure 3). At this time, his blood pressure was 100 mmHg and pulse rate was 50 $beats\cdot min^{-1}$. The patient was transferred to the coronary care unit for further management, and thrombolysis was done using tenecteplase. Subcutaneous enoxaparin 50 mg twice daily, aspirin 75 mg, clopidogrel 75 mg, and atrovastatin 40 mg were started with cardiac monitoring. The troponin I level (6 h after the onset of chest pain) was 0.012 $ng\cdot mL^{-1}$ (normal <0.034). The 2D-echocardiogram showed anterior and apical wall hypokinesia with preserved left ventricular muscle mass with an ejection fraction of 50%. There was no apical ballooning. Because his repeat troponin I level was 0.01 $ng\cdot mL^{-1}$ (24 h after the first), enoxaparin, aspirin, and clopidogrel were omitted, and the atrovastatin dose was reduced to 20 mg.

During the hospital stay, the patient's urine output was normal and wound debridement was done for the finger (site of bite). He was discharged on hospital day 6 on atrovastatin with arrangements for clinic follow-up. His follow-up ECG (done on day 20 after snakebite) was normal (Figure 4). His fasting blood sugar and lipid profile (done 1 mo after snakebite) were normal. Three months after the snakebite, his exercise ECG was also normal (Figure 5). According to the standard key, the hump-nosed viper was identified as *H zara* (Figure 6).

Discussion

Deficient basal release of nitric oxide due to endothelial dysfunction and enhanced vascular smooth muscle contractility with the involvement of the Rho/Rho-kinase

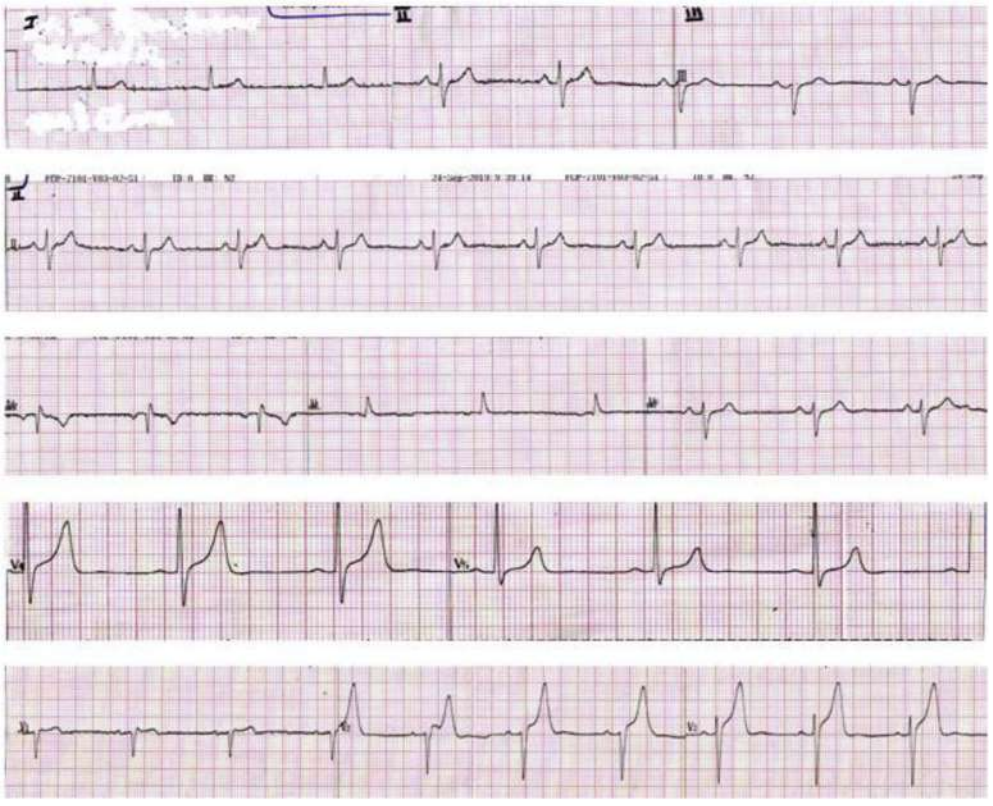

Figure 4. Normal electrocardiogram 20 d after snakebite.

pathway play important roles in the pathogenesis of coronary artery spasm.[13] In Kounis syndrome, however, coronary vasospasm is caused by inflammatory mediators such as histamine, chymase, and tryptase released from mast cells as the allergic response. Although we had no facilities to test serum histamine or tryptase levels, evidence points to the coronary vasospasm of our patient as being due to an allergic reaction to snake venom because he had elevated eosinophil counts in peripheral blood (3000 μL^{-1}, normal range 20-500 μL^{-1}).

In ECG, the ST segment represents the interval between depolarization and repolarization of the ventricles. ST elevation is significant when it is found on 2 contiguous leads in a 12-lead ECG. However, about 93% of healthy young men have ST elevation of 1 to 3 mm in 1 or more precordial leads, mostly in V_2, and the ST segment is concave.[14] Despite its high sensitivity, ST segment deviation has poor specificity because, in addition to myocardial infarction, there are other cardiac and noncardiac causes for ST elevation. The cardiac causes are early repolarization, left ventricular hypertrophy, hypertrophic cardiomyopathy, left bundle branch block, pericarditis, myocarditis, aortic dissection, Takotsubo cardiomyopathy, Brugada syndrome, and vasospastic angina. The noncardiac causes are hyperkalaemia, pulmonary thromboembolism, pneumothorax, acute pancreatitis, acute cholecystitis, hemorrhagic cerebrovascular disease, and drug-induced ST elevation.[15] Snake venom is a good source of vasoactive substances, and envenomation can enhance the activity of vasoactive mediators.[16] This may cause coronary artery spasms resulting in ST elevation on ECG. Eosinophilic coronary periarteritis, which typically presents as vasospastic angina, is a rare localized vasculitis and is almost always associated with sudden cardiac death. Diagnosis is almost exclusively found at autopsy.[17] Acute allergic myocarditis is another possibility, but it has fatal outcomes.

H zara is recorded in lowland rainforests of Sri Lanka and therefore is called the lowland hump-nosed viper. Among systemic effects, acute kidney injury, coagulopathy, microangiopathic haemolysis, and thrombocytopenia have previously been reported after *H zara* bites.[7,18-20] Its venom is complex and causes thrombotic microangiopathy[20] or even death.[18] In Sri Lanka, cardiotoxicity is mainly observed in Russell's viper (*Daboia russelii*) bites, which have fatal outcomes[21]; such effects with *Hypnale* bites are very rare. However, by using troponin T and ECGs, 1

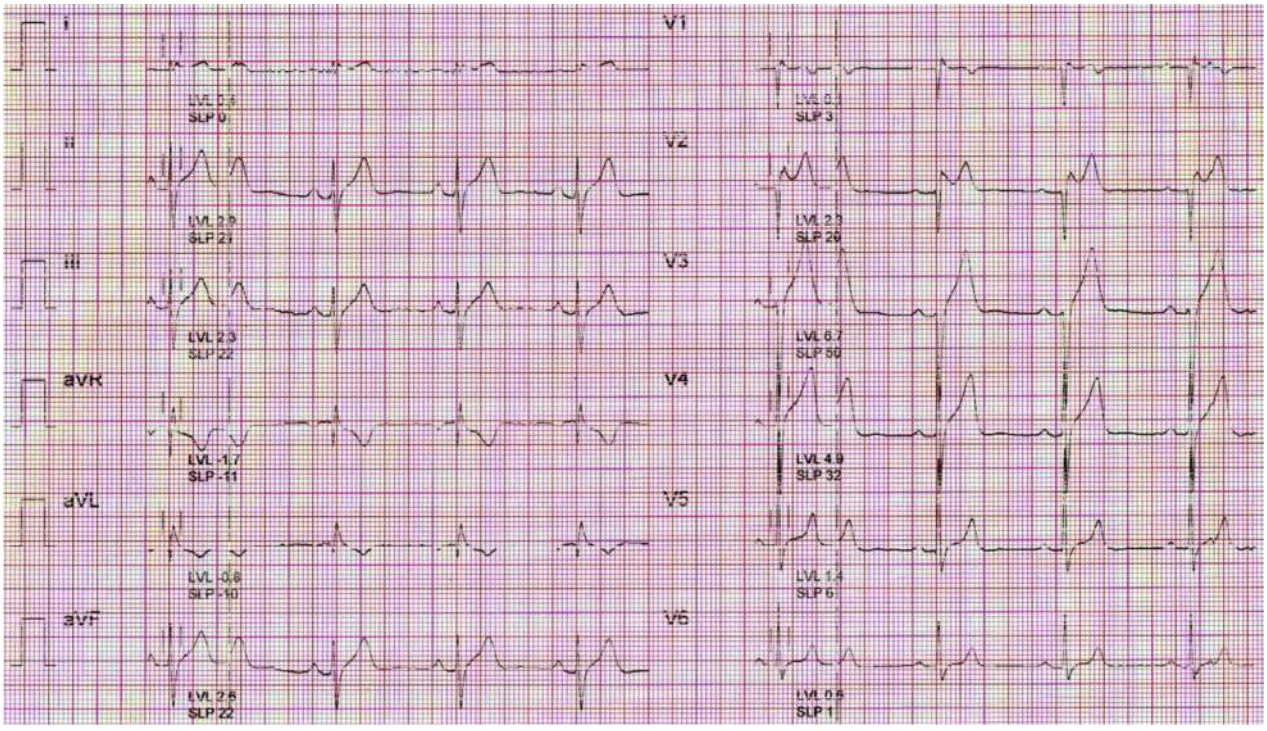

Figure 5. Normal exercise electrocardiogram done 3 mo after the snakebite.

study concluded that myocardial damage does not seem to be an important feature of hump-nosed and Russell's viper bites.[22]

One of the main investigations needed in the management of this patient is the coronary angiogram, but there are no facilities in our center for angiography or percutaneous coronary interventions. In our patient, the initial working diagnosis was anterior STEMI; therefore, immediately thrombolysis was done. However, ST elevation was not corrected and continued for more than 12 h with normal troponin I levels. With actual thrombosis of coronary vessels, troponin should be elevated owing to reduced blood

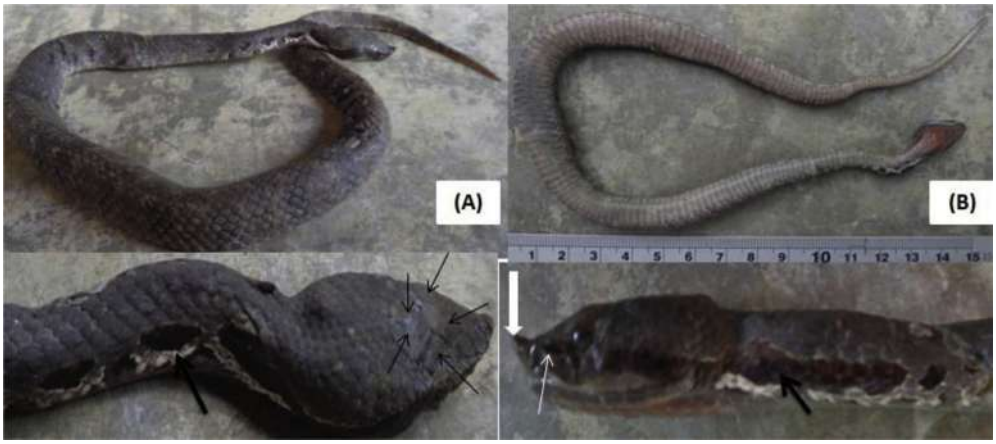

Figure 6. Species of hump-nosed viper responsible for the bite: *Hypnale zara*—a male snake whose total length is 342 mm, head length 15 mm, tail length 56 mm, and snout to vent length 288 mm, ventrals 146, subcaudals 45, and mid dorsal rows 15, from Ratnapura, Sri Lanka (06°40'N 80°24'E, elevation 130 m [430 ft]). (A) Dorsolateral view. (B) Ventral view. Note that thin black arrows indicate large 5 head scales (characteristic feature of hump-nosed vipers), the thick black arrow indicates color bands on the neck of *H zara* (characteristic feature of *H zara*), the thin white arrow indicates the loreal pit (characteristic feature of pit vipers), and the thick white arrow indicates the prominent raised hump (characteristic feature of *H zara*).

supply to cardiac muscle, or ST elevation might be corrected when tenecteplase is administered. Therefore, the possibility of vasospastic angina was suggested later, and thrombolytic agents were omitted. It was difficult to differentiate vasospastic angina from STEMI until the troponin I levels were available; therefore, thrombolysis was performed immediately after reviewing the ECG and treating the patient for STEMI prior to receiving other laboratory results. Thus, thrombolysis was done unnecessarily for this patient because snake venom may cause both vasospastic angina and myocardial infarction, both of which result from transmural ischemia of the heart muscle. However, almost 11% of patients with suspected acute coronary syndrome receive unnecessary thrombolytic therapy.[23]

The management of this patient was further complicated because there is no antivenom currently available in Sri Lanka for hump-nosed viper bites. However, our patient is a previously healthy nonsmoker or nonalcoholic and has no family history of heart disease. His body mass index was in the normal range (23.1 $kg \cdot m^{-2}$). The fasting blood sugar and lipid profile were also normal. Considering all of the available circumstantial evidence, it can be concluded that he had no risk factors for heart disease. *Hypnale* venom is procoagulant, which is hypothesized as being due to thrombin-like enzymes,[24] and may cause thrombosis of coronary vessels resulting in acute myocardial infarction.[11,12] In the absence of elevation of troponin levels, however, this can be excluded, and we have to think of nonischemic cardiac causes for ST elevations for which the best explanation is snake venom causing coronary vasospasms. Normal exercise ECG conveys that the patient has no mismatch between myocardial oxygen delivery by coronary blood flow and myocardial oxygen demand.

This case highlights the complex nature of *Hypnale* envenoming that may rarely include Kounis syndrome and emphasizes the need for the production of specific antivenom for *Hypnale* spp.

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Author Contributions: Patient management involvement and literature search (RMMKNR, PEANR); snake identification and getting its morphological features (RMMKNR); drafting and writing the case history (RMMKNR, PEANR, SAMK); review and approval of the final manuscript (RMMKNR, PEANR, SAMK).

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CASE REPORT

Acute Bilateral Fixed Mydriasis Caused by Lupini Bean Intoxication

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Lupini beans are legume seeds of the genus *Lupinus*, consumed in many parts of the world. The main species are *Lupinus mutabilis, Lupius angustifolius,* and *Lupinus albus.* The latter is commonly eaten as a snack in Mediterranean countries. The beans are very rich in alkaloids, which give them a bitter taste. One of these alkaloids was shown to cause anticholinergic effects. Lupini beans, if improperly prepared, can cause toxicity manifesting as an anticholinergic syndrome. We present the case of a 50-y-old woman who presented with bilateral mydriasis, mouth dryness, and anxiety. We confirmed that the patient consumed partially debittered lupini beans a few hours before presentation. The rest of her physical and ophthalmic examination results were within normal limits. Her symptoms resolved without therapy within 12 h from presentation and were attributed to ingestion of incorrectly prepared lupine seeds.

Keywords: lupini seeds, anticholinergic syndrome, pupillary abnormalities, ophthalmology, toxicity

Introduction

Lupini beans are the yellow seeds of the Lupinus plant (Figure 1). They are commonly consumed in the Middle East and in Southern Europe. In the Middle East, they are known as turmus and are usually ingested as a snack or an appetizer.[1] They are also very nutritious and high in protein. Lupini beans are extremely bitter and contain more than 150 quinolizidine alkaloids. Lupanine, the most prevalent alkaloid in lupini seeds, is associated with high anticholinergic effects.[2]

Lupini poisoning occurs as a result of consuming incorrectly prepared lupini beans. Insufficient soaking allows an important amount of the anticholinergic alkaloids to remain in the beans. To avoid this toxicity, the seeds should be "debittered" before consumption. Debittering involves methodical rinsing for several days, which eliminates the toxic alkaloids and makes them more satisfactory for human consumption by reducing their associated bitterness.[3]

To the best of our knowledge, only a limited number of adverse events associated with the ingestion of incorrectly prepared lupini beans have been reported.

Case Report

A 50-y-old Lebanese woman presented to the emergency department with acute onset of bilateral nonreactive mydriasis. She reported dryness of her mouth and eyes. Although she was very anxious, we found no positive findings on review of her medical history. She had not been medicated and had no known allergies. Her last ophthalmic examination was 8 mo before presentation and was within normal limits. Before this incident, she had been healthy. She denied any recent drug use or head injury, and no focal neurologic findings were noted on physical examination. She reported that her husband was experiencing similar symptoms, except for pupillary dilatation, and reported stomach pain and general malaise.

Upon further questioning regarding suspicious food intake, she, along with her spouse, noted having consumed

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Figure 1. Lupinus plant: Iake Tekapo and Lupine flower in New Zealand. Shutterstock photo ID:1602625402. Republished with permission.

an excessive amount of lupini beans (Figure 2) hours before presentation. She emphasized the bitterness of the seeds and explained that she had not soaked them long enough before consumption. She reported the onset of her current symptoms shortly after ingestion of the seeds.

On ophthalmic examination, her visual acuity was 20/20 and her intraocular pressure was 12 mm Hg bilaterally. On slit lamp test, no significant findings were noted except for some corneal dryness with a short tear film break-up time and few punctate epithelial erosions. The mydriasis (Figure 3) was completely nonreactive, and her pupillary light reflex (direct and consensual) and accommodation-convergence reflex were abnormal. All extraocular movements were intact. Her vital signs were also normal (blood pressure: 120/80 mm Hg, heart rate: 88 beats·min^{-1}, temperature: 37.2°C, respiratory rate: 14 breath·min^{-1}).

Figure 2. Lupini beans.

Figure 3. Bilateral fixed mydriasis.

The improperly prepared lupini beans were determined to be the cause of her anticholinergic symptoms, including bilateral mydriasis, dry mouth, and anxiety. When other potential causes of intoxication were ruled out and the patient was stabilized, she was discharged home and was asked to return the next day for follow-up. Her symptoms gradually resolved within 12 h of presentation with no therapy or intervention.

Discussion

Anticholinergic agents block the action of the neurotransmitter acetylcholine. They inhibit the transmission of parasympathetic nerve impulses, leading to reduced contraction of smooth muscles.[4] Anticholinergic toxicity may affect the nervous, circulatory, and digestive systems in humans. Symptoms include vital signs disturbances (tachycardia, hypertension, hyperthermia), confusion, anxiety, dizziness, malaise, flushed face, tremors, slurred speech, dry mouth, urinary retention, stomach pain, and constipation.[5]

Ophthalmologically, they may cause dilated and unresponsive pupils, dry eyes, and decreased accommodation; moreover, in susceptible individuals, they can increase the risk of or worsen closed-angle glaucoma.[6] Pupillary abnormalities usually cause a diagnostic challenge in the emergency department because of the wide associated differential diagnosis. Establishing a correct diagnosis, after taking a thorough history, is imperative because etiology often dictates therapy and prognosis.[7]

Sudden-onset mydriasis should prompt a careful evaluation of the patient's history and recent medication and substance intake. Anticholinergic agents are found in numerous drugs such as antihistamines, antitussives, anticonvulsants, and antidepressants as well as in a wide variety of plants including *Atropa belladonna* and *Datura stramonium*. Intoxication in general is not uncommon, whereas intoxication specifically caused by lupini beans remains rare.[8,9]

To our knowledge, 7 similar case reports have been published to date.[3,10-15] In all of the cases, patients

presented with manifestations of anticholinergic toxicity, but presenting symptoms varied among the publications. In all other published cases, the symptoms were more numerous and more pronounced compared to our patient (eg, lid drop, palpitations, respiratory distress, photophobia, nausea, and vomiting), but they all shared 1 symptom: bilateral mydriasis. Our case is the one with the fewest symptoms, which made our diagnosis more challenging.

In one of the cases, a 48-y-old man with osteoarthritis presented with anticholinergic syndrome after having ingested lupini beans for their presumed role as an analgesic and antidiabetic, prompting a careful consideration of these seeds' role in homeopathic medicine.[16] Another case reported in Lebanon, similar to our case, involved 2 patients presenting with intoxication after consuming excessive amounts of bitter lupini seeds. In fact, yellow lupini seeds (turmus) are frequently used as a snack in Lebanon.[12] For this reason, it is important to raise awareness concerning the toxic potential of lupini beans, as well as the necessity of proper handling and soaking, especially in countries where their consumption is common.

In all the reported cases we found, the duration of the symptoms varied from 12 to 48 h.[17] Compared with other drug toxicity, the symptoms can last longer and usually depend on the time needed for clearance of the ingested drug from the body.

Ultimately, diagnosis of anticholinergic syndrome caused by lupini beans is clinical and highly dependent on adequate history-taking. No laboratory workup is required, but vital signs should be recorded and monitored. Patients should remain under clinical observation until symptoms resolve, usually within 24 h from presentation. In cases of severe anticholinergic toxicity, treatment is mainly supportive. Benzodiazepines such as diazepam or lorazepam can be given intravenously (IV) for tachycardia, agitation, and delirium. If these symptoms are refractory to standard doses of benzodiazepines, the antidote physostigmine should be considered (0.5–2 mg slow IV). Contraindications to physostigmine administration include seizures, bradycardia, A-V block, QRS widening, or QTC interval prolongation on electrocardiogram. Oral activated charcoal may be given early in the clinical course (1–2 h post-ingestion), but only if the patient is alert and cooperative. There is no proven role for gastric lavage with these types of ingestions.[18] Prognosis is generally favorable, as was the case of our patient, whose symptoms resolved in less than 12 h.[19]

We agree with the recommendations that include adequate handling and soaking of lupini beans; they should be soaked in water for 12 h, then cooked, and finally rinsed for about 30 s, 3 times per day for 5 d before ingestion.[20]

Conclusions

Obtaining a detailed medical history is imperative in cases of pupillary abnormalities. When an anticholinergic syndrome is suspected, intoxication by not only drugs but also plants should be considered. Recent intake of lupini beans should prompt consideration of these seeds as a possible etiology. Lupini intoxication and its prevention are important, especially in areas where this food is commonly consumed. Presentation of this toxicity can vary, and bilateral mydriasis can sometimes be the only presenting symptom. Awareness of this potential cause can assist the physician with the diagnosis and help guide appropriate management.

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CASE REPORT

Venom-Induced Consumption Coagulopathy Unresponsive to Antivenom After *Echis carinatus sochureki* Envenoming

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Snakebite envenoming is a serious and life-threatening but neglected problem in the tropics. The focus in the Indian subcontinent is usually on the Indian cobra (*Naja naja*), common krait (*Bungarus caeruleus*), Russell's viper (*Daboia russelii*), and Indian saw-scaled viper (*Echis carinatus*). The Indian polyvalent antivenom contains hyperimmunized horse antibodies against only these 4 species. However, regional intraspecific variations are important in viper envenomings, leading to marked differences in clinical presentation and response to the available polyvalent antivenom. *Echis carinatus sochureki*, a subspecies of *Echis carinatus*, has been linked to serious morbidity in the Thar Desert regions of Rajasthan, although consistent reports are lacking. We report a patient with prolonged venom-induced consumption coagulopathy owing to *Echis carinatus sochureki* envenoming who did not respond to Indian polyvalent antivenom in Jodhpur, India. Features of local and hemotoxic envenoming resolved after a week with supportive care. *Echis sochureki* venom has been shown to be different from *Echis carinatus* in terms of composition and in vitro neutralization by antivenom. Clinicians in the tropical desert regions must suspect *Echis sochureki* envenoming in the setting of nonresponsiveness to Indian polyvalent antivenom. This will help optimize antivenom use in these patients, preventing potentially life-threatening antivenom associated reactions. Because the usefulness of Indian polyvalent antivenom appears to be limited in this setting, there is an urgent need to advocate for region-specific antivenom or monovalent antivenom for this area.

Keywords: snakebite, saw-scaled viper, arid desert

Introduction

Snakebite envenoming is a serious and neglected tropical problem causing considerable mortality and morbidity.[1] In 2019, the World Health Organization launched a global strategy for the prevention and control of snakebite envenoming, which aims to halve deaths and disability by 2030. Prevention and effective first response are the central themes of this program.[2]

The focus in the Indian subcontinent is usually on 4 common venomous snakes: Indian cobra (*Naja naja*), India krait (*Bungarus caeruleus*), Russell's viper (*Daboia russelii*), and Indian saw-scaled viper (*Echis carinatus*).[3] The Indian polyvalent antivenom is widely used in this region and is available as a freeze-dried powder. It is immunoglobulin purified from the plasma of horses hyperimmunized with the venom of these 4 snakes. Each milliliter after reconstitution neutralizes not less than 0.60 mg of *Naja naja*, 0.45 mg of *Bungarus caeruleus*, 0.60 mg of *Daboia russelii,* and 0.45 mg of *Echis carinatus* venom.[4] The efficacy of the antivenom depends on the venom used in manufacturing it.[5] Most of the venom collected for antivenom production in India comes from a small geographic area through the Irula cooperative society, Tamil Nadu, India.[4]

However, apart from these 4 species, several other medically significant venomous snakes are found in India, and there is significant regional intraspecific variation among snakes found in geographically distinct regions, resulting in definite differences in venom composition,

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clinical features, and degree of neutralization by the polyvalent antivenom.[4,6-9] This appears to be especially important for *Echis carinatus*, common throughout the Indian subcontinent, and its subspecies *Echis sochureki*, which has a geographical distribution including the neighboring regions of Pakistan, Iran, and the Arabian Peninsula.[5,10] *Echis sochureki* has been linked to serious morbidity in the Thar Desert regions of Rajasthan, Western India, although consistent reports are lacking.[4,11] We describe a patient with *Echis sochureki* envenoming who did not respond to the polyvalent antivenom.

Case report

A 60-y-old farmer from the Barmer district of the Thar Desert region of Western Rajasthan, India, sustained a snakebite on his right foot when he went to feed his cows just outside his home in the evening in September 2019. His family members who were nearby caught and killed the snake. The family took a photograph of the snake specimen and discarded the snake. He developed swelling and bleeding at the bite site (Figure 1A) and reported severe headache.

He was transported to a nearby district hospital by a hired vehicle within an hour of bite, and he did not receive any native treatments before this. Ten vials of Indian polyvalent antivenom were given at the district hospital. He was then referred to our emergency department, a tertiary care teaching hospital, 6 h after the bite.

A whole blood clotting test was performed using 2 mL of freshly sampled venous blood in a dry test tube left undisturbed for 20 min at ambient temperature. Unclotted blood after tilting once at the end of 20 min was considered positive. Because the test was positive at 6 h, 10 vials of antivenom (Premium Serums and Vaccines Private Limited, Nagpur, India; batch number 28A104) was administered (Figure 1B). A single loose tourniquet tied just above the wound was removed slowly at presentation while antivenom was being given. A further 10 vials of antivenom were administered because the whole blood clotting test was persistently uncoagulable after the next 6 h. The coagulation profile revealed prolonged prothrombin time and activated partial thromboplastin time (testing performed with Sysmex CA1500 Hemostasis System and Thromorel S kit—reagent and control plasma from Siemens Healthcare Diagnostics). These remained prolonged (>120 s, limit of detection) until 7 d after the bite. Local bleeding subsided in 24 h. Platelet counts and renal function were normal throughout.

The photograph of the snake taken by the patient's family and the patient's unresponsiveness to polyvalent antivenom strongly suggested *Echis carinatus sochureki* envenoming. The photograph was identified as *Echis carinatus sochureki* by a herpetologist (Figure 1C). The patient received supportive care. No transfusions were required. Coagulation parameters normalized completely by day 10 (Table 1).

Discussion

Saw-scaled viper envenoming presents with local tissue swelling, vascular involvement, bleeding manifestations, and rarely with atypical features of renal dysfunction and thrombotic microangiopathy leading to considerable morbidity and mortality.[4,12] A recent nationally representative snakebite mortality study classified Rajasthan state as a high-burden region.[1] Regional intraspecific variation appears to be especially important in vipers, leading to different clinical manifestations. Severe envenoming from *Echis sochureki* has been reported from the United States and Eastern Europe in snake handlers.[8,9] Despite its widespread distribution in Northern India, Pakistan, Afghanistan, Iran, and the Arabian Peninsula, *Echis sochureki* has received considerably less attention in comparison to the other common subspecies, *Echis carinatus*. A series of 12 patients with *Echis sochureki* envenoming causing serious morbidity was reported from the Thar Desert region of Rajasthan in 2007. This report described persistent coagulopathy and use of large doses of Indian polyvalent antivenom.[11]

Prolonged venom-induced consumptive coagulopathy (VICC) with poor or partial response to polyvalent antivenom has been a prominent feature in various reports of *Echis sochureki* envenoming.[11,13-15] This unresponsiveness to antivenom and the length of the snake (>30 cm) as assessed from the photograph of the snake brought by the patient's relatives were key points in suspecting *Echis sochureki* envenoming. Although the photograph was useful for snake identification in our case, it is pertinent to note that any handling of the snake by the family or killing of offending snakes must be strongly discouraged because it places those individuals at risk for bites and does not support snake conservation efforts from an ecologic perspective.

Our patient experienced no clinical bleeding, but the persistent coagulation abnormalities were a cause for concern. Local bleeding with prolonged persistent VICC in most of the reported cases suggests that apart from poor neutralization by polyvalent antivenom, the pharmacokinetics of *Echis sochureki* venom may differ from other vipers. Several studies examining venomics

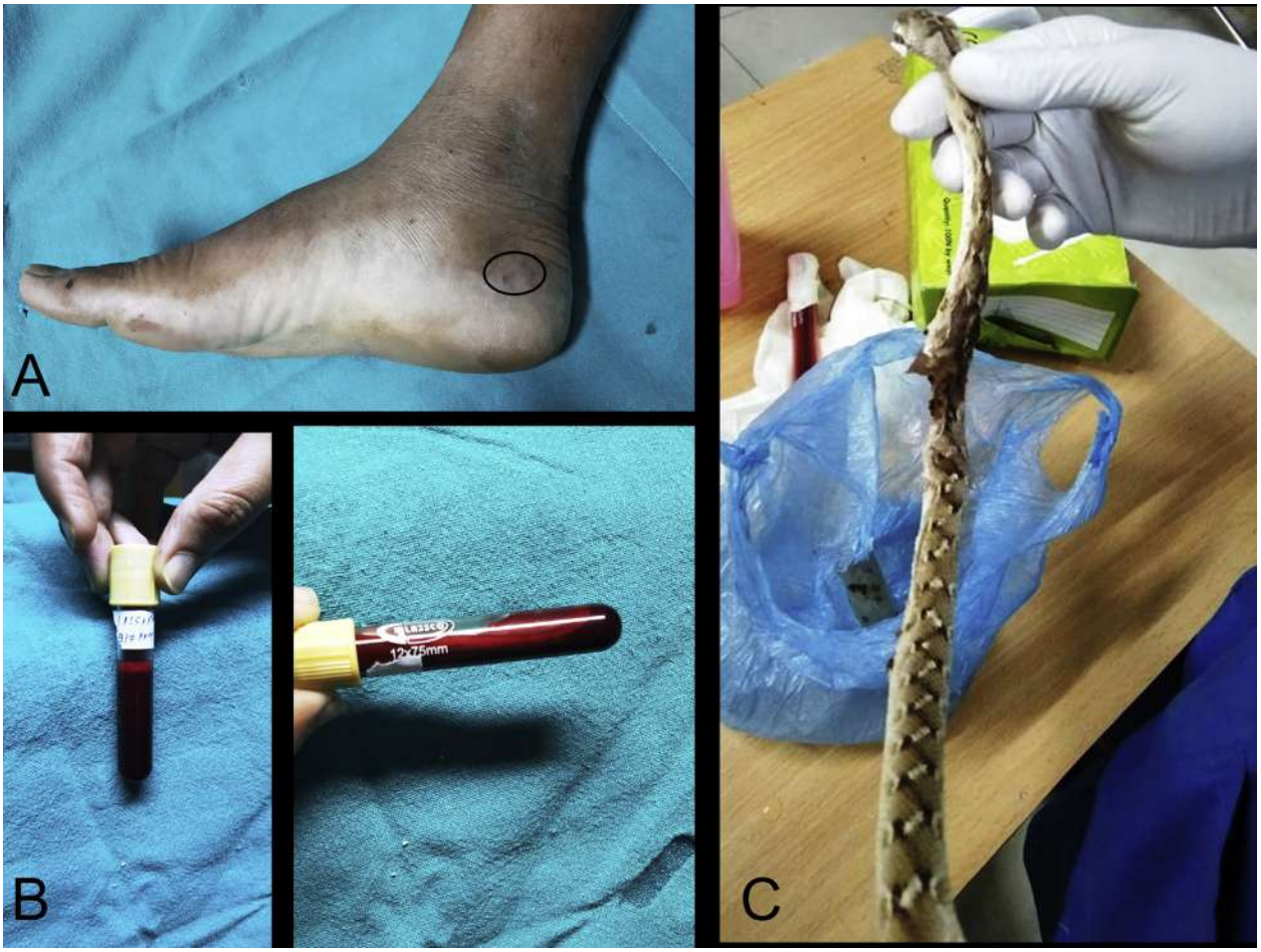

Figure 1. (A) Local swelling with bite mark over right foot. (B) Whole blood clotting test showing uncoagulable blood at 24 h. (C) Photograph of the snake provided by the patient's family.

and proteomics of different *Echis* species have confirmed this. Caswell et al highlighted the difference in the interspecific changes in the number of toxin genes, their transcription in the venom gland, and their translation into proteins secreted in the venom of various *Echis* species, including *Echis sochureki* (collected from United Arab Emirates (UAE). They demonstrated that *Echis* venoms exhibit significant interspecific variation in their ability to cause hemorrhage in murine in vivo models.[5]

Patra et al performed proteomic analysis of *Echis* spp. from India and demonstrated that the proteome composition of venom correlated well with its in vitro coagulant action and clinical manifestations.[6] Senji Laksme et al showed notable differences in the relative abundance of various toxins, such as phospholipase A2, snake venom metalloproteinase, and small molecules such as cysteine-rich venom secretory proteins, between *E carinatus* and *E sochureki*.[16] An important finding by both Patra et al and Senji Laksme et al relates to small-molecular-weight proteins (<30 KDa), which are more abundant in *E sochureki* compared to *E carinatus*.[6,16] This was confirmed by Bhatia and Vasudevan in their recent report; they found low-molecular-weight components such as phospholipase A2 and cysteine-rich venom secretory proteins were abundant in *E sochureki* venom obtained from Rajasthan when compared to *E carinatus* from Tamil Nadu or Goa states of India.[7] Snake venom serine proteases, which catalyze the coagulation pathway, showed 5-fold variation in snakes from the 3 regions, suggesting that the clinical profile of coagulopathy may significantly differ in these envenomings.

Another interesting speculation relates to the low abundance of L-aminoacid oxidases, responsible for tissue edema and platelet apoptosis in *E sochureki* venom.[7] Thus, absence of significant local swelling

Table 1. Coagulation and other laboratory parameters after envenoming

Test/Parameter (normal range)	*Day 1*	*Day 1 (after ASV)*	*Day 2*	*Day 3*	*Day 4*	*Day 5*	*Day 10*
PT (s) (10–15)	>120	25	68	>120	>120	>120	13
INR (<1.1)	Not calculated	2	9	Not calculated	Not calculated	Not calculated	1
aPTT (s) (25–35)	38	–	57	>120	–	>120	26
WBCT 20 (min)	>20	>20	>20	>20	–	>20	clotted
Hemoglobin ($g \cdot dL^{-1}$) (12.1–15.1)	13	–	13	–	–	–	–
Platelet ($10^9 \cdot L^{-1}$) (150–400)	278	–	249	251	–	–	250
Serum fibrinogen ($mg \cdot dL^{-1}$) (180–350)	13	–	–	–	–	–	–
Blood urea ($mg \cdot dL^{-1}$) (8–20)	27	–	–	–	26	–	–
Serum creatinine ($mg \cdot dL^{-1}$) (0.5–1.1)	0.8	–	–	–	0.9	–	–
Serum sodium ($mmol \cdot L^{-1}$) (135–145)	129	–	–	–	132	–	–
Serum potassium ($mmol \cdot L^{-1}$) (3.5–5.2)	4.4	–	–	–	4.2	–	–
Serum lactate dehydrogenase ($U \cdot L^{-1}$) (130–280)	186	–	–	–	–	–	–

aPTT, activated partial thromboplastin time; ASV, Indian polyvalent antivenom; INR, international normalized ratio; PT, prothrombin time; WBCT 20, 20-min whole blood clotting test.

(usually reported in *Echis* envenomings from Tamil Nadu) and absence thrombocytopenia in *E sochureki* envenoming may be explained by this relative reduction in L-aminoacid oxidases abundance.[7,12,16] It is pertinent to note that these small molecules were poorly neutralized by Indian polyvalent antivenom.[6]

Recently, camelid immunoglobulins have been explored and found to work successfully against *Echis sochureki* venom from Rajasthan in a preclinical setting.[17] The in vitro neutralization efficacy of camelid IgG for venom-induced coagulant effects, hemorrhage, and necrosis were comparable to equine antivenom against other *Echis* sp. outside India.

Because the usefulness of Indian polyvalent antivenom appears limited, such innovations need to be translated rapidly into clinical practice to effectively address *Echis* envenoming in the region. We suggest that early suspicion of *Echis sochureki* envenoming can help clinicians limit the use of polyvalent antivenom in these patients, thus avoiding unnecessary exposure to potentially life-threatening antivenom-associated reactions.

Conclusions

We document a case of clinical antivenom failure after *Echis sochureki* envenoming. There appears to be limited value of Indian polyvalent antivenom in neutralizing VICC caused by *Echis carinatus sochureki.*

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(MG and PY). Critical revision for intellectual content (NM, RM, MG and MKG). All authors read and approved the final manuscript.

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CASE REPORT

Rhabdomyolysis and Multisystem Organ Failure Due to Fulminant Ehrlichiosis Infection

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A previously healthy 51-y-old male presented to his local emergency department with subjective fevers, myalgias, dyspnea, and generalized weakness that had been progressive for several weeks. He was initially diagnosed with bilateral pneumonia, septic shock, and rhabdomyolysis requiring transfer to a tertiary care facility. He was treated for sepsis with broad-spectrum antibiotics, steroids, and a fluid bolus before transfer. Once he arrived at the tertiary care facility, he developed respiratory failure requiring intubation and ventilatory support. Ceftriaxone and metronidazole were started in the intensive care unit to cover common causes of community-acquired versus aspiration pneumonia, and doxycycline was included to cover tick-borne disease based on a history of tick exposure from working in his rural yard. Blood polymerase chain reaction testing later confirmed ehrlichiosis. The patient had a prolonged hospital course requiring ventilatory support and vasopressors, followed by a 4-wk stay in a rehabilitation unit after discharge. Wilderness medical providers should counsel their patients on prevention of tick bites and keep tickborne illness in the differential for acute illness, based on local epidemiology.

Keywords: tickborne disease, vector range, zoonosis

Introduction

Cases of tickborne disease (TBD) have doubled in the United States in recent years.[1] There were 47,743 cases of TBD reported to the US Centers for Disease Control and Prevention in 2018, including 6123 cases of ehrlichiosis and the related disease anaplasmosis.[2] However, there are few reports of septic shock, rhabdomyolysis, and multisystem organ failure secondary to ehrlichial infections. This is the case of a previously healthy 51-y-old male with untreated ehrlichiosis infection leading to septic shock and rhabdomyolysis.

Case

The patient was a 51-y-old male from southern Illinois who presented to his local emergency room in May with a chief symptom of weakness and shortness of breath.

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The patient presented with pain and weakness of the bilateral lower extremities that had progressed over the previous month such that he could no longer stand unassisted. He had developed shortness of breath, anorexia, and chills over the previous 3 to 4 d. On questioning, he reported spending a lot of time outdoors working in his rural wooded yard and recalled multiple tick bites over the previous 2 mo. The patient denied having any medical or surgical history, but he had not seen a physician since childhood. The patient's social history was significant for daily drinking, which he quantified as 6 to 7 shots per day and 5 to 6 cans of beer per day.

The patient was noted to be a cachectic middle-aged male in moderate respiratory distress. Vital signs were notable for a pulse of 128 beats·min^{-1}, a respiratory rate of 33 breaths·min^{-1}, a blood pressure of 93/67 mm Hg, and pulse oximetry of 93% on room air. Physical examination was positive for tachycardia and increased respiratory effort with bilateral wheezing. The patient's skin was warm and dry without rash. The patient was awake and alert on arrival to the intensive care unit and was following commands.

Routine laboratory testing was sent (Table 1). Initial chest x-ray showed bilateral infiltrates. Computed

Table 1. Laboratory values on initial patient presentation

Test	*Value*	*Reference range*
Sodium ($mEq \cdot L^{-1}$)	124	136–145
Potassium ($mEq \cdot L^{-1}$)	2.0	3.5–4.5
AST ($U \cdot L^{-1}$)	554	5–34
ALT ($U \cdot L^{-1}$)	80	0–55
Creatinine kinase ($IU \cdot L^{-1}$)	24,000	30–200
C reactive protein ($mg \cdot dL^{-1}$)	8.57	0.00–0.50
Procalcitonin ($ng \cdot mL^{-1}$)	3.71	≤0.10
Lactic acid ($mmol \cdot L^{-1}$)	13.9	0.5–2.0
D-dimer ($mg \cdot L^{-1}$)	9.67	<0.5
Platelets ($\cdot \mu L^{-1}$)	49,000	150,000–400,000

AST, aspartate aminotransferase; ALT, alanine aminotransferase

tomography of the chest showed diffuse alveolar process in all lung fields. Hepatomegaly with fatty changes versus acute inflammation was seen on computed tomography of the abdomen and pelvis. Blood was submitted for polymerase chain reaction (PCR) testing for common respiratory viral pathogens and for tickborne pathogens including *Erhlichia* and *Anaplasma* species.

The patient was started on ceftriaxone, metronidazole, and doxycycline based on our differential diagnosis, which included bacterial pneumonia, viral pneumonia, fungal pneumonia, aspiration pneumonitis, TBD, and subsequent organ failure from severe sepsis. He developed hypoxic respiratory failure on hospital day 2, requiring endotracheal intubation and mechanical ventilation for a total of 6 d. Once his disease progressed to respiratory failure, he underwent bronchoscopy, with multiple cultures of his bronchial washings. His blood was additionally recultured for bacterial and fungal pathogens; he was tested for *Legionella pneumophilia* and *Streptococcus pneumoniae* with urinary antigen testing. He also required vasopressor support for profound hypotension. His clinical course was complicated by alcohol withdrawal. Antibiotics were weaned to doxycycline alone after PCR testing was positive for Ehrlichiosis and sputum cultures, blood cultures, viral panels, and urine tests were negative for other pathogens. He completed a 10-d course of doxycycline. The patient spent 9 d in the intensive care unit, 3 wk in the hospital, and 4 wk in a rehabilitation unit.

Discussion

Ehrlichiosis is the general name used in the United States to describe a group of diseases caused by the bacteria *Ehrlichia chaffeensis*, *E ewingii*, or *E muris eauclairensis*.[3] Tick vectors carry these bacteria; most common are the lone star tick *Amblyomma americanum* and the blacklegged tick *Ixodes scapularis*. The natural host of ehrlichiosis is the white-tailed deer, which shows minimal symptoms in the carrier state. Tick feedings transmit the disease. When the larval phase feeds on an infected mammal, it acquires the infection; when the tick feeds during the nymph stage, it transmits the infection to a second host.[4]

Although the first human case of ehrlichiosis was reported in 1987, the disease has long been known to veterinarians owing to cases in livestock and companion animals.[5] Ehrlichiosis became a reportable disease in 1999; since then, the number of cases has steadily risen, and the case fatality rate has decreased to about 3%.[6,7] Ehrlichiosis is most often seen in the southeastern and midwestern United States, and cases have a seasonal distribution, likely based on human-tick encounters being higher in the spring and summer months. Early ehrlichiosis infections present with flu-like symptoms, including fevers, chills, headache, myalgia, malaise, and gastrointestinal symptoms. A rash is reported in 60% of children and less than 30% of adults.[8] Respiratory symptoms are present in 28% of patients.[9] If the infection goes untreated, there are cases of progression to cerebral infections, cerebral hemorrhage, acute respiratory distress syndrome, septic shock, renal failure, and hepatic failure.[10-12] Although myalgias are common in ehrlichiosis, clinically significant rhabdomyolysis is rare.[13,14] A history of tick bite within 14 d can only be elicited in 68% of individuals diagnosed with ehrlichiosis.[15] With tick bite providing such a low sensitivity of disease presence, practitioners in endemic areas must consider TBD regardless of tick bite history.

Pneumonia caused by ehrlichiosis has been described; however, the typical regimen for community-acquired pneumonia does not cover this pathogen. If TBD are not caught and treated in their early stages of infection, the practitioner must consider them in the presentation of more severe diseases by eliciting a history of tick bites or time spent outdoors with potential tick exposure. Practitioners must also integrate their location and local prevalence of TBD because a history of a bite alone is not sensitive for disease presence.

Risk factors for severe disease include immunosuppression, extremes of age, and delay in diagnosis.[9] Our patient likely experienced a more severe course owing to immunosuppression from undiagnosed diabetes and alcoholism, as well as a delay in presentation.

If the diagnosis is suspected, TBD should be covered empirically while awaiting confirmatory testing. Clues in routine bloodwork include leukopenia, thrombocytopenia, hyponatremia, and elevated liver enzymes. Anemia, coagulopathies, and lactic acidosis may also be seen.[8,9] Morulae (a neutrophil with an intracytoplasmic inclusion body) may be seen on blood smears

Figure 1. Reprinted by permission from Springer Nature Customer Service Centre GmbH: Springer Nature, *Modern Pathology*, Characteristic peripheral blood findings in human ehrlichiosis, Hamilton KS, Standaert SM, Kinney MC, Copyright 2004. https://www.nature.com/articles/3800075#:~:text=In%20this%20study%2C%2038%25%20of,of%20detection%20was%20only%2017%25.[33]

(Figure 1).[16] A rapid PCR test for ehrlichiosis and anaplasmosis is currently under development.[17] In the United States, tetracyclines, including doxycycline, are the preferred agents for the treatment of most tick-borne illnesses, regardless of the patient's age.[18] Ehrlichiosis, spotted fever rickettsiosis, and tularemia are endemic TBD in the lower midwestern United States; doxycycline provides coverage for these organisms. It should be noted that tetracyclines do not cover babesiosis.

Cases of ehrlichiosis are increasing, and cases are being reported in more counties than in previous years.[12] Although this may represent increased knowledge of the disease and better reporting of cases, most authors believe the disease remains underreported and the incidence is indeed rising.[6-8,12,18-20] It has been theorized that ehrlichiosis and other TBD are becoming more common as the tick vectors appear to be expanding their ranges because of climate change.[21-23] The range of *A americanum* has expanded into the midwestern and the northern United States from its origins in the southeastern United States, and the number of counties with *Ixodes* ticks has more than doubled in the past 20 years.[24,25] Although previously considered diseases of rural areas and wilderness recreation, several recent studies have found disease-carrying ticks in urban and suburban parks.[26-28] The tick vector range has been predicted to expand further with climate change modeling.[29]

Wilderness medical practitioners should be prepared to recognize TBD in their patients and start empiric treatment while awaiting confirmatory testing. Patients at risk for TBD may not recall an exposure or recognize early symptoms.[30,31] Knowledge of local disease patterns is crucial. The US Centers for Disease Control and Prevention has excellent resources for TBD, including images of tick vectors, maps of disease prevalence, and information on clinical presentations, diagnosis, and management.[32] Counseling patients on personal protective measures, including appropriate clothing, repellents, need for tick checks of both humans and companion animals, and proper removal of ticks may also reduce the burden of disease.

Conclusions

Although the current mortality rate associated with ehrlichiosis is only 3%, this case demonstrates that TBD can become fulminant and life threatening if left untreated. Vector-borne disease should be included in the differential of patients presenting with febrile illness during the spring, summer, and fall months, with diagnostic testing initiated in patients with possible exposures even without a recalled history of a tick bite. More acutely ill patients may require empiric treatment while awaiting confirmation.

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CASE REPORT

A Mixed Case Report of Long-Term Management of Ascites and Anasarca From Liver Disease on a Remote Pacific Island

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Remote and austere medicine can be challenging when caring for healthy individuals and requires ingenuity when managing patients with unexpected, complex disease processes, whether acute or chronic. We report the case of an adult male who presented to an isolated clinic setting with acute exacerbation of chronic liver disease. Medical management was complicated by limited transportation resources, both for supplies and patient movement. There was concern that the patient's clinical status could decompensate into a life-threatening illness that might not be adequately treated in this remote setting. Through the use of collaborative decision-making with the patient and by telemedicine consultation, the patient was successfully stabilized on the island until routine transport to a higher level of care was available. Knowledge of climbing systems and alternative transportation arrangements were integral to our ability to provide prolonged care for this patient in a remote setting. Knowledge of the use of medicinal plants was key in offsetting the side effects of other medications used. We are reminded that creative and collaborative problem-solving epitomizes wilderness and austere medicine.

Keywords: chronic disease, austere, hepatitis, herbal medicine

Introduction

Improvements in the management of chronic disease, ease of travel, and increased staffing in remote settings have resulted in medical providers caring for patients in austere settings who may not have previously met remote medical clearance requirements. Healthcare professionals in remote settings should possess a thorough understanding of acute, as well as chronic, disease management and have an aptitude for dealing with unexpected circumstances by using nontraditional medical therapies and interventions.

Case Report

Midway Atoll is a 6.2-km^2 island that is part of a national wildlife refuge managed by the US Fish and Wildlife Service. Because the refuge is not open to public visitation and has no indigenous population, its inhabitants consist of US Fish and Wildlife Service personnel and support contractors, totaling fewer than 100 people. The island clinic is staffed by a contract physician assistant and is intended for the evaluation of routine and occasionally urgent medical issues, but it has limited emergency capability and monitoring equipment. The facility contractors also include personnel for the small fire department (1–3 persons) who are certified emergency medical technicians and can assist with patient care as needed.

Our case report presents a male in his late 50s with chronic hepatitis C infection and suspected liver cirrhosis who was working as a contractor on Midway Atoll. Preemployment medical screening for contractors was limited, and the patient's chronic medical history was not shared with workplace clinic providers. Although clinic staff were aware the patient would embark on a once-monthly, 2100-km, fixed-wing contracted flight to see his gastroenterologist in Honolulu, medical staff were unaware that his liver disease had recently progressed and required intermittent therapeutic paracentesis by this gastroenterologist to remove excess fluid from the abdomen.

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The wildlife refuge has specific requirements for aircraft that use the airfield. At the time of our case study, only 1 company had 1 aircraft with the necessary certifications. Three days before the next scheduled monthly flight was due to arrive, the company went out of business, leaving the island's inhabitants without commercial aviation support for supplies or routine transportation.

The following week, the patient presented to the clinic. He had been scheduled to see his gastroenterologist but had had to cancel his appointment because of the canceled fixed-wing flight. He was concerned about his worsening abdominal distention and endorsed weight gain from his baseline of 70 kg to approximately 85 kg, which had occurred in the month since his previous routine evaluation by his gastroenterologist. He was unable to button his pants or shirts. Additionally, he had developed spontaneously weeping wounds at multiple sites across his body. During the physical examination, we noted no evidence of jaundice or scleral icterus. Lower extremity pitting edema was noted to the level of the knees, and bilateral periorbital edema was evident. To manage his fluid status, the patient had decreased his oral fluid intake. He had not taken any diuretics in the past and did not endorse taking any chronic disease medications or supplements currently. His vital signs were within expected ranges, and there was no evidence of pulmonary edema. The diagnosis of symptomatic ascites with anasarca was made based on the clinical presentation.

Diagnostic assessment capability at the clinic was limited. No point-of-care testing was available except for blood glucose levels. Standard clinic practice had been to send any necessary diagnostic studies to a Honolulu laboratory on the monthly flight, but this was no longer possible. A 12-lead electrocardiogram was available in the clinic, but quality assurance testing and calibration documentation could not be found. No ultrasound machine was available on the island.

Available interventions and medication in the island clinic were limited. The small dry goods store had relied on the scheduled monthly flight to deliver products and thus would receive no further supplies. There would be no further fresh food available until a new logistics chain was established for the island; residents were left with only the food stores in the island's converted shipping container freezers. The small tropical island's few natural resources were protected because of its federal refuge status.

Paracentesis was not considered a viable solution for this patient for multiple reasons. The clinic lacked the necessary procedural supplies, and improvisation with sterile needles, tubing, and vacuum canisters was not feasible. The lack of ultrasound capability was considered a safety concern. Furthermore, should the procedure cause complications, such as bowel perforation, infection, or epigastric vessel injury, these could not be handled locally.

A telemedicine discussion was initiated with the clinic's contracted physician medical director to discuss the patient's presentation and available treatment options. It was agreed that the patient did not meet the criteria for an urgent medical evacuation via a commercial fixed-wing aircraft. The decision was made to start treatment with daily low-dose (20 mg) oral furosemide, a low-sodium diet, and close monitoring. Risks, benefits, and alternatives of the care plan were reviewed with the patient, who agreed and believed that all alternatives should be explored before pursuing an evacuation.

Spironolactone, alone or in combination with furosemide, is generally regarded as the preferred diuretic regimen for ascites-related fluid overload.[1] However, spironolactone was not available in the clinic pharmacy. The inventory included a single bottle of 90 tablets of unexpired oral furosemide 20 mg.

The patient initiated furosemide therapy the following morning and was instructed to increase his fluid intake to prevent renal injury. He was evaluated daily in the clinic to monitor symptoms, evaluate vital signs, and record body weight on the clinic scale. Initially, the patient reported challenges with nocturia, which compromised his quality of sleep. Within the first 72 h, he noted resolution of his weeping wounds. Before the end of the first week, the patient's abdominal distention had decreased, and he was able to close the top button of his pants.

We were concerned that continued furosemide therapy might cause an electrolyte abnormality that could not be monitored in the clinic. No medical-grade supplemental potassium was available in the pharmacy to offset possible hypokalemia. However, the tropical island was populated with coconut palm trees with ripe coconuts.

Thus, where the island pharmacy fell short, the "island farmacy" offered an opportunity. Coconut water has been identified as a good source of natural potassium.[2,3] Some research has estimated that 1 L of coconut water can contain approximately 51 mEq of potassium.[4] The typical volume of water in a coconut is approximately 500 mL, thus providing a potassium dose within the range of empiric replacement while the patient was on the loop diuretic.[5,6] In a clinic visit during the first week of therapy, the patient agreed to consume the water from 1 coconut daily to replenish his potassium stores (Figure 1).

Coconut water is most nutritious when taken from fresh coconuts harvested while still on the palm tree.[2] To harvest the coconuts, a system was needed to ascend the approximately 10-m tall palm trees, but no heavy equipment was available on the island. Therefore, an

Figure 1. Picture of a "prescription" on one of the harvested coconuts. Note that the patient name "John Smith" is fictitious.

improvised ascender system was created from rope debris found on the island beaches. The clinic provider climbed palm trees each week to harvest sufficient coconuts for the patient (Figure 2). The coconuts were then stored in the island's commercial kitchen, where staff had experience cutting open and harvesting coconut water.

Within 14 d of starting diuretic therapy, the patient endorsed significant weight loss. He was no longer having difficulty with abdominal distention, and he was able to button his shirts. After another 2 wk of continued furosemide therapy, he was still approximately 7 kg above his baseline weight. Daily evaluations with the scale in the clinic showed no further weight loss.

Soon thereafter, the island was notified that a research vessel would be passing by on a patrol of the area. Because of the prolonged supply chain interruption to the island, a unique medication delivery system was implemented. The clinician was able to phone in a prescription for spironolactone to a commercial pharmacy at the ship's home port in Honolulu, and the patient phoned the pharmacy with payment information. The prescription was delivered by the pharmacy to the vessel, whose medical personnel delivered it to the patient during a brief stop at the island.

Within 14 d of adding spironolactone 100 mg daily, the patient noted that he had returned to his baseline weight and experienced resolution of all symptoms.

Subsequently, the patient was able to leave the island via a government fixed-wing aircraft that was in the area for a familiarization flight. He had a follow-up appointment with his gastroenterologist, who recommended continuing spironolactone as monotherapy and took over prescribing responsibility.

Discussion

Cirrhosis with anasarca is an example of complex chronic disease management that is usually managed within the confines of traditional healthcare facilities but can now be seen in austere settings, where the consequences of acute exacerbation are significant and potentially life-threatening. Standard care typically is sodium restriction and diuretic initiation (spironolactone with or without furosemide).[7,8]

Fluid status monitoring can be challenging in the austere environment. Ascites can be tracked via serial recording of abdominal girth. In the present case, exact measurements were not recorded, but girth was tangentially evaluated by the patient's ability to successfully use the snaps on the bottom of his shirts and close the top button on his pants.

Hypokalemia is a known complication of liver disease, and monotherapy with loop diuretics could have exacerbated the issue.[7] Empiric potassium supplementation was warranted given the inability to reliably check potassium levels in the austere setting.

Coconut water has been shown to have applicability in multiple areas of medical practice.[2] Studies have shown equivalency to normal saline when used as an intravenous resuscitation fluid in healthy volunteers.[9] Coconut water taken orally has been studied for recovery and as an oral rehydration fluid.[2] Medical professionals operating in austere environments may benefit from knowledge of the use of coconut water for patient care.

Potassium supplementation is available through myriad food sources. Potassium-rich foods include both shelf-stable and fresh foods. Fruit sources include dried apricots (1900 mg K^+ per 100 g), dates, and bananas.

Figure 2. Clinic provider using ad hoc rope system to climb coconut tree to harvest coconuts for patient consumption.

Legumes are known sources of potassium, with white beans having the highest content (561 mg K^+ per 100 g). Other foods common to athletes, such as salmon, avocado, and sweet potato, are also relatively high in potassium.[10]

This case highlights how telemedicine, second-line therapies, ingenuity, and knowledge about prevention of complications can temporize a disease process before problems progress to emergencies. Nonmedical skills, such as local plant use, nonstandard transportation platforms for supplies, and familiarity with rope systems, can potentially assist in the care of patients in austere environments.

Conclusions

Astute clinicians in remote locations will likely continue to encounter patients with unexpected health conditions that require creativity, collaboration, and skill to manage. This case illustrates many aspects of wilderness and austere medicine, including limited resources, difficult evacuation logistics, telemedicine, prevention of complications through an understanding of medication side effects, and even nonmedical technical skills, such as improvised climbing techniques. Creative problem-solving is key to successful medical management in the austere environment. Potassium supplementation using coconut water may be a safe and effective alternative when traditional therapies are unavailable, although a lack of objective dosing and the limited prospective data should be carefully considered before implementing the practice.

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CASE REPORT

Marine Penetrating Injury to the Shoulder of Uncertain Origin

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Penetrating injuries from marine animals are rare events; however, published case reports have detailed critical injuries including death occurring as a result of such incidents. We present a case of a marine penetrating injury to the right posterolateral shoulder of a 10-y-old boy. The patient underwent open surgical debridement and a course of oral antibiotics before returning to normal function. Clinicians should have an appreciation of various clinical patterns of marine penetrating wounds, the need for prompt imaging to exclude foreign bodies, and appropriate antibiotics to cover gram-negative bacteria and *Vibrio* species, which are commonly found in marine-related injuries.

Keywords: emergency medicine, trauma, Australia, orthopedic, aquatic, musculoskeletal

Clinical Record

A 10-y-old boy presented to the emergency department in the northwest of Western Australia after being struck in the right posterolateral shoulder while paddling in whitewash while surfing. The boy sustained a 2 cm crescentic wound, which was actively bleeding on arrival to hospital. On presentation to the emergency department, he reported mild pain in his shoulder at the wound site but was otherwise hemodynamically stable and exhibited no systemic signs of envenomation, such as nausea/vomiting, respiratory distress, abdominal pain, or altered conscious state. Additionally, there was no localized edema, erythema, bony deformity, or foreign body at the wound site (Figure 1). Although examination of shoulder strength and mobility was difficult owing to mild pain, the patient had no signs of neurovascular compromise. The clinical impression of the treating team was that the pain on examination was in keeping with a penetrating injury rather than due to envenomation. Given the rurality of the location, the treating team was unable to access imaging to exclude the presence of a foreign body. The patient underwent examination under local anesthesia, which revealed no foreign body, and the wound was vigorously irrigated. Given the clinical impression that there was no remaining foreign body, the wound was sutured to promote wound healing by primary intention. Oral azithromycin was commenced to cover *Staphylococcus* and *Vibrio* species.

Although the exact mechanism of injury was unclear, hospital staff explored the possibility of a shark attack or stingray barb; however, neither fit the clinical picture. Hospital staff raised the possibility of a needlefish injury given the prevalence of large schools of needlefish in the region and the clinical pattern of a single penetrating wound with an absence of systemic signs of envenomation.

Twenty-four hours after initial injury, the patient was systemically stable; however, he reported worsening pain in his upper arm, with pain radiating down his biceps. Biceps pain in the context of a penetrating injury to the posterior shoulder was of concern because there was a suspicion of intra-articular penetration and risk of septic arthritis. Given the patient's worsening pain and the possibility of a septic joint or foreign body, the patient was transferred to Perth, Western Australia, via a commercial flight the following day to a higher center of care for advanced imaging and surgical intervention.

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Figure 1. Open penetrating wound at the posterolateral shoulder on initial presentation.

Magnetic resonance imaging (MRI) performed on arrival to Perth revealed that the patient had sustained a deep injury to the posterior scapula involving the posterior fibers of the deltoid, the full thickness of the infraspinatus muscle belly, and the deep aspect of the supraspinatus muscle belly. There was edema at the spinoglenoid notch, narrowly avoiding the shoulder joint itself and the suprascapular nerve (Figure 2). No foreign body, hematoma, or fluid collection was noted.

Ongoing anterior shoulder and biceps pain in conjunction with suspected intra-articular penetration 48 h after initial injury prompted surgical examination (Figure 3). Surgical examination revealed that the injury did extend to the intra-articular space. The suprascapular nerve was visualized and was found to be intact but contused. Swabs were taken for microscopy, culture, and sensitivity, which subsequently revealed no growth of pathological microorganisms.

After infectious disease consultation, the patient commenced oral ciprofloxacin and amoxicillin-clavulanic acid because marine wounds are prone to infection with gram-negative bacteria and *Vibrio* species.

Follow-up at 2 wk after the original injury revealed the wound had healed and the patient had regained full shoulder mobility and strength.

Discussion

This case represents a rare penetrating injury that presented several challenges for clinicians, particularly given the rurality of the location.

The initial challenge was that the exact mechanism of injury was unclear. The patient was initially concerned that the injury may have been caused by either a shark bite or stingray barb. However, the pattern of injury of a single penetrating wound to the posterior shoulder did not match what would generally be expected in either of these events. In cases in which the exact diagnosis is unclear, a broad range of differential causes should be explored by the treating team.

Stingrays are most often trodden on; as such, injury to the lower limb is most commonly involved, with penetrating injuries to major body cavities being rare but life-threatening. Those impaled by a stingray barb may have a retained barb or, if the barb has been removed, may have a macerated appearance to the wound owing to the retroserrated barb. The pain is severe immediately and worsens over the next few hours as a result of envenomation.[1] A shark attack would typically present with multiple crescent-shaped penetrating wounds, extensive soft tissue injuries, and damage to aquatic gear such as surfboards.[2] The clinical pattern of a single penetrating injury to the posterior shoulder did not match what would generally be expected in either of these events.

The possibility of the injury being caused by a needlefish was raised by hospital staff, given the clinical picture of a single penetrating wound, no systemic signs of envenomation, and local knowledge of large schools of needlefish in this region. Furthermore, hospital staff described a prior experience with a patient who had sustained pneumothorax as a result of a penetrating injury from a needlefish. Previous case reports have detailed presentations of retained foreign bodies, neurovascular

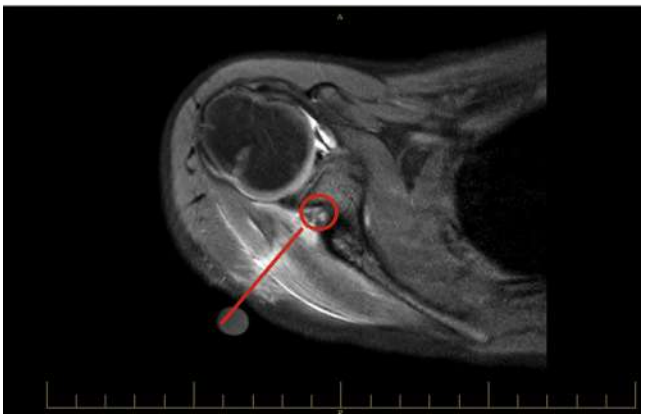

Figure 2. Magnetic resonance imaging axial view showing penetration tract from skin into the spinoglenoid notch. Red line indicating path of penetrating wound; red circle indicating the location of suprascapular nerve.

compromise, persistent pain, and localized infection resulting from needlefish injuries.[3,4]

Needlefish species commonly found in temperate waters of northern Western Australia include the Garfish (*Belone belone*) and Longtom (*Tylosurus gavialoides*).[4] These species are silvery in color with elongated cylindrical bodies up to 70 cm in length and long narrow jaws that extend into a sharp beak with jagged teeth (Figure 4). These surface-swimming, predatory fish can achieve speeds of up to 60 $km \cdot h^{-1}$ when hunting small baitfish or avoiding predators.[4] These species are commonly observed leaping out of the water, making them a risk to swimmers, surfers, and fishermen. The majority of reported penetrating injuries due to needlefish are to the head, neck, and torso.[4,5]

Appropriate and prompt imaging after a penetrating injury may be challenging in remote locations. The wound margins can underrepresent the depth of the injury, with physical examination alone being an unreliable means of determining the severity of penetrating injuries. Clinicians should select an imaging modality that is appropriate to the suspected type, size, and depth of foreign body, anatomic location, and injury to adjacent tissues.[6] X-rays are an excellent screening tool and

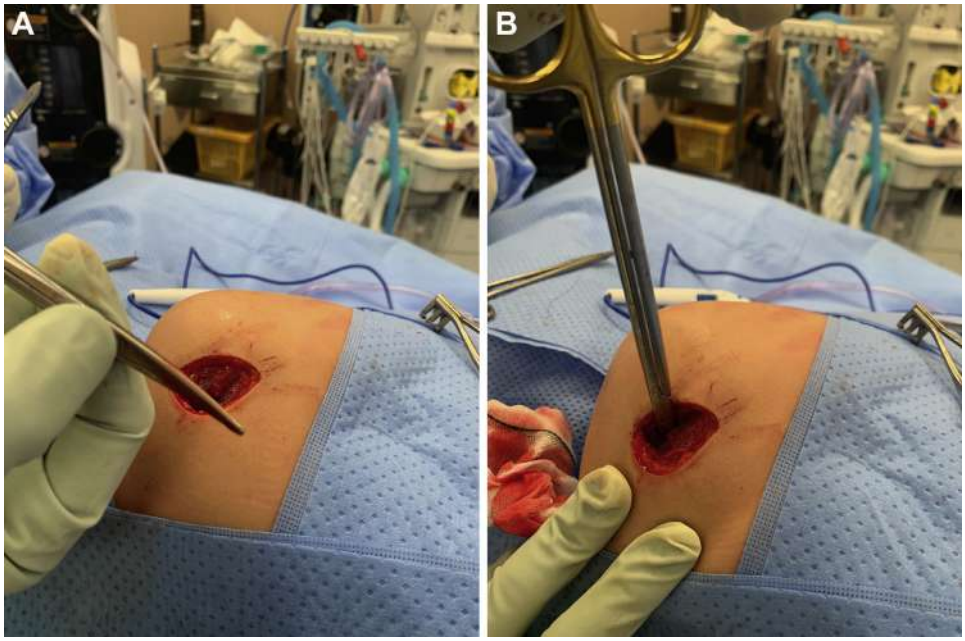

Figure 3. Open penetrating wound at the posterolateral shoulder when examined under anesthesia.

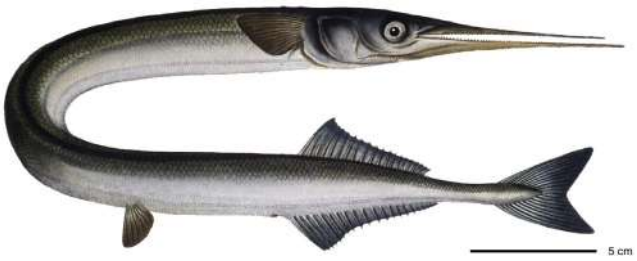

Figure 4. Longtom fish (*Tylosurus gavialoides*).

should be routinely employed to rule out residual radiopaque foreign bodies. Computed tomography scanning is superior to x-ray if there is a high suspicion for retained foreign body because it allows for precise localization of foreign bodies, which is a prerequisite to surgical removal.[6] Ultrasonography is more useful for locating superficial foreign bodies and has the advantage of not involving radiation exposure. However, ultrasound is unsuitable for foreign bodies located within deeper tissues or inside the air-filled cavities. In this case, as in many others, MRI was the superior imaging option and was chosen given the assumed depth of injury, ability to visualize retained soft tissues such as cartilage, and need to localize the exact position of the foreign body before surgical removal.[6] MRI is also seen as a superior modality particularly in children because there is no exposure to radiation.

Finally, there are both broad principles and species-specific protocols for the management of marine penetrating injuries, which may be challenging owing to the often-remote nature of these incidents. As with all penetrating injuries, there is a need to achieve hemostasis and commence early resuscitation if required. Appropriate imaging should be performed to exclude the presence of a foreign body. In the case of a stingray barb, medical treatment includes achieving hemostasis followed by submersion of the affected region in hot, but not scalding, water (42–45°C) until pain resolves because stingray venom is heat labile.[7] All wounds should be examined under anesthesia in either the emergency department or the operating room.[1] This decision is based on the size of the wound, depth of the wound, presence of debris, and any potential joint involvement.[1] Wounds should be thoroughly cleansed, and delayed closure should be allowed. Surgical exploration may be necessary to remove residual foreign bodies and to conduct debridement.[8] Given the clinical impression that there was no remaining foreign body, the wound was sutured to promote wound healing by primary intention. In wounds with the possibility of retained foreign material, the decision to leave the wound open (healing by secondary intention) or suture the wound should be made based on the clinician's risk assessment.[8] Clinicians should consider the likelihood of a retained foreign body, need for hemostasis, and potential for infection when deciding whether to suture the wound. Marine penetrating injuries are prone to secondary infection, and antibiotics are routinely indicated to provide coverage for gram-negative species, such as *Aeromonas, Escherichia coli,* and *Vibrio*.[9] Empirical antibiotic therapy should include a combination of a beta-lactam antibiotic (flucloxacillin) and tetracycline (doxycycline), or clindamycin and ciprofloxacin in the penicillin-sensitive patient.[10] Tetanus immunization status should be reviewed and immunization given if appropriate.

Clinicians should explore a broad range of differential causes when presented with marine penetrating wounds. Radiologic examination is important in all cases of penetrating injury to identify retained foreign bodies, with the choice of imaging modality based on anatomic location and adjacent tissues, as well as suspected type, size, and depth of the foreign body itself. The definitive management of marine penetrating wounds may be challenging in remote locations and may require transfer to a higher center of care capable of advanced imaging and surgical intervention. The mainstay of treatment involves exploration under sterile conditions, irrigation, debridement, suturing (where clinically appropriate), and antibiotics as guided by the organisms of concern, the depth of injury, and the potential anatomic structures involved.

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REVIEW ARTICLE

Underrecognized Tickborne Illnesses: *Borrelia Miyamotoi* and Powassan Virus

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Over the past 2 decades, tickborne disease has been increasingly recognized as a threat to humans as a result of the growing geographic range of ticks. This review describes 2 tickborne diseases, *Borrelia miyamotoi* and Powassan virus, that likely have a significant impact on humans, yet are underdiagnosed compared to most other tickborne diseases. We performed a literature search from 2015 to 2020. *Borrelia miyamotoi* is a tickborne pathogen that infects and co-infects ticks along with other pathogens, including *Borrelia burgdorferi*. Because *B miyamotoi* infects the same *Ixodes* ticks as *B burgdorferi*, *B miyamotoi* may cover a similar geographic range. *B miyamotoi* infection may be underdiagnosed for 2 reasons. First, a presumptive treatment approach to Lyme disease may result in *B miyamotoi* infection treatment without identification of the actual cause. Second, the absence of readily available testing and diagnostic criteria makes it difficult to diagnose *B miyamotoi* infection. Powassan virus is a tickborne flavivirus similar to the dengue virus. Powassan virus disease appears to have an asymptomatic or minimally symptomatic presentation in most people but can cause devastating and fatal encephalitis. The Powassan virus may be transmitted in less than 15 min of tick feeding. Powassan virus disease is a difficult diagnosis because testing capabilities are limited and because there may be co-infection with other tickborne pathogens.

Keywords: relapsing, fever, encephalitis, flavivirus, spirochete, Ixodes

Introduction

Over the past 2 decades, the public health threat posed by ticks has increased significantly and results from greater exposure of the human population to the expanding geographic range of ticks.[1] This human exposure is multifactorial and includes the increased spread of humans to suburban areas, increased use of outdoor areas, and greater tick populations. The discovery of several new tickborne human pathogens has increased awareness and recognition of more diseases spread by ticks.

Over the past 13 y, the number of cases of tickborne diseases reported in the United States has more than doubled, with state and local health departments reporting a record number of cases of tickborne conditions to the Centers for Disease Control and Prevention.[2] The reported number of cases of Lyme disease, anaplasmosis, ehrlichiosis, spotted fever rickettsioses (including Rocky Mountain spotted fever), babesiosis, tularemia, and Powassan virus disease increased from a total of 48,610 reported cases in 2016 to a total of 59,349 reported cases in 2017.[3] These reported cases likely capture only a fraction of the overall number of people with tickborne illnesses.[3]

In this review, we describe 2 tickborne diseases that are less familiar and uncommonly diagnosed yet have potential morbidity and mortality owing to limited diagnostic criteria and testing: *Borrelia miyamotoi* and Powassan virus disease. We chose to discuss *B miyamotoi* because it is likely a more prevalent disease than currently known, and recognition is challenging because there are no diagnostic criteria and there is no widely available test. This could result in physicians overlooking this infection and failing to treat a patient properly if other more recognized infections such as Lyme, babesiosis, rickettsioses, anaplasmosis, and ehrlichiosis are ruled out by testing that is widely available. We chose to review the Powassan virus because it only requires 15 min of tick attachment for transmission, and the sequelae of the neurologic disease are devastating, in addition to a 10% mortality rate.

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Methods

We performed a literature search capturing 2015 to 2020 in Embase, MEDLINE, and Cochrane Database using the keywords *Borrelia miyamotoi*, Powassan virus, humans, epidemiology, and clinical presentation. This search was limited to these years because we had performed previous research on the topic but did a recent formal review to ensure complete information.

BORRELIA MIYAMOTOI

What is it?

Borrelia miyamotoi is the causative agent of *Borrelia miyamotoi* disease (BMD). *Borreliae* are long, highly motile corkscrew-shaped bacteria that are part of the spirochete phylum. The genus *Borrelia* comprises the Lyme disease group (*Borrelia burgdorferi* and other similar spirochete species) and the relapsing fever group (including *B hermsii*, *B turicatae*, *B parkeri,* and *B miyamotoi*). Although other tickborne relapsing fever spirochetes infect the soft-body ticks of the *Ornithodoros* genus, *B miyamotoi* is the exception: It infects the hard-shell *Ixodes* tick that also carries *B burgdorferi*.

When was it discovered?

B miyamotoi was first isolated from an *Ixodes* tick in Japan in 1995.[4] The first description of human infection was a Russian case series in 2011.[5] Since then, human cases have been reported elsewhere in Europe, China, and the United States in 2013.[6-9] *B miyamotoi* has been found in various *Ixodes* ticks, including *I scapularis* (also called black-legged or deer tick) in the northeastern and northern midwestern United States, *I pacificus* in California, *I ricinus* in Europe, and *I persulcatus* in Europe and Asia.[4,10-13]

How is it transmitted?

The *Ixodes* ticks have increased their geographic range in the past decade[14,15] owing to human and natural factors,[13] such as land use, climate change, and change in vector populations. The geographic range of BMD is not well described, but it likely mirrors Lyme disease, with about 2% of *Ixodes* ticks carrying the spirochete.[16,17] *I scapularis* is documented in 1420 (45.7%) of the 3110 continental United States counties.[15] *Ixodes* ticks can be co-infected with multiple pathogens and can transmit more than 1 pathogen.[18-20] One study evaluating patients in California found antibody seropositivity with *B burgdorferi* in 33%, relapsing fever *Borrelia* in 27%, and both in 11%.[21] This suggests that co-infection is possible and that the prevalence of *B miyamotoi* and other relapsing fever *Borrelia* species may be underestimated. *B miyamotoi*, unlike *B burgdorferi*, can be maintained both transstadially (persisting through different life stages of the tick) and transovarially (passing from parent to offspring via the ovaries) within a host tick.[22] The tick can transmit *B miyamotoi* at any stage of life, resulting in a possibility of human infection throughout a more significant part of the year than Lyme disease.[23] Ticks become infected with *B burgdorferi* by feeding on an infected host, which usually occurs at the larval stage. They then take a second feeding the next spring, when they are likely to transmit *B burgdorferi.* For *B miyamotoi,* the relation to tick stage is less critical. The tick may hatch already infected with and able to transmit *B miyamotoi*. This means it may transmit *B miyamotoi* with the first feeding as a larva in spring, summer, or fall and later in its nymphal and adult stages.

How long does the tick have to be attached to transmit Borrelia miyamotoi?

Nymphs that are transovarially infected with *B miyamotoi* harbor the spirochetes in their salivary glands before attaching to a host. This allows for rapid transmission.[24] *B miyamotoi* can be transmitted 10% of the time within the first 24 h of attachment, increasing steadily to reach 73% for a complete feeding.[24] There is no evidence of transmission of *B burgdorferi* in the first 24 h of attachment and only 10% in 48 h.[24] Thus, transmission of *B miyamotoi* is more rapid than transmission of *B burgdorferi*. Understanding this more rapid transmission of infection of *B miyamotoi* may be a consideration in determining prophylactic treatment for tick bites with a shorter time of attachment in endemic areas for *B miyamotoi*. It also underscores the need for frequent tick checks for those exposed to tick infested settings.

Clinical presentation

The original 2011 Russian case series described the symptomatology of 46 patients, including viral-like symptoms of fever (98%), malaise (98%), headache (89%), and myalgias (59%), with onset about 2 wk after a tick bite. Eleven percent had 1 or more recurrent episodes of symptoms, 68% had liver enzyme abnormalities, and 9% had an erythema migrans type rash. Patients generally did well after treatment with ceftriaxone or doxycycline.[5] These disease features were echoed in the 2015 Northeastern United States case series: Patients had a nonspecific febrile illness (96%) with headaches (96%), myalgias (84%), fatigue (82%), and arthralgias (76%), as

well as elevated liver function tests (53%), thrombocytopenia (51%) and leukopenia (43%). Only 8% had a rash. The absence of a rash in conjunction with the leukopenia, thrombocytopenia, and elevated liver function tests found in many tickborne diseases may help to distinguish BMD from Lyme disease.[25]

There are 4 case reports of *B miyamotoi* meningitis in immunocompromised patients and 1 case report in an immunocompetent patient. The cerebrospinal fluid in all cases showed pleocytosis with elevated protein levels.[9,26-28]

Although symptoms and laboratory testing abnormalities in BMD are similar to those of anaplasmosis, patients with anaplasmosis usually defervesce within 24 h after starting doxycycline. BMD should be considered for patients with presumed anaplasmosis who do not improve within 24 h after the initiation of antibiotics.[29] Tickborne relapsing fever usually presents with multiple febrile episodes that correspond to peak spirochete loads in the blood. With BMD, the highest number of reported relapsing events after the initial fever was 2.[5] Although more relapsing febrile events may have occurred in untreated patients, the natural history of febrile events may be underestimated because many patients received empiric treatment after exposure to ticks.[5]

Making the diagnosis

Making a definitive diagnosis of BMD is challenging. Suspicion should be based on clinical features, geographic location, time of year, and exposure to ticks. Many patients may not be aware of an inciting bite because of the very small size of tick larvae and nymphs that can still spread the infection. No test specific to *B miyamotoi* has been approved by the United States Food and Drug Administration as of October 2020. Although several testing options for other *Borrelia* species are available, there is potential for cross-reactivity. Serologic testing cannot accurately differentiate between acute and past infections and may be less likely to be positive during the initial infectious phase.[30] Blood smears are not sensitive enough to consistently confirm the presence of *B miyamotoi* when there is a positive antigen test.[31]

Serologic testing of *B miyamotoi* IgM and IgG antibodies is possible by a few commercial laboratories.[13] Depending on the test chosen, it is possible to differentiate *B miyamotoi* from *B burgdorferi*.[32] However, other tests for *B burgdorferi* might allow for a cross reaction with *B miyamotoi*. This could lead to an incorrect diagnosis of Lyme disease in a patient who actually has BMD.[33,34]

The most specific test currently available in several public health and commercial laboratories is polymerase chain reaction (PCR) testing of blood or cerebrospinal fluid for the *B miyamotoi* GlpQ enzyme. When interpreting the test results, consideration must be given to other endemic relapsing fever spirochetes present in the area. These other spirochetes may give a false-positive test result for *B miyamotoi*. PCR testing does provide the benefit of directly confirming an acute infection, rather than relying on detection of an antibody response to the pathogen. However, accessibility is still very limited. One test using this approach, the TBD serochip, is an array-based assay testing for 8 different tickborne diseases, including *B miyamotoi*. Developed in 2018, it is promising but has not yet become widely available.[35]

Treatment

There is no clear evidence-based treatment for *B miyamotoi*. The use of doxycycline as the primary therapeutic agent has been derived empirically from the treatment of *B burgdorferi* and has shown success in the published literature.[5,6,26] In vitro analysis has shown the susceptibility of *B miyamotoi* to ceftriaxone, azithromycin, and doxycycline, with resistance to amoxicillin.[36]

POWASSAN VIRUS

What is it?

Powassan virus is a flavivirus that encodes for a single polypeptide that replicates in host cells.[37] Other more commonly known diseases caused by flaviviruses include dengue, yellow fever, West Nile encephalitis, and tickborne encephalitis (primarily found in Europe). Other tickborne viruses that have been recently discovered in the United States are the Heartland virus and the Bourbon virus. The Heartland virus is a phlebovirus first discovered as a cause of human illness in 2009 in Missouri.[38] The Bourbon virus is a thogotovirus discovered in the United States in 2017 that has been identified in the Midwest and the southern United States.[39]

Powassan virus is 1 of 6 mammalian tickborne flaviviruses. It exists in 2 lineages: the prototype Lineage I Powassan virus and Lineage II, called the deer tick virus. The deer tick virus is very similar genetically to the Powassan virus. The clinical presentation of Powassan virus and the deer tick virus infection are the same.[40] For this review, Powassan (POW) virus refers to both the Powassan virus and the deer tick virus, and Powassan virus disease (PWVD) refers to the clinical presentation from infections caused by each of these viruses.

When was it discovered?

Powassan virus is named for the Ontario, Canada, town where it was first isolated from the brain of a 5-y-old boy who died of severe encephalitis in 1958.[41] It was later

identified in New Jersey in 1970 and in Eastern Russia in 1978.[42,43] PWVD has not been reported in any other country. In the United States, in the years 2014 to 2019, there were 7, 6, 21, 33, 21, and 37 cases recorded by the Centers for Disease Control. In 2019, 13 US states reported cases: Connecticut, Indiana, Massachusetts, Maine, Minnesota, North Carolina, North Dakota, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Wisconsin.[44]

How is it transmitted?

The hard-bodied *Ixodes* ticks are the vector for the POW virus. *I cookei*, the groundhog tick, and *I scapularis* are the principal vectors. *I cookei* usually lives in burrows or nests and only travels on its host groundhogs and skunks when they move to new burrows.[45] *I cookei* rarely feeds on humans, probably because it has less exposure to humans than other tick species. Therefore, it has been relatively difficult to study. In contrast, *I scapularis* ticks are more easily harvested and easier to study. *I scapularis* ticks in their larval form feed on the white-footed mouse, which is thought to be the primary reservoir of the POW virus. The nymphs feed on small mammals, birds, reptiles, and humans. The adult females feed primarily on larger animals such as deer and livestock.[13] Studies of wild *I scapularis* populations have shown a POW virus infection rate in the range of 0 to 5%.[46,47] Human exposure is thought to be increasing with the expanding geographic range of *I scapularis*.[48]

In experimental conditions, the transmission of POW virus from viremic hosts to adult *I scapularis* females occurred at a rate of 57%. Infected ticks at any life stage can transmit the POW virus to new hosts.[49] Although many flaviviruses have mosquitos as competent vectors, there is no evidence of human POW virus disease transmitted by mosquitos, nor is there evidence that mosquitos are a competent vector for transmission to humans.[50]

POW virus has been shown to co-infect *I scapularis* ticks along with many other diseases, such as *B burgdorferi*, *Anaplasma phagocytophilum*, *B miyamotoi*, and *Babesia microti*.[20,47] *Ixodes* ticks also transmit *Ehrlichia* species and have potential for co-infection with POW virus, although this has not yet been reported. Unfortunately, much of the virome of ticks has been unknown until recently, and the scope of human disease caused by these is largely undiscovered.[51]

How long does the tick have to be attached to transmit the Powassan virus?

POW virus differs from many tickborne pathogens such as *B miyamotoi, B burgdorferi*, *Anaplasma*, and *Babesia microti* because there is no substantial lag time between tick attachment to the host and transmission. This is attributed to the virus already being present in the salivary glands at the time of feeding,[52] as opposed to many other nonviral tickborne diseases that are harbored in the tick's midgut. Transmission in mice has been shown to occur within 15 min of *I scapularis* attachment.[14,52]

Clinical presentation

Few people who become infected with the POW virus have clinically significant disease. However, POW virus can cause serious central nervous system infections, including encephalitis. In a 1962 study of Northern Ontario residents, 11 of 1008 individuals were found to be seropositive for the virus, indicating that they had been previously infected, either asymptomatically or at least without severe disease.[53] The rate of seropositivity was 1% in the entire study population but up to 3% in areas with a higher incidence of known illness.[53] The typical incubation period is 8 to 34 d before the development of a nonspecific viral syndrome of malaise, sore throat, and nausea. After 1 to 3 d, symptoms may worsen to include disorientation, headache, neck stiffness, and fever up to 40°C. The symptoms may be accompanied by clonus, ocular, and other motor palsies, as well as obtundation and convulsions.[54-56] Temporal lobe involvement with symptoms, including olfactory hallucinations, can mimic herpes simplex encephalitis.[57] Neuroinvasive disease is often severe and disabling, with long-lasting neurologic sequelae. Approximately 50% of cases result in lasting hemiplegia, memory problems, and muscle wasting.[56] Ten percent of cases are fatal.[56]

A definitive diagnosis of PWVD can be made by PCR of blood or cerebrospinal fluid. However, the virus can only be detected during the early viremic period. In most cases, this has passed before the onset of neurologic symptoms, severely limiting the ability of PCR to diagnose the disease.[58] IgG by enzyme-linked immunosorbent assay is the mainstay of diagnosis, but confirmation requires specialized testing. Cerebrospinal fluid analysis is consistent with aseptic meningitis. Poliomyelitis-like anterior horn involvement may be seen on magnetic resonance imaging,[59] and changes have been observed on electroencephalogram.[60]

Diagnosis of PWVD can be further complicated by co-infection with other pathogens such as *B burgdorferi*.[61] For example, 1 patient presenting with hemolytic anemia, thrombocytopenia, mild hepatitis, and acute kidney injury later developed a declining mental state, myoclonus, and tremors. The final diagnosis included concurrent Lyme carditis, babesiosis, and Powassan encephalitis.[62] Because illness due to PWVD is

nonspecific, taking a careful history may be vital to making this rare but important diagnosis. The history should include potential exposure to environments where ticks are present.

Treatment

There is no specific treatment for PWVD other than supportive care. Candidate vaccines have so far lacked generalizability, efficacy, or lasting immunity. This highlights the importance of preventing tick bites by avoiding tick-infested areas, wearing appropriate clothing, and using chemical repellant.

Conclusions

Borrelia miyamotoi and Powassan virus, as well as most other tickborne diseases that affect humans, are uncommonly recognized. They are transmitted by many of the same ticks as Lyme disease and may present as a primary infection or a co-infection. One should have a high index of suspicion for these diseases in patients presenting with possible Lyme disease. Unfortunately, at this time, there are no widely available, definitive diagnostic tests or diagnostic criteria for either disease.

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REVIEW ARTICLE

Finger Flexor Pulley Injuries in Rock Climbers

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Finger flexor pulley system injuries are the most common overuse injury in rock climbers. These injuries occur rarely outside of rock climbing, owing to the sport's unique biomechanical demands on the finger. As rock climbing continues to grow and earn recognition as a mainstream sport, an understanding of how to diagnose and treat these injuries also has become important. Our purpose is to describe current concepts in anatomy, biomechanics, clinical evaluation, imaging, prevention, and treatment strategies relating to finger flexor pulley system injuries. Our literature search was performed on PubMed with MeSH terms and keywords as subject headings to meet the objectives of this review. The "crimp grip" used in rock climbing is the mechanism for these injuries. The A2, A3, and A4 pulleys are at the highest risk of injury, especially when loaded eccentrically. Physical examination may reveal clinical "bowstringing," defined as the volar displacement of the flexor tendons from the phalanges; however, imaging is required for characterization of the underlying injury. Ultrasound is highly sensitive and specific for diagnosis and is recommended as the initial imaging technique of choice. Magnetic resonance imaging is recommended as an additional imaging study if ultrasound is inconclusive. Properly warming up increases the amount of physiologic bowstringing and is thought to prevent injury from occurring. Pulley injuries may be classified as grade I through IV. Conservative treatment, including immobilization, the H-tape method, and the use of a protective pulley splint, is recommended for grade I to III injuries. Surgical repair is reserved for grade IV injuries that are not amenable to conservative treatment.

Keywords: pulley rupture, ultrasound, climbing biomechanics, climbing injury prevention

Introduction

The popularity of rock climbing has increased remarkably, both recreationally and competitively, over the past 30 y. Indoor climbing, primarily, has seen exponential growth globally, and especially within the United States. The proposed debut of competition climbing in the now postponed 2020 Tokyo Summer Olympics speaks to its ever-growing recognition as a sport.[1] "Free Solo," a rock-climbing film, won best documentary at the 2019 Oscars ceremony, further emphasizing its broad appeal. According to the 2019 annual report by the International Federation of Sport Climbing, 44,500,000 people worldwide climb regularly, with 9,000,000 of those in the United States.[2,3]

As a consequence of the sport's growth, climbing-related injuries have increased concomitantly. An average of 2237 climbing injury-related emergency department visits in the United States occurred annually between 1990 and 2007.[4] Lower extremity injuries were most common, and overuse injuries were most frequent in the upper extremities.[4] More recently, 2 large retrospective national database studies estimated an average of 3419 rock climbing injuries were seen in emergency departments annually between 2008 and 2014, representing a 50% increase between 1990 through 2007 and 2008 through 2014.[5,6] The injury patterns remained consistent, with the lower extremities being most vulnerable, likely owing to falls that cause most injuries.[4,5]

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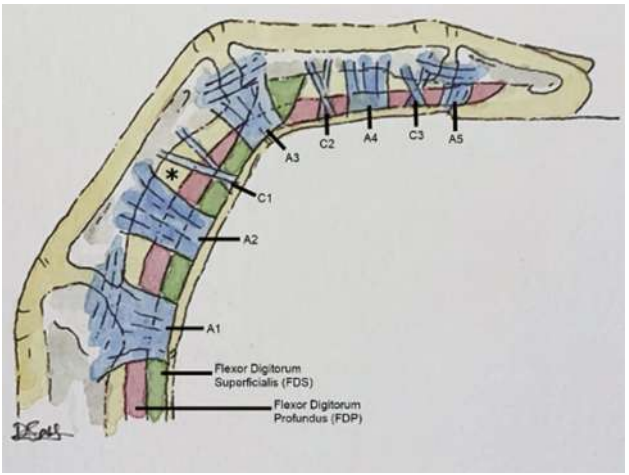

Figure 1. Normal anatomy of the finger flexor pulley system. Flexor tendons (flexor digitorum profundus and flexor digitorum superficialis) and associated annular (A1-A5) and cruciate (C1-C3) pulleys. Intact tendon-to-bone distance (*).

Fingers sustain the most overuse injuries in rock climbers.[7-9] Specifically, isolated closed injury of the finger flexor pulley system (FFPS) is a diagnosis rarely seen outside of rock climbers. FFPS injuries were first described in 1990, and the climbing community has been uniquely susceptible owing to the patterns of training and biomechanical demands on the hands in this sport.[10,11] Subsequently, numerous case reports document FFPS injuries as the most common overuse injuries of the upper extremities in rock climbers.[8-10,12-17] The most recent statistics of injury patterns show that finger injuries are the most common injury, accounting for 41% of all injuries.[7] Pulley injuries of the fingers were the most frequent diagnoses, representing 30% of the finger injuries and 12% of all injuries.[7] The shoulder was the second most common site of injury, accounting for 20% of all injuries.[7] As a further sports rarity, an isolated small case series documented FFPS injuries in baseball pitchers.[18]

Our goal is to summarize the available literature and describe current concepts in anatomy, biomechanics, clinical evaluation, imaging, prevention, and treatment strategies relating to FFPS injuries.

Anatomy

The finger flexor tendon system of the hand consists of a series of fibro-osseous sheaths through which the tendons of the flexor digitorum profundus (FDP) and flexor digitorum superficialis (FDS) course.[19] The sheath is composed of 2 distinct tissue components, synovial and retinacular (pulley), that serve distinct functions. The synovial component acts as "packing" that allows for movement of adjacent nondeformable tissues, while also lubricating surrounding cartilage. The retinacular or pulley component consists of localized thickenings of the sheath and serves to maintain the flexor tendons adjacent to the phalanges that promote biomechanical efficiency in finger flexion. The sheath, as a whole, prevents anteroposterior and lateral movement of the tendons from the axis of the finger.[20,21] The second through fifth digits contain 5 annular pulleys (A1–A5) and 3 cruciate pulleys (C1–C3) interposed segmentally throughout the digit, beginning in the distal palm and ending at the distal interphalangeal joints (Figure 1).[22] Annular pulleys are ring-shaped in configuration, whereas the cruciate pulleys have an eponymous cruciform shape.

Annular pulleys can be further subdivided into true fibro-osseous pulleys that insert directly into bone and those that insert onto the volar plate. The volar plate, found on the palmar aspect of the metacarpophalangeal and interphalangeal joints, is a ligamentous structure that functions primarily to prevent hyperextension of the digits. The A2 and A4 pulleys are broader, stronger, and insert directly into bone. Traditionally, they have been considered most important to prevent volar displacement of the flexor tendons away from the phalanges, a phenomenon termed "bowstringing."[22,23] Importantly, an intact FFPS may exhibit a small amount of bowstringing, termed "physiologic bowstringing." The A2 and A4 pulleys insert directly into the proximal and middle phalanges, respectively. The others (A1, A3, and A5) are less rigid and are not considered true fibro-osseous pulleys.[24] The A1, A3, and A5 pulleys are located over the metacarpophalangeal, proximal interphalangeal (PIP), and distal interphalangeal (DIP) joints, respectively.

The first cruciate pulley is located between the A2 and A3 pulleys and attaches to the proximal phalanx. The

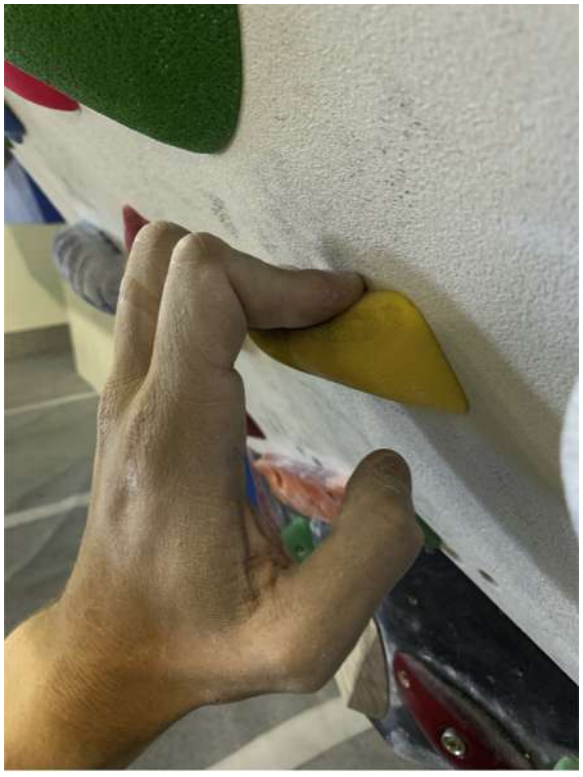

Figure 2. The crimp grip position.

second cruciate pulley is found between A3 and A4, distal to the PIP joint. The third cruciate pulley is found between A4 and A5, proximal to the DIP joint.[25] No pulleys are present directly over the distal phalanx.

The lumbrical muscles are intrinsic muscles of the hand that originate from the FDP and insert into the lateral aspect of the extensor tendon mechanism of the fingers.[26] They function to extend the PIP and DIP joints and flex the metacarpophalangeal joints.[26] Although a relatively small muscle, the proximity of the lumbricals to the FFPS renders them important in the grading of FFPS injuries.

Biomechanics

The high frequency of overuse injuries of the fingers in rock climbing can be explained by the sport's unique biomechanical demands. The upper extremities often support much of the climber's weight through a few fingers at a time. Specifically, the "crimp grip" is used in rock climbing to maximize contact of the fingertips on rock climbing holds with little surface area. Accordingly, the crimp grip is the mechanism for FFPS injuries.[27,28] The crimp grip is characterized by the PIP joints being flexed at about 90 degrees, while the DIP joints are maximally hyperextended (Figure 2).

Anatomically, the crimp grip position puts high levels of stress on the FDP and the FDS as they contract to maintain the conformation of the digits against the weight of the body.[29,30] The stress on the FDP and FDS, paired with flexion of the PIP, is counterbalanced by the flexor tendon pulleys as they bend in resistance. The highest tension is on the A2 pulley, with forces 3 to 4 times greater than at the distal phalanges.[24] As a result, the crimp grip position is associated with a higher incidence and distance of physiologic bowstringing compared with other common grip positions ("slope grip," "pinch grip") used by rock climbers.[27,31]

The A2 and A4 pulleys are the least deformable and have a higher breaking strength than the A1, A3, and A5 pulleys.[32,33] The A2 pulley typically is estimated to withstand forces of approximately 431 N, equivalent to nearly 45 kg (100 lbs) of force.[34] Recreational rock climbers can load the A2 pulley with forces between 380 N and 700 N, which equates to an average of 54 kg (120 lbs) of force.[27,35] These magnitudes of force are well in excess of the normal limit of the annular pulleys; therefore, with the repetitive supraphysiologic loading of the pulleys, overuse injuries occur with disruption of the pulleys.

Injuries

The list of differential diagnoses for an injured finger in a rock climber is broad, including injuries of the tendons, tendon sheath, bones, and FFPS.[36] We will focus on injuries of the FFPS. Injury of the FFPS in rock climbers was first described in a case report in 1990.[10,11] Since then, injuries of the annular pulleys in rock climbers have been documented extensively.[9,12,14-16,19] The fourth digit (ring finger) is the most commonly affected, followed by the third digit (middle finger).[37-39] Isolated A2 pulley ruptures are the most frequently reported of all significant finger injuries.[40,41]

The pattern of FFPS injury is thought to be progressive and predictable, due to the known biomechanical limits of the annular pulleys. Most often, the distal part of the A2 pulley becomes disrupted, and injury can progress from partial to complete rupture, even before the A3, A4, and A1 pulleys become involved.[42]

Biomechanical analyses have suggested that injury to the pulleys is most likely to occur when they are loaded eccentrically.[31,43,44] Given that the pulleys are not muscular tissue, eccentric loading in this context denotes the direction of movement of the finger (eg, extension that occurs in a sudden opening of the hand). This

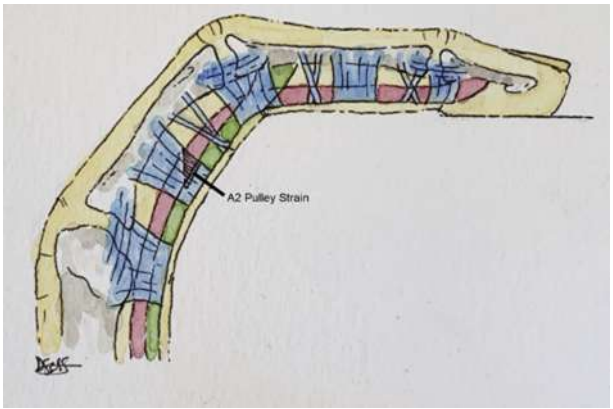

Figure 3. Grade I injury manifested as an isolated strain of the distal aspect of the A2 pulley (triangle).

suggests different pulley capacities under eccentric versus concentric loads.[31,43,44] The fourth digit A2 pulley strength is the weakest, whereas the A4 pulley strength is consistently stronger in the second digit (index finger).[33] Although rare, isolated cruciate pulley injuries also have been described in the literature.[45]

The grading system for FFPS has 4 levels of injury.[16] Grade I injuries include isolated pulley strains (Figure 3). Grade II injuries are those in which a complete rupture of A4, or partial rupture of A2 or A3, is present (Figure 4). Grade III is a complete rupture of A2 or A3 (Figure 5). Lastly, grade IV injuries are those that involve multiple pulley ruptures, or a single A2 or A3 rupture with involvement of a lumbrical muscle or surrounding collateral ligaments.

In summary, the A2, A3, and A4 pulleys have the highest risk of injury, and the A2 pulley is the most frequently injured. The fourth digit (ring finger) is most commonly affected. The risk of injury is highest in the crimp grip position, particularly with eccentric loading, owing to the increased biomechanical demands on the FFPS in this position.

Evaluation of Injury

Climbers who have sustained an FFPS injury report hearing a "pop" while grasping a climbing hold. More commonly, there is a sudden onset of pain and swelling over the affected pulley or pulleys, with possible numbness and local hematoma formation. Many climbers disregard the discomfort and continue climbing for weeks, with chronic pain over the volar aspect of the affected digits.[9]

In a 2006 systematic review, the most commonly recommended diagnostic criterion in all 29 papers was clinical "bowstringing" over the volar aspect of the PIP joint on physical examination.[46] Although diagnostic, bowstringing on physical examination does not characterize the extent of underlying injury. If it is the only finding, this does not allow prognostic determination. Furthermore, it is not an infallible diagnostic sign; some less severe injuries of the FFPS may not demonstrate clinically detectable bowstringing, thus yielding a false negative finding.[47] There is no documented relationship between the severity of FFPS injury and presence of clinical bowstringing on physical examination.

A report described an isolated complete A2 pulley rupture that was diagnosed simply by placing a silicone wedding ring over the region of the suspected injury. This resulted in a degree of restored range of motion, provided temporary relief by bolstering the ruptured pulley, prevented anterior movement of the flexor tendons, and confirmed disruption of the A2 pulley without the need for imaging evaluation.[48] However, this method does not reveal the extent of injury and may, if used alone as a method of diagnosis, lead to underdiagnosis of injuries that would have warranted surgical intervention. Furthermore, it is likely that the silicone ring, similar to conventional taping, may compromise the neurovascular bundle of the finger, rendering it a questionable, and perhaps harmful, diagnostic method.[49]

Clinical bowstringing, swelling, and tenderness over the anatomic pulleys are helpful signs on physical examination, but no detailed studies have confirmed their diagnostic accuracy, sensitivity, or specificity. Physical examination may be limited because of the presence of pain and soft tissue swelling, and often there is restricted range of motion of the digits. Furthermore, the degree of bowstringing does not predict the extent of injury accurately. Thus, given all these limitations, imaging evaluation should be obtained with these suspected injuries to characterize the injuries accurately.

Imaging

Imaging evaluation is indicated when a suspected injury of the FFPS is present to assess the integrity of the internal structures. Ultrasound (US) and magnetic resonance imaging (MRI) provide adequate visualization of the FFPS and are useful to confirm the diagnosis and degree of pulley injury. The distance between the flexor tendons and the phalanges, termed tendon-to-bone distance, traditionally has been used in both US and MRI as an indirect marker of FFPS integrity or disruption. The tendon-to-bone distance is an objective measurement used to quantify bowstringing, which, using proposed diagnostic thresholds, allows the physician to distinguish

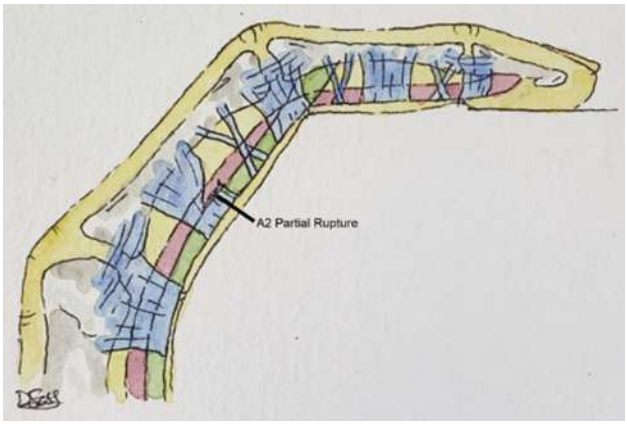

Figure 4. Grade II injury manifested as a partial rupture of the distal aspect of the A2 pulley.

between physiologic and pathologic bowstringing that results from a pulley rupture.

ULTRASOUND

US has been used to evaluate FFPS injuries since 1999, and it is the most widely used imaging technique to detect these injuries.[50] US is inexpensive, noninvasive, and more readily available than computed tomography and MRI. US permits dynamic evaluation of the digits in different degrees of flexion and provides live, direct visualization of the A1, A2, A3, and A4 pulleys and all cruciate pulleys.[51] The disadvantages of US include its accuracy being operator-dependent, and the space the US transducer occupies during the examination that sometimes limits PIP flexion to about 60 degrees.[42,46]

The normal flexor tendons are visible on US as hyperechoic fibrillar structures.[20] The appearance of the pulleys on US is variable, owing to anisotropy.[20] Anisotropy is an artifact that is seen in musculoskeletal US due to the ability of tissues to conduct or reflect soundwaves differentially based on the angle of incidence of the soundwaves. When the angle of the US beam is perpendicular to tissue being observed, the resultant image appears hyperechoic. Therefore, the volar surfaces of the pulleys generally are visible as thin hyperechoic bands surrounding the flexor tendons.[16,20,42] As the insonating US beam degree moves away from the perpendicular position, the resulting image becomes hypoechoic. This is why the lateral aspects of the pulleys often are hypoechoic.[20] A US example of a finger with an intact A2 pulley is shown in Figure 6.

Pulley injury is manifested on US directly as pulley discontinuity and indirectly as bowstringing of the flexor tendons (Figure 7).[52] Regarding specific tendon-to-bone distances used for thresholds to indicate injury, the

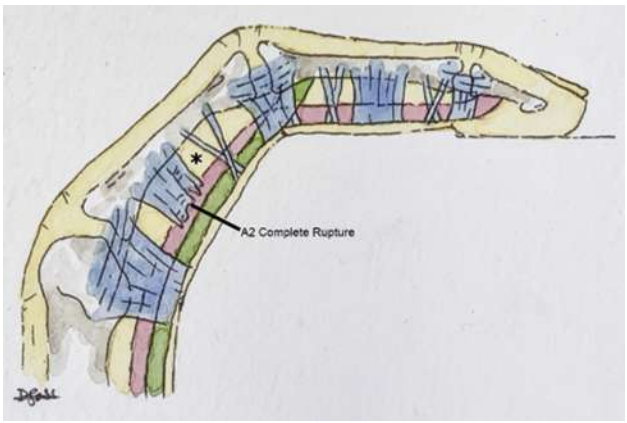

Figure 5. Grade III injury manifested as a complete rupture of the A2 pulley. As a result of the injury, bowstringing is seen, illustrated by the volar displacement of the flexor tendons from the phalanges, resulting in an increased tendon-to-bone distance (*) (compare to Figure 1).

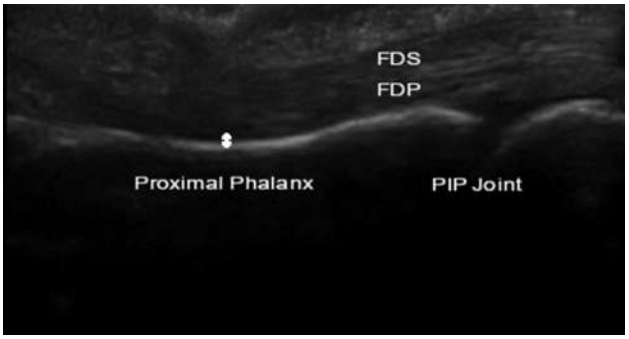

Figure 6. Intact A2 pulley. Ultrasound image in the longitudinal plane of a patient with an intact A2 pulley. Note that the flexor tendons are close to the proximal phalanx (double-sided arrow).

literature is inconclusive. Tendon-to-bone distances ranging from 1 to 3.3 mm have been recommended as sensitive thresholds to diagnose A2 and A4 pulley ruptures.[42,53-55] Despite the wide range of thresholds suggested in the literature, we prefer using a tendon-to-bone distance of >2 mm as an adequate, highly sensitive and specific threshold to diagnose A2 and A4 pulley ruptures. Diagnosis of A3 pulley ruptures via measurement of the distance between the volar plate and the flexor tendons has been suggested, with a distance >0.9 mm providing 76% sensitivity and 94% specificity.[56] The sensitivity and specificity of US to diagnose A2 and A4 pulley ruptures have been reported to be 90 to 98% and 98 to 100%, respectively.[16,56] Thus, US is highly sensitive and specific in diagnosing injuries of the FFPS and is the initial imaging technique of choice to evaluate these suspected injuries.

PLAIN FILM X-RAYS

Radiographs are unable to provide necessary visualization of the soft tissue FFPS in suspected pulley injuries. However, the routine use of radiographs in these patients may be useful to diagnose bony injuries, such as fractures and epiphyseal injuries.[54,57]

MRI

As with most soft tissue musculoskeletal injuries, MRI can provide valuable information about FFPS injuries. MRI was first demonstrated to be sensitive to diagnose FFPS injuries in the late 1990s and early 2000s; it is now widely accepted as a valid imaging technique for FFPS injuries.[42,50,58-60] Healthy flexor tendons have low signal on both T1 and T2 images.[25] A 3T MRI allows direct visualization of the annular pulleys, although the thinner cruciate pulleys are difficult to visualize.[25,61]

Abnormalities seen on MRI that may be suggestive of FFPS injuries include dehiscence of the flexor tendon from bone, discontinuity of the pulley tendons, hematoma between the phalanx and flexor tendon, and tilting of the flexor tendon toward the ruptured side.[16,25] MRI was reported to be 100% sensitive and specific to diagnose A2 and A4 injuries in cadaveric digits.[42]

When available, 3T MRI with dedicated surface coils allows for the highest resolution imaging of the digits.[25,62] Transverse images using gadolinium-enhanced and fat-saturated T1-weighted sequences provide the best visualization of the FFPS.[61,63] The use of crimp grip positioning during MRI evaluation enhances the sensitivity and specificity of pulley injury diagnoses.[64,65]

Semidynamic MRI of the digits, defined as consecutive images obtained in various degrees of flexion, may aid in the diagnosis of A3 pulley injuries.[66] Because of its insertion onto the volar plate, the A3 pulley places traction force on the volar plate during finger flexion. This force promotes palmar motion of the volar plate, and the distance created is termed the "volar plate translation distance." Therefore, palmar displacement of the volar plate is dependent on the integrity of the A3 pulley; decreased distances have been used to diagnose A3 pulley ruptures.[67] Volar plate translation distances of <2.8 mm and volar plate-tendon distances of >1.4 mm may be used as thresholds to diagnose A3 pulley ruptures.[56]

MRI may be preferred over US in clinical settings when experienced ultrasonographers are not available and when visualization of an injured pulley requires greater than 55 to 60 degrees of PIP joint flexion. MRI has the disadvantage of cost and an inability to provide dynamic imaging of the digits, as US is able to do. Despite its disadvantages, MRI is recommended as an additional imaging study, particularly if US is inconclusive.[16,46]

Prevention

A structured warm up as part of any rock-climbing session has been shown to increase the amount of

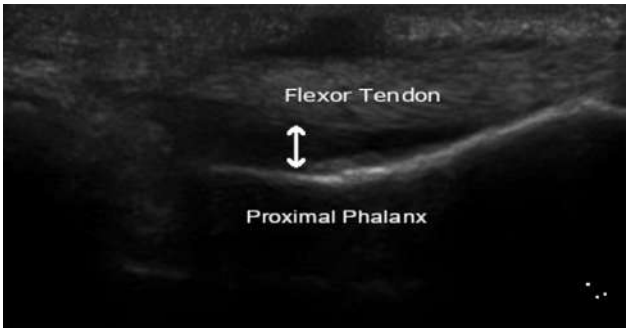

Figure 7. Ruptured A2 pulley. Ultrasound image in the longitudinal plane of a patient with a ruptured A2 pulley. Note the increased tendon-to-bone distance compared to Figure 6 (double-sided arrow).

physiologic bowstringing of the flexor tendons, specifically in the crimp grip position, by up to 30%.[27] Warming-up in rock climbing involves climbing routes of a less difficult grade than a climber may be able to climb normally. This is analogous to a runner, for example, who may warm-up by running a mile at a slower pace than normal. Specifically, a warm-up of 100 individual climbing moves of increasing intensity increases the amount of physiologic bowstringing.[27] Importantly, this increase in physiologic bowstringing has not been shown to prevent injuries from occurring and is only inferred to do so. Additionally, the author suggests that warming up may make the course of the flexor tendons more even, preventing peak forces at distinct points of the flexor tendon sheath.[27] Therefore, it is recommended to set a goal of 50 moves for each hand, for a total of 100 moves, with increasing intensity, as a proper warm-up to prevent pulley injuries.

When climbers experience discomfort over the area of the pulleys, some may circumferentially tape the affected finger at the level of the pulley in question, with the rationale that this will prevent a pulley injury (Figure 8). However, biomechanical evidence does not support the validity of this concept.[49,68] This style of taping decreases the amount of bowstringing by 3% and only absorbs approximately 10% of the force that is demanded from the pulley.[49] Another study found no statistical difference when testing the A2 pulley's load to failure in digits that were taped, compared with those that were not.[68] These studies used simple inelastic cloth adhesive tape.[49,68] Therefore, circumferential taping is probably an ineffective method to prevent injury. Nonetheless, a theoretical positive effect of circumferential taping is that flexion of the PIP joint is limited to less than 80 to 90 degrees if the tape is applied over the PIP joint, which limits the force acting on the A2 pulley.[49]

In addition to prevention, taping of the digits around the pulleys is also commonly done after injury. Circumferential taping, H-taping, and figure of 8 taping are 3 different taping techniques used. Their roles following a pulley injury will be discussed in the "Conservative Treatment" section.

Treatment

Less severe injuries of the FFPS allow for relatively unimpaired function and tend to self-resolve after a period of rest.[69] More extensive injuries that do not resolve with rest or that result in functional impairment require treatment. Both conservative and surgical treatment of FFPS injuries can be effective. Until 2003, there were no therapeutic guidelines for these injuries. Table 1 depicts the grading system with an accompanying therapeutic decision tree that was revised recently.[70] Owing to the lack of other literature on therapeutic guidelines, we will focus our discussion on that proposed grading system and treatment strategy.

CONSERVATIVE TREATMENT

The majority of pulley injuries are managed conservatively. Conservative strategies include immobilization and rest from climbing, anti-inflammatory agents, physical therapy, and pulley protection using different taping methods or a protective splint.[16]

The gradual return to climbing after a pulley injury always should be done under pulley protection. A device called the pulley-protection splint, specifically designed for conservative treatment of pulley ruptures, has shown good clinical results.[49] The device is recommended to be placed shortly after injury and to be worn for 6 to 8 wk.[49] When used correctly, the pulley-protection splint reduced the initial bowstringing at the A2 and A4 pulleys by 50 and 40%, respectively.[49] The device's unique shape is one of its main advantages over alternative methods, with collateral bulges designed to prevent compression of the

Figure 8. Circumferential taping over the A2 pulley of the third finger of the left hand.

Table 1. Pulley injury grading system and therapeutic guidelines (adapted from Lutter et al.[69])

	Grade I	*Grade II*	*Grade III*	*Grade IV a*	*Grad IV b*
	Pulley strain	Complete tear of A3 or A4, partial tear of A2	Complete tear of A2	Multiple ruptures: - A2/A3 or A3/A4 Rupture if: - No major clinical bowstringing - Ultrasound-proven possibility of reposition of the flexor tendon to the bone - Therapy starting <10 d after injury - No contracture	Multiple ruptures: - A2/A3 or A3/4 with obvious clinical bowstring - A2/A3/A4 rupture - Singular pulley rupture with FLIP phenomena - Singular rupture with increasing contracture - Singular rupture with secondary, therapy-resistant tenosynovitis
Therapy	Conservative	Conservative	Conservative	Conservative, if secondary onset of PIP contracture >20° secondary surgical	Surgical
Immobilization	None	Optional, <5 d	Optional, <5 d	Optional, <5 d	Post-surgical 14 d
Functional therapy with pulley protection (defined)	2–4 wk H-tape (during daytime) or thermoplastic ring (pulley protection splint)	6 wk thermoplastic pulley ring (pulley protection splint)	6–8 wk thermoplastic pulley ring (pulley protection splint)	8 wk thermoplastic pulley ring (pulley protection splint)	4 wk thermoplastic ring (after 2 wk of immobilization)
Easy sport specific activities	After 4 wk	After 6 wk	After 8 wk	After 10 wk	After 4 mo
Full sport specific activities	After 6 wk	After 8–10 wk	After 3 mo	After 4 mo	After 6 mo
H-taping during climbing	3 mo	3 mo	3 mo	>12 mo	>12 mo

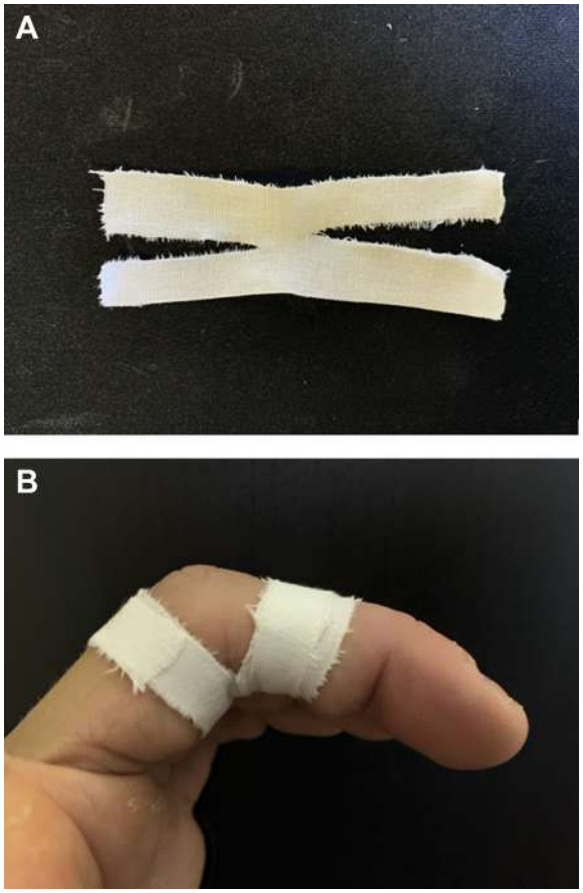

Figure 9. The H-tape before (a) and after application (b) on the third finger of the left hand.

surrounding neural and vascular supply of the finger, while still maintaining close apposition of the flexor tendons against the phalanx.[49]

The use of inelastic tape is the most widely used method to provide pulley protection after an injury. Taping of the fingers at the level of the A2 and A4 pulleys is thought to act as a replacement bolster for the injured pulleys. It is frequently done both to decrease the strain on an already injured pulley and to prevent further, or new, injury. Three different techniques of taping after a finger flexor pulley rupture have been studied: the circumferential, H-tape, and figure of 8 methods.

Circumferential taping around the proximal phalanx in the area of the affected pulley is only minimally effective in decreasing the demand on the injured pulley.[71] Circumferential taping has a greater, albeit still minimal, effect when the tape is applied over the distal end of the proximal phalanx, as opposed to the proximal end.[72] The figure of 8 method is applied by crossing the tape strips over the PIP on the palmar side of the digit.[71] Figure of 8 taping does not reduce the tendon-to-bone distance significantly. Therefore, the circumferential and figure of 8 taping methods are not recommended as a conservative treatment after pulley injury.[71]

The H-tape method has shown promising results in restoring the normal tendon-to-bone relationship and strengthening the injured finger.[73] The H-tape method focuses on placing tape circumferentially around the digit at the level of the A3 pulley in finger flexion (Figure 9). This method decreased the tendon to bone distance by 16% in an injured finger and is the only taping method to reach a statistically significant difference. The H-taping method also resulted in a 13% increase in strength of the injured fingers in the crimp grip position, compared to other taping methods.[73] Leukotape was used in this study.[73] Taping of the injured finger with the H-taping method should be done for 3 mo in grade I to III injuries and for at least 12 mo in grade IV injuries.

The H-tape method is the only method that has been shown to be effective in decreasing the tendon-to-bone distance and increasing the strength of the injured finger.[73] It is recommended after a pulley injury.[73] There is no evidence to support the use of H-taping for prophylaxis of these injuries, however.

SURGICAL TREATMENT

Surgical repair of pulleys is reserved for patients with multiple closed pulley ruptures, persistent pain, or dysfunction after 2 mo of conservative management, or when there is a flexion contracture as a result of the pulley injury.[74] These are all grade IV injuries, according to the aforementioned grading system. Although surgery is the acknowledged treatment of choice for grade IV injuries, occasionally they can be managed conservatively.

Surgical repair restores the tendon to joint relationship, while also providing good biomechanical outcomes.[75] Several different pulley reconstruction techniques have been described, including the "belt-loop," the "single loop," the "loop and a half," and the "triple loop" techniques.[76-79] Pulley reconstructions are performed with grafts, or most commonly with a combined repair technique.[80-82] Nevertheless, these encircling techniques can lead to atrophy of the cortex of the proximal phalanx.[83]

Postoperative care generally includes immobilization via a ring splint, such as the pulley-protection splint. The

duration for ring splinting is not well defined, with recommendations ranging from 14 d to 6 wk.[57,79] We recommend full arm and finger splinting for 2 wk after surgery, followed by 4 wk of pulley splinting. Recommendations regarding when to initiate finger motion therapy and the use of postoperative supportive taping vary widely.[68,72,73]

Summary

FFPS are the most commonly diagnosed overuse injuries in rock climbers. FFPS injuries have increased over the past 2 decades as a result of the sport's sustained growth and popularity. The crimp grip used in rock climbing is the mechanism for these injuries, due to the supraphysiologic demand of the annular pulleys in this position. The A2, A3, and A4 pulleys are at the highest risk of injury, especially when loaded eccentrically.

Evidence of FFPS injuries on physical examination includes tenderness over the anatomic pulleys when the patient reports pain and, in some cases, clinical bowstringing. Physical examination of a suspected FFPS injury often is clouded by limited range of motion and soft tissue swelling. For these reasons, physical examination alone is insufficient for diagnosis and extent, hence the need for imaging.

Imaging evaluation permits visualization of the FFPS and is imperative to establish the correct diagnosis and extent of injury. US is the preferred initial imaging technique for suspected injuries of the FFPS system because it is highly sensitive and specific and provides direct visualization of the structures in question in a dynamic fashion. MRI is the recommended additional imaging study if US is inconclusive.

Warming up appropriately is critical and is the only approach thought to prevent injuries of the FFPS.[27] Treatment of grade I to III injuries generally is conservative, with surgical intervention reserved for grade I to III injuries that are refractory to treatment, or any grade IV injury not amenable to conservative treatment. The use of the pulley protective splint or taping the digits in the H-tape method after injury decreases the likelihood of further injury and provides adequate support to the damaged pulleys. In surgical cases, the involved digit should be immobilized and splinted for at least 2 wk postoperatively, and many authors recommend even longer durations of splinting.

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Letters to the Editor

Possible Indirect Adverse Effects to the Eyes in Skiers Wearing Helmets

To the Editor:

Use of helmets among skiers started to increase some 20 y ago, becoming mandatory for children under 16 y old in Austria in 2009.[1] Today, 86% of skiers wear helmets.[2] Helmet use has reduced the risk of head injuries by 35%.[3] Widespread use of helmets implies thorough study of the possible adverse effects of wearing helmets that could offset their protective effects. Helmet use does not increase the risk of neck or cervical spine injury or the risk of compensation behavior.[4] However, the 2 groups of head injuries, traumatic brain injury and other types of head injuries, are not equally prevented by ski helmets, and the number of traumatic brain injuries is not reduced to the same extent as the number of other types of head injuries.[5] Over time, the protective effects of helmets have been somewhat reduced.[6] A direct influence of ski helmets on hearing was investigated and established.[7]

In addition to helmets, usual head gear for skiers includes ski goggles. Their role is to prevent ultraviolet (UV) radiation (wavelengths smaller than 400 nm) from reaching the eye and the surrounding soft tissue; to reduce the total amount of light reaching the eye; to reduce the production of tears; to enhance contrast; and to provide mechanical protection of the eyes and the surrounding soft tissue. Optimal performance depends on ventilation of humid air. Proper ventilation prevents fogging and should not induce the overproduction of tears. Helmets tend to reduce goggle ventilation; therefore, goggles fog much more easily, and when skiers are moving at reduced speed or when they stop moving, fogging becomes almost inevitable. This is probably one of the principal reasons why many helmeted skiers remove their goggles when they stop moving; when using chair lifts, drag lifts, and gondolas; and sometimes during descent.

There are, however, skiers who use an extra pair of sunglasses in situations when goggles are removed, and a certain number who use sunglasses exclusively. All the aforementioned personal observations were confirmed by the inspection of photos from 12 large ski resorts in Austria, Italy, France, and Bulgaria. The largest proportion of skiers, more than 50%, remove their goggles at the base stations of lifts and approximately 30% when using chairlifts, whereas skiers in descent rarely remove them. At the top stations of ski lifts, the percentage of skiers with removed goggles is nearly the same as among chairlift users. Approximately 10% of skiers were wearing sunglasses and helmets (skiers with removed goggles included). There were no skiers with removed sunglasses. Goggles and sunglasses do not protect the eyes and the surrounding tissue to the same extent because goggles cover a larger area and have a tighter fit.

The effects on the eyes of removing goggles and not replacing them with sunglasses is that they are exposed to increased levels of UV radiation in situations when the UV index is usually very high. As a result, adverse effects of UV radiation are possible. The most notable adverse effects include snow blindness as an acute effect; cataract and macular degeneration are chronic effects.[8] Owing to increased sensitivity to UV radiation among children, the removal of ski goggles could exacerbate such effects.[9]

Although eye injuries are just a fraction of head injuries, in the only identified study covering skiing, one-third of eye injuries were acute injuries caused by UV radiation.[10] There are no data on the long-term effects of UV radiation to the eyes in skiing.

The almost uniform habit of wearing ski helmets amplifies the importance of addressing possible adverse effects of UV radiation to the eyes of helmeted skiers who remove their goggles while skiing. The proportions of skiers with removed goggles in different situations suggest the possibility that many skiers remove their goggles after descent and put them on just before descent. Information about the high intensity of UV radiation at high altitudes and snow-covered slopes, as well as the need for permanent eye protection, should be promoted. Because chronic effects to the eyes emerge after many years and the effects are not immediately recognized as acute, it is important that skiers be aware of the risk.

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Toxic Brain Edema and Brain Death After Scorpion Envenomation

To the Editor:

Scorpion sting is common in some global regions and mostly causes local envenoming of varying severity. Some estimates suggest that perhaps only 10% of those stung by even the most medically important species develop severe systemic envenomation; however, the frequency of severe envenomation is higher in children. With few exceptions, systemic envenomation by medically important species is characterized by relatively similar neurotoxic stimulation syndromes.[1]

Here, we report a 12-y-old girl who developed toxic encephalopathy and lethal brain edema owing to scorpion envenomation (*Leiurus abdullahbayrami* [Scorpiones: Buthidae]). She was transferred to our pediatric intensive care unit from another center after 2 d of follow-up. Based on the documents provided by the other medical unit, the patient was intubated 1 to 2 h after the scorpion sting owing to confusion and respiratory distress and was given tetanus vaccine, 1 vial of antivenom (polyvalent antiscorpion antivenom, Refik Saydam Hygiene Center, Ankara, Turkey), and doxazosin therapy in the initial treating facility. Because the patient experienced cardiac arrest for 5 min, cardiopulmonary resuscitation was performed. When the ejection fraction of the left ventricle was 30% on echocardiography, she was referred to us with a diagnosis of toxic myocarditis.

Immediately after admission to our unit, she was intubated and had decompensated shock findings. Her Glasgow Coma Scale score was 6 of 15. Blood gas findings showed mild acidosis. Laboratory results showed white blood cell levels were $15.73\times10^{3}\cdot\mu L^{-1}$, hemoglobin was 11.9 $g\cdot dL^{-1}$, platelets was $254\times10^{3}\cdot\mu L^{-1}$, serum creatinine was 0.87 $mg\cdot dL^{-1}$, blood urea nitrogen was 24 $mg\cdot dL^{-1}$, aspartate aminotransferase was 244 $U\cdot L^{-1}$, alanine aminotransferase was 71 $U\cdot L^{-1}$, creatinine kinase was 8191 $U\cdot L^{-1}$, troponin T was 1924 $pg\cdot mL^{-1}$ (normal range: 0–14), N-terminal pro-brain natriuretic peptide was >35 $ng\cdot L^{-1}$, and CK-MB was 75 $ng\cdot mL^{-1}$ (normal range: 0–3.6); international normalized ratio was 1.59; activated thromboplastin time was 24.9 s; prothrombin time (PT) was 18.4 s; D-dimer was 3789 $ng\cdot mL^{-1}$ (normal range: 0–243); and fibrinogen was 5.58 $g\cdot L^{-1}$ (normal range: 2–3.93).

To stabilize the patient, we continued with maintenance fluid, milrinone, and doxazosin therapies and dobutamin to correct hypotension. On the first day of admission, the patient was given 1 more vial of Refik Saydam Hygiene Center polyvalent antiscorpion antivenom therapy. On echocardiography, the ejection fraction was 45 to 50%. Cranial computed tomography (CT) was performed on the third day of admission, with decreased alertness and a Glasgow Coma Scale score of 4. The CT report revealed existing widespread hypodense areas in both cerebral hemispheres, loss of gray and white matter separation, and deletion in sulcal structures compatible with edema (owing to possible toxicologic effects) (Figure 1). Because the patient had brain edema, 0.1 $cm^{3}\cdot kg^{-1}\cdot h^{-1}$ 3% NaCl was started. The patient's neurologic condition worsened gradually. On the 10th day of hospitalization, neurologic examination revealed absence of all brainstem activity. CT angiography was performed, and the report was compatible with brain death.

It has been reported that after scorpion sting, cortical disruption may develop before other organ disruptions.[2] One study with 9 fatal cases documented central nervous system (CNS) symptoms before terminal hypotension and cardiac arrest.[3] Lethal cerebral edema occurred in a 2-y-old male patient who had diffuse brain edema and ischemic changes on CT after scorpion sting and was later reported to be diagnosed with brain death.[4] Similarly, our patient had diffuse brain edema

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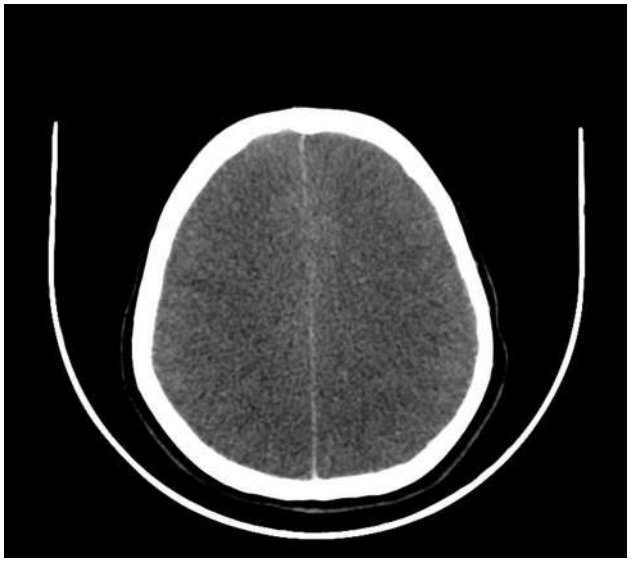

Figure 1. On the third day of admission, cranial computed tomography revealed widespread hypodense areas in both cerebral hemispheres, loss of gray and white matter separation, and deletion in sulcal structures compatible with severe edema.

(owing to possible toxicologic effects) on CT and was diagnosed with brain death on the 10th day of admission. CNS pathophysiologic effects have been hypothetically assigned to venom penetration through the blood-brain barrier because of possible increased permeability in young children.[3] Symptoms of systemic envenomation may be caused by the venom (toxins) itself or neurotransmitters (catecholamines) and proinflammatory cytokines released owing to the venom.[4]

In conclusion, we believe that this patient had unusually severe brain damage either owing to venom-related direct CNS toxicity (increased permeability of the blood-brain barrier, affecting the CNS neurons) or the secondary effect of proinflammatory cytokines and neurotransmitters affecting blood vessels, which induced brain ischemia and cytolytic brain edema.

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Methoxyflurane May Be a Suitable Analgesic for Extreme Prehospital Conditions

To the Editor:

Methoxyflurane (MEOF) is a fluorinated hydrocarbon anesthetic that was introduced to the market by Abbott Laboratories as Penthrane in the early 1960s. MEOF was found to have analgesic properties at subanesthetic doses. A disposable inhaler was developed for self-administration of MEOF for pain relief in minor surgical procedures and obstetrics in the late 1960s. In 1966, the first report of postoperative nephrotoxicity associated with MEOF emerged. Although MEOF has been withdrawn for use as an anesthetic agent, low-dose MEOF delivered via a handheld inhaler has continued to be in wide use in Australia and New Zealand since 1975. MEOF as an analgesic has been used by more than 6 million people over the course of 40 y in Australia and New Zealand. Common nonserious adverse effects are reactions such as headache, nausea, dizziness, and somnolence. Generally, adverse effects are mild and transient, resolving after inhalation is stopped. MEOF administered at analgesic doses via a disposable inhaler has recently become available in Europe for emergency treatment of moderate-to-severe pain in conscious adult trauma patients.

A recent study demonstrates that prehospital pain management is characterized by ongoing oligoanalgesia owing to undelivered analgesia.[1] Data from the control arms of the studies that compared MEOF to standard

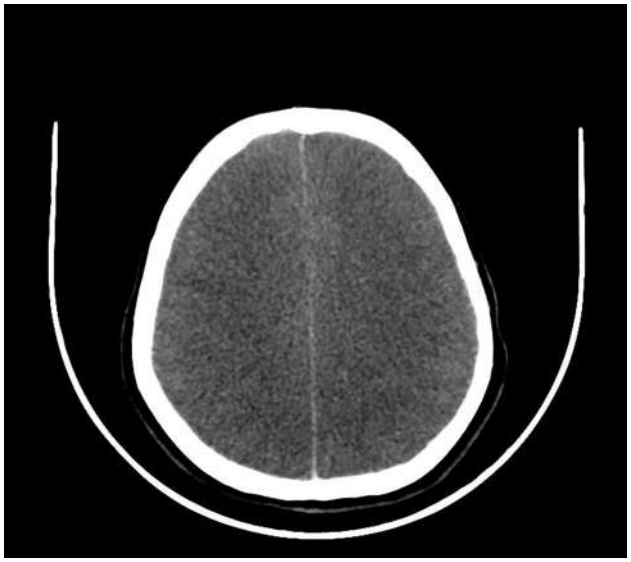

Figure 1. On the third day of admission, cranial computed tomography revealed widespread hypodense areas in both cerebral hemispheres, loss of gray and white matter separation, and deletion in sulcal structures compatible with severe edema.

(owing to possible toxicologic effects) on CT and was diagnosed with brain death on the 10th day of admission. CNS pathophysiologic effects have been hypothetically assigned to venom penetration through the blood-brain barrier because of possible increased permeability in young children.[3] Symptoms of systemic envenomation may be caused by the venom (toxins) itself or neurotransmitters (catecholamines) and proinflammatory cytokines released owing to the venom.[4]

In conclusion, we believe that this patient had unusually severe brain damage either owing to venom-related direct CNS toxicity (increased permeability of the blood-brain barrier, affecting the CNS neurons) or the secondary effect of proinflammatory cytokines and neurotransmitters affecting blood vessels, which induced brain ischemia and cytolytic brain edema.

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Methoxyflurane May Be a Suitable Analgesic for Extreme Prehospital Conditions

To the Editor:

Methoxyflurane (MEOF) is a fluorinated hydrocarbon anesthetic that was introduced to the market by Abbott Laboratories as Penthrane in the early 1960s. MEOF was found to have analgesic properties at subanesthetic doses. A disposable inhaler was developed for self-administration of MEOF for pain relief in minor surgical procedures and obstetrics in the late 1960s. In 1966, the first report of postoperative nephrotoxicity associated with MEOF emerged. Although MEOF has been withdrawn for use as an anesthetic agent, low-dose MEOF delivered via a handheld inhaler has continued to be in wide use in Australia and New Zealand since 1975. MEOF as an analgesic has been used by more than 6 million people over the course of 40 y in Australia and New Zealand. Common nonserious adverse effects are reactions such as headache, nausea, dizziness, and somnolence. Generally, adverse effects are mild and transient, resolving after inhalation is stopped. MEOF administered at analgesic doses via a disposable inhaler has recently become available in Europe for emergency treatment of moderate-to-severe pain in conscious adult trauma patients.

A recent study demonstrates that prehospital pain management is characterized by ongoing oligoanalgesia owing to undelivered analgesia.[1] Data from the control arms of the studies that compared MEOF to standard

Table 1. Summary of inhaled methoxyflurane study design in patients with trauma pain

Study	*Design*	*Primary endpoint*	*Conclusion*
Coffey et al. (2014)[4] NCT01420159 Medical Developments International	Phase 3 randomized, double blind, multicenter, placebo-controlled trial; United Kingdom; 300 patients (149 MEOF)	Change in pain intensity as measured using the VAS from baseline to 5, 10, 15, and 20 min after the start of study drug inhalation	MEOF administered with the inhaler is an efficacious, safe, and rapidly acting analgesic
Borobia et al. (2020)[2] NCT03256903 Mundipharma Pharmaceuticals	Phase 3b randomized, active-controlled, open-label, parallel-group trial performed in 13 emergency department and 1 out-of-hospital unit; Spain; 305 patients (156 MEOF)	Change from baseline in NRS pain intensity score during the first 20 min of treatment and time to first pain relief	MEOF is a nonnarcotic, easy-to-administer, rapid-acting, first-line alternative to currently available analgesic treatments for trauma pain
Mercadante et al. (2019)[8] NCT03585374 Mundipharma Pharmaceuticals	Phase 3b randomized, open-label, active-controlled, multicenter trial; Italy; 272 patients (136 MEOF)	Change in VAS pain intensity from baseline to 3, 5, and 10 min	MEOF provided superior pain relief compared with standard analgesic treatment in patients with moderate-to-severe trauma pain and may offer a simple, fast, effective, nonopioid treatment option
Lim et al. (2020)[5] NCT01887951	Prospective, phased, cluster-randomized crossover study; Singapore; 369 patients (167 MEOF)	Reduction in pain as measured by reduction in NRS at 5, 10, 15, and 20 min after treatment; time taken from arrival at scene to administration of treatment	MEOF was superior to 50 mg intramuscular tramadol in terms of analgesic efficacy, onset of effective analgesia, and speed of administration. It was also associated with better paramedic and patient satisfaction
Ricard-Hibon et al. (2020)[3] NCT03798899 Mundipharma Pharmaceuticals	Phase 3b randomized, open-label, multicenter, placebo-controlled trial; France; 351 patients (178 MEOF)	Time until pain relief ≤30 mm, as assessed on the VAS	MEOF, initiated by triage nurse as part of a multimodal analgesic approach, is effective in achieving pain relief for trauma patients. This effect was particularly pronounced in the severe pain subgroup
Marinangeli et al. (2018) (study still in progress)[6] Mundipharma Pharmaceuticals	Phase 3b prospective, single-arm, multicenter trial; Approximately 200 adult patients with a pain score of at least 4 on the NRS owing to limb trauma who were rescued by HEMS will be enrolled. Patients will receive up to 2×3 mL MEOF, self-administered by the patient by inhalation under medical supervision. Rescue medication will be permitted if required.		

HEMS, helicopter emergency medical services; MEOF, methoxyflurane; NRS, numeric rating scale; VAS, visual analog scale.

treatment revealed that trauma pain is often undertreated in current clinical practice, and there is no established standard of care. In the first randomized trial of MEOF in the emergency setting in Europe, only 23% of patients with severe pain (>7 on the numeric rating scale) received opioids in the standard-of-care arm or as a rescue medication.[2] This was also reported in a French study in which 31 patients, 18% of the control arm, did not achieve pain control.[3]

A growing body of evidence demonstrates that inhaled MEOF is well tolerated and effective in providing rapid onset of analgesia. However, few randomized controlled trials have been carried out regarding the matter. There are now several European randomized trials, summarized in Table 1. It is regrettable that 4 of these 5 trials are sponsored by the same pharmaceutical group; this should encourage great caution in interpreting the results and developing new protocols.

It would appear that MEOF is an efficacious, safe, and rapidly acting analgesic; is easy to administer as a first-line alternative to currently available analgesics; and is associated with better paramedic and patient satisfaction.[2,4,5]

The use of MEOF in hostile environments, where patient mobilization is difficult, is of particular interest because of MEOF's ease of use and speed of action. To assess whether inhaled MEOF is effective, safe, and practical in treating trauma pain in remote and hostile environments, a prospective, single-arm, multicenter trial was designed. This Italian study is currently ongoing and enrolling approximately 200 patients with single-limb trauma.[6] The first patient was enrolled in March 2019.

In prehospital settings, where interventions are performed by a variety of professionals where and physicians may not be present, administration of MEOF is practical, with a safe administration route and rapid onset of action. The inhaler is lightweight, robust, and easy to use, even with gloved hands, and it requires minimal training for effective use. It can be easily transported and stored at a wide range of temperatures (−20 to 40°C). In a case report, MEOF provided rapid, effective analgesia for visceral and procedural pain at an altitude of 4470 m.[7] Currently, treatment is to be supervised by a healthcare professional and is self-administered through patient-controlled analgesia. MEOF may be particularly relevant in patients who are able to self-administer analgesia while isolated in hostile environments.

Low-dose MEOF administered via a handheld inhaler has provided well-tolerated, short-term pain relief for trauma patients for many years. The availability of a nonnarcotic, well-tolerated, rapid-acting, and effective treatment option may improve acute trauma pain management. The ease of use of MEOF may make it particularly useful in the management of acute pain in hostile environments. MEOF could be a suitable option in the following situations:

- For emergency prehospital doctors—more specifically, when involved in mountain rescue
- For paramedics and mountain rescuers while waiting for the arrival of medicalization or transfer to the hospital
- For the victim, in the context of self-medication, particularly in isolated environments

To date, several randomized trials support the use of MEOF, and it is probably time to carry out a meta-analysis on this subject.

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Can't See the Wood for the Trees

To the Editor:

While revising for examinations recently, I found myself staring out the window at the trees and an unusual thought occurred to me. I noticed during the clinical examination of compartment syndrome, uterine rupture, and Ludwig's angina, several sources use the tactile sensation of "woody tense" to describe all of these different entities. In university, I remember learning the subtleties of breath sounds with a stethoscope, but I never recall being schooled on the sensation of wood. A literature review demonstrated no fewer than 8 different serious medical conditions associated with the same "woody tense" sensation from thyroiditis to zygomycosis and from priapism to pyomyositis.[1-6] Based on this review, I concluded that the sensation of "woody tense" has a strong association with potentially dangerous conditions. This similar clinical finding in multiple serious pathologies is an unusual phenomenon. The sensitivity and specificity of this finding is unknown and will vary from condition to condition. I believe the description of this phenomenon is unique.

As medical professionals, we are increasingly reliant on diagnostic technologies, and this has potentially allowed us to forget this simple pearl of the clinical examination, lost in the tide of time. Our ignorance of this phenomenon somewhat reflects our treatment of wood and trees in general. Since the dawn of our species, we have used trees as shelter; as tools for hunting, protection, and war; and for warmth and cooking. We have exploited trees to advance society tremendously. Now we reject them, choosing to live lavishly, consuming and wasting in densely populated urban areas. Increasing population and urbanization coupled with politico-economic policies of mass production and consumption have led to deforestation on an enormous scale. These factors are exacerbating climate change, increasing our susceptibility to transmissible disease, worsening our general health and happiness, and will eventually lead to the end of our species.

In antithesis to urban areas, simply walking in the woods or "forest bathing" has been demonstrated to have profound benefits for physical and mental health and has been practiced in Japan for centuries.[7] For the happiness and health of the human race, perhaps we all should return to the trees. Certainly, the medical students should.

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Book Review

The Mohave Rattlesnake—And How it Became an Urban Legend
Mike Cardwell
Rodeo, NM: ECO Publishing, 2020
US $14.95, 138 pages, paperback

This short but information-laden and richly illustrated book provides a summary of the biology and behavior of the Mohave rattlesnake. Resident to the Mohave, Sonoran, and Chihuahuan Deserts of the American Southwest and Northern Mexico, the Mohave rattlesnake, *Crotalus scutulatus* (commonly known as the Mohave green) is one of more than 30 species of rattlesnake native to North America, but one with an especially sensational folklore.

Encompassing 14 chapters and a substantive bibliography and glossary, the book's nontechnical but authoritative text will resonate with experts and nonexperts alike. The narrative reads quickly and is abundantly supplemented with clear illustrations and outstanding photographs, most of which were taken by the author. Although it focuses on the Mohave rattlesnake, it offers many insights into rattlesnakes broadly.

Individual chapters cover the naming, taxonomy and identification, distribution, behavior, ecology, and evolution of the Mohave rattlesnake. Chapters 5, 9, 13, and 14 may be of most interest to clinicians insofar as they discuss the venomology, biting behavior, and first aid and medical management of bites. While underscoring their potential seriousness, the author debunks claims that bites from this species have exceptional lethality. The discussion of antivenom succinctly reviews its evolution from polyvalent Crotalidae antivenom to CroFab to AnaVip and issues regarding its use for bites from this species. However, clinicians treating crotalid envenomation, Mohave or otherwise, should consult more detailed clinical references (eg, Auerbach's *Wilderness Medicine*, seventh edition) for more specific guidance.

An especially useful feature of this book is its focus on distinguishing fact from fiction about what it calls "America's most misunderstood rattlesnake." Chapters 10, 11, and 12 especially discuss misinformation and myths (including some conspiracy theories) surrounding Mohave rattlesnakes and convincingly put many of the urban legends to rest.

This is a well-written and visually pleasing book that will both inform and entertain armchair and expert herpetologists, first responders, and clinicians who may be called upon to manage rattlesnake bite victims, and all manner of outdoor enthusiasts recreating where Mohave rattlesnakes live.

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WILDERNESS IMAGE

Fatal Avalanche on Mount Everest

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On the morning of May 7, 2009, a section of the hanging glacier on the west shoulder of Mount Everest broke free, creating this avalanche that spread across the Khumbu icefall. Avalanches are common in this area. Climbers in Everest Base Camp become accustomed to hearing distant avalanches and somewhat desensitized to it, in the same way that a solder in combat might become accustomed to nonthreatening distant mortar explosions. However, the sound and proximity of this avalanche caused everyone to take notice immediately. Unfortunately, Lhakpa Nuru Sherpa, age 33 y, perished in this avalanche.

Photo taken from Base Camp (GPS: 28°00'15.60" N and 86°51'20.39" E).