and the web space had a complex laceration. The wound was irrigated with a Pulsavac (Zimmer Inc, Swindon, UK), and with fluoroscopic guidance, 2 cross-oblique 0.035 K wires were placed across the fracture site. The extensor mechanism was repaired with 5-0 Ethibond sutures, and the skin was closed using 4-0 interrupted nylon sutures. The total length of the closure was 8 cm. The hand was placed in a splint for protection. The patient was later discharged home with no further antibiotics. At follow-up the patient was started on active range of motion of the left hand with occupational therapy. Six weeks later, the pins were removed. The patient recovered well, with full function of her left hand and complete recovery of her fourth finger.

The trajectory force from the tine of the deer antler affected the patient's left ring finger and resulted in an open proximal phalanx fracture. Any accident caused by contact with a deer carries a heightened possibility for transmission of infectious disease. The most common infectious diseases associated with deer collisions include *Arcanobacterium pyogenes*, *Haemophilus contortus*, *Salmonella*, *Mycobacterium avium*, and *Paratuberculosis*.⁵

About 13% of road-killed deer exhibit debilitating conditions predisposing them to motor vehicle collisions.⁵ The typical problems revealed during necropsy include chronic purulent infections involving the cranial region, usually intracranial abscesses and/or purulent meningoencephalitis. At necropsy many deer have evidence of missing antlers, open wounds, or scars around the antlers or atypical antlers secondary to structural fragility. Other bacteria cultured from wounds in close proximity to the deer antlers have included *Bacteroides*, *Citrobacter*, *Corynebacterium*, *Enterobacter*, *Pasteurella multocida*, *Serratia marcescens*, *Staphylococcus aureus*, and *Staphylococcus sciuri*.⁵ Given this vast range of bacteria, a broad antibiotic coverage, to include gram negatives and anaerobes, would be an appropriate regimen in patients injured by deer. The literature is limited with regard to the surgical treatment of patients after traumatic contact with deer, and the optimal duration of antibiotic therapy for grossly contaminated wounds is still controversial. Generally a 3-day course is recommended, though 24 hours of antibiotics may be sufficient.⁶ However, if the injury or wound site becomes infected, a 14-day course of treatment should be considered for soft tissue wounds and a 21-day course for infections involving joints or bones.⁷ For prophylaxis, all such patients should receive tetanus prophylaxis as indicated, aggressive irrigation of wounds, and an antibiotic regimen with good gram-negative and anaerobic coverage. The antibiotics should include a third- or fourth-generation cephalosporin, such as ceftriaxone or

cefepime, respectively, for broad gram-positive and gram-negative coverage and a tetracycline, such as doxycycline or minocycline, for anaerobic and atypical coverage. An alternative for penicillin- or cephalosporin-allergic patients would be a fluoroquinolone such as ciprofloxacin or levofloxacin.

In summary, for a patient presenting to the emergency department within 3 hours of an open fracture from a penetrating deer antler injury, we recommend a one-time prophylactic dose of intravenous ceftriaxone (2 g) and oral doxycycline (100–200 mg).

Martina Okwueze, MD
Troy Thompson, MD
Marcia Spear, ACNP
Lillian Nanney, PhD
Nashville, TN, USA

Sean Wolford, MD
Johnson City, TN, USA

References

1. Haikonen H, Summala H. Deer-vehicle crashes extensive peak at 1 hour after sunset. *Am J Prev Med*. 2001;21:209–213.
2. Ashton A. Insurance group estimates annual costs of deer-related traffic accidents. *The Miami Herald*. November 4, 2003. Available at: <http://lists.envirolink.org/pipermail/ar-news/Week-of-Mon-20031103/009466.html>. Accessed online January 8, 2007.
3. Sigler B, Philips J, Agent K, et al. Effectiveness in disease and injury prevention injuries from motor-vehicle collisions with deer—Kentucky, 1987–1989. *MMWR*. 1991;40:717–719.
4. Rattey TE, Turner NE. Vehicle-moose accidents in Newfoundland. *J Bone Joint Surg*. 1991;73:1487–1491.
5. Nettles VF, Quist CF, Lopez RR, et al. Morbidity and mortality factors in key deer (*Odocoileus virginianus clavium*). *J Wildl Dis*. 2002;38:685–692.
6. Zalavras CG, Patzakis MJ, Holtom PD, Sherman R. Management of open fractures. *Infect Dis Clin North Am*. 2005;9:915–929.
7. Benson LS, Edwards SL, Schiff AP, Williams CS, Visotsky JL. Dog and cat bites to the hand: treatment and cost assessment. *J Hand Surg Am*. 2006;31:468–473.

Wild Flowers and Mad Honey

To the Editor:

One unusual form of food poisoning, commonly seen in the eastern Black Sea region, is caused by toxic honey, which is familiar to local people and which is known as

Figure 1. *Rhododendron ponticum*, also known as the “mountain rose” or “black poison.”

“mad honey.” It has been known to be poisonous since 401 BC.¹

Grayanotoxins, which are extracted by bees from the flowers of rhododendron species, are the main compounds responsible for this poisoning. Ericaceae is one of the largest dicotyledon genera. The name “rhododendron” derives from the Greek *rhodos*, meaning “rose,” and *dendron*, meaning “tree.” There are some 700 different species in the area comprising China, Tibet, Myanmar, Assam, and Nepal; nearly 300 species in New Guinea; many in Japan; others in tropical Asia from Indochina to Indonesia and the Philippines; and a small number occur in Europe and North America. Purple-flowered (*Rhododendron ponticum*) (Figure 1) and yellow-flowered (*Rhododendron luteum*) (Figure 2) rhododendrons have become pernicious woody weeds in northern Turkey.² The former are also known as “black poison” and the latter as “yellow poison.” As a result of the grayanotoxins they contain, the flowers of these 2 rhododendron species, which are widely found in the mountains that extend along the Black Sea coast of northern Turkey, may lead to poisoning of honey made by local producers and produced by wild bees. The grayanotoxins bind to the sodium channels in cell membranes. These compounds prevent inactivation; thus, excitable cells (nerve and muscle) are maintained in a state of depolarization, during which entry of calcium into the cells may be facilitated.³

Toxic *Rhododendron* species, particularly *R. ponticum*, are commonly used in folk medicines of the Black Sea region. *R. ponticum* is widely used both internally and externally as an analgesic for treatment of rheumatic or dental pain, common viral upper respiratory infections, and edema.⁴ “Mad honey” is used as an alternative medicine for the treatment of gastric pains, bowel dis-

Figure 2. *Rhododendron luteum*, also known as the “mountain rose” or “yellow poison.”

orders, and hypertension and is also believed to be a sexual stimulant.

Beekeeping is a common activity among the inhabitants of the eastern Black Sea region. Native Caucasian bees (*Apis mellifera caucasica*), which fly in an area of only some 5 km,² are used in the traditional method of honey production. Since rhododendrons are long-lived plants, beekeepers know which honey is “mad.” Honey produced in springtime is more toxic and sometimes contains higher concentrations of grayanotoxins than that produced in other seasons.⁵

Well-known toxic effects of honey poisoning are bradycardia, cardiac arrhythmia, hypotension, nausea, vomiting, sweating, salivation, dizziness, weakness, loss of consciousness, fainting, blurred vision, chills, cyanosis, and convulsions.^{5,6} Poisoning is frequently seen in this region,⁷ and patients generally recover spontaneously without reporting to health institutions. The majority of those who do seek medical care recover with 0.5 to 1.0 mg of atropine and controlled fluid replacement. In rare, life-threatening cases, cardiac arrhythmias requiring cardiac pacing may occur. The potential hazards of these 2 wild flowers need to be appreciated along with their aesthetic qualities.

Abdülkadir Gunduz, MD
Habib Bostan, MD
Suleyman Turedi, MD
İrfan Nuhoğlu, MD
Tevfik Patan, MD
Trabzon, Turkey

References

1. Avcı M. Rhododendrons and their natural occurrences in Turkey. *CografyaDergisi*. 2004;12:1319.
2. Terzioğlu S, Merev N, Anşın R. A study on Turkish Rho-

- dodendron L. (Ericaceae). *Turk J Agric For*. 2001;25:311–317.
3. Onat FY, Yegen BC, Lawrence R, et al. Mad honey poisoning in man and rat. *Rev Environ Health*. 1991;9:3–9.
 4. Baytop T. *Therapy With Medicinal Plants in Turkey, Past and Present*. 2nd ed. Istanbul, Turkey: Nobel Tip Basimevi; 1999.
 5. Dilber E, Kalyoncu M, Yarifi N, et al. A case of mad honey poisoning presenting with convulsion: intoxication instead of alternative therapy. *Turk J Med Sci*. 2002;32:361–362.
 6. Sutlupinar N, Mat A, Satganoglu Y. Poisoning by toxic honey in Turkey. *Arch Toxicol*. 1993;67:148–150.
 7. Yavuz H, Özel A, Akkus I, et al. Honey poisoning in Turkey. *Lancet*. 1991;337:789–790.

High-Altitude Pulmonary Edema Getting Worse After Descent? An Unlikely Event

To the Editor:

We read with great interest the article by Strote and Prutkin¹ because it potentially represents the first reported case of high-altitude pulmonary edema getting worse following descent to sea level. Their claim is based on an assertion that they effectively ruled out pulmonary embolism or other underlying cardiac disease. After reading their case description, however, we take issue with their arguments.

The authors presented the results of an echocardiogram performed at the time of the patient's emergency room presentation. Unfortunately, pulmonary artery pressure measurements were not provided, and the authors do not make it clear as to whether they were simply not measured or could not be measured due to the absence of a tricuspid regurgitant jet. This is an important oversight, because pre-existing pulmonary hypertension could provide a reason why dyspnea might worsen, even with descent to lower elevation; the alveolar hypoxemia induced by the pulmonary edema might cause hypoxic pulmonary vasoconstriction and a further rise in pulmonary artery pressure. We presented a case in which an individual with no history of baseline pulmonary hypertension developed pulmonary edema at 7800 feet in elevation and upon descent to 4900 feet in elevation had a systolic pulmonary artery pressure of 45 to 50 mm Hg.²

More important, we do not think the authors definitively ruled out the possibility of pulmonary embolism as a cause of his worsening dyspnea. Their work-up consisted of a negative D-dimer assay and no further imaging. Published research has shown that among patients with a low clinical probability of pulmonary edema, a normal D-dimer level is associated with a 99% likelihood of not having pulmonary embolism.³ We argue that

this patient did not have a low clinical probability of pulmonary embolism. Upon presenting to the emergency room, he had a hematocrit of 51%, which suggests he may have been volume depleted. In addition, the drive from Mount Rainier to Seattle takes 2.5 to 3 hr, and the patient went to sleep upon arriving home, suggesting that he had a period of immobility during his dehydrated state that might have increased his risk of deep venous thrombosis and pulmonary embolism. Finally, it is not known if this patient has a previously unrecognized predisposition to thromboembolism. This could be significant, because a recent study⁴ indicates that moderate degrees of hypoxia (equivalent to 1800–2100 m in altitude) lead to increased levels of thrombin-antithrombin complex, prothrombin 1+2, and D-dimer in people with underlying risk factors for coagulopathy, such as the Factor V Leiden mutation. Taking into account these clinical factors, this patient warranted additional imaging studies to effectively rule out pulmonary embolism. It is also important to keep in mind that whereas the sensitivity of the D-dimer assays is high for segmental or larger emboli, it falls to roughly 50% for subsegmental emboli.⁴ In the setting of resolving edema, subsegmental emboli would be enough to increase this patient's dyspnea.

Finally, the fact that he was asymptomatic at a follow-up appointment 2 months after his acute episode does not rule out the possibility of a pulmonary embolus. With resolution of the pulmonary edema, it is possible a young, fit person would be asymptomatic with a subsegmental pulmonary embolism. In addition, the body's natural lytic mechanisms would be expected to resolve the thrombus, and, in the absence of an underlying coagulopathy or other predisposing factor for clot formation, he would not be expected to have further episodes of pulmonary embolism.

In the absence of complete data regarding pulmonary artery pressures and further diagnostic imaging, we do not feel the authors can make the claim that this is a case of high-altitude pulmonary edema that worsened with descent to lower elevation.

Andrew M. Luks, MD

Erik R. Swenson, MD

*Pulmonary and Critical Care Medicine
Seattle Veterans Affairs Hospital
Seattle, WA, USA*

References

1. Strote J, Prutkin J. High altitude pulmonary edema presenting 18 hours after descent. *Wilderness Environ Med*. 2006;17:137–139.