CASE REPORT

Compartment Syndrome, Fasciotomy, and Neuropathy After a Rattlesnake Envenomation: Aspects of Monitoring and Diagnosis

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Compartment syndrome resulting from pitviper envenomation is uncommon in North America; however, when it does occur, early diagnosis, optimal antivenom therapy, and possible surgical decompression are the primary means of preventing the complication of neuropathy. Here, we report a case of a rattlesnake envenomation in the anterior compartment of the lower leg that required high doses of morphine to control pain. Although compartment syndrome was considered a possible outcome, subfascial pressures were not monitored and antivenom was discontinued at 24 hours. At 36 hours, the patient developed dorsal foot numbness and foot drop, and 15 hours later pressures within the anterior compartment were >68 mm Hg. Emergency fasciotomy was performed 59 hours postenvenomation. Peroneal neuropathy was evident after surgery and only partially recovered postoperatively. Earlier monitoring of subfascial pressures and using those pressures as a guide for decisions about time and dose of CroFab antivenom treatment may have permitted earlier surgical treatment after onset of compartment syndrome or even prevented the onset of this condition.

Key words: pitviper envenomation, compartment syndrome, fasciotomy, peroneal neuropathy, black-tailed rattlesnake, Crotalus molossus, snake, snakebite

Introduction

Pitviper envenomations with subfascial deposition of venom can result in damage to tissues because of the direct cytotoxic effects of the venom or because of compartment syndrome, the elevation of subfascial pressures within muscle compartments. Compartment syndrome is an unusual complication of pitviper envenomation, and although infrequent, the potential outcomes can be quite serious, ranging from loss of muscle mass and neuropathy to amputation and even death of the patient. Therefore, the diagnosis and treatment of compartment syndrome should be accurate and as early as possible. The single most accurate monitor for compartment syndrome is the measurement of subfascial pressures.3

In some cases, the location and severity of envenomation might heighten the risk of increased pressures within the affected compartments. In those high-risk cases, qualitative monitoring of physical symptoms such as degree of pain, sensation, motor function, and vascular perfusion should be complemented with quantitative direct measures of compartment pressures. These data can then be used to guide decision making in antivenom treatment and, if necessary, timely surgical decompression.

Clinical reports of envenomation by the black-tailed rattlesnake (Crotalus molossus) are uncommon. Here, we describe an additional case of envenomation by a black-tailed rattlesnake in the anterior compartment of the lower extremity that resulted in unusually late onset of increased compartment pressures and the late diagnosis of compartment syndrome. We use this case study to emphasize various aspects of evaluation, monitoring, and treatment.

Case report

A 35-year-old biologist and her graduate student were hiking in the Peloncillo Mountains of southeastern Arizona. At 0911 hours on August 8, 2002, she inadvertently stepped on an unseen adult black-tailed rattlesnake, which struck her on the anterior aspect of her
lower leg 20 cm above the ankle. Two punctures were evident 2 cm lateral to the tibia. The victim, who was wearing shorts and low-cut hiking boots, kept the bitten leg below heart level and was driven without delay to Douglas, AZ, 40 miles away. Upon arrival at 1020 hours, she was experiencing significant discomfort and was administered morphine 10 mg intravenously (IV). Because of discomfort, induration, and location of bite, 4 vials of antivenom (CroFab, Savage Laboratories, Melville, NY) were started at 1120 hours along with normal saline IV. Vital signs and the initial physical examination at time of arrival at the hospital were within normal limits. At 1210 hours, the patient was air lifted to a general hospital in Tucson, AZ, and admitted at 1300 hours to the emergency department 4 hours postbite. Vital signs and coagulation profile remained normal (Table). Morphine 12 mg IV was administered during the next 4 hours to control discomfort. An additional 4 U of CroFab antivenom IV was started at 1650 hours because of continued swelling, and the patient was transferred to the intensive care unit.

Upon admission to the intensive care unit, swelling had progressed to the knee with no local reaction in the area of the 2 fang punctures. Vital signs and laboratory results, including coagulation factors, remained normal (Table). Pain in the lower leg required a patient-controlled analgesia (PCA) morphine pump set to deliver a maximum of 20 mg IV q4h. Progression of edema was monitored with extremity circumference measurements, and an arterial line was established for frequent blood draws. Hospital medication records indicate that additional CroFab antivenom was administered (2 vials IV q6h at 15 hours and 21 hours postbite) for a total of 12 vials during the first 24 hours postbite.

The resident physician consulted the Arizona Poison Center and was advised that because of the size of snake, location of bite, and lack of protective garment, compartment syndrome should be considered a high potential risk and that subfascial pressures should be monitored in addition to physical examination. A vascular surgeon was consulted at 9 and 24 hours postbite, and his examination indicated peroneal nerve function was intact and no indication of compartment syndrome. The patient was instructed to be aware of decreased sensation to the dorsal surface of the foot and lack of control in movement of the affected ankle. However, compartment pressures were not measured or monitored. Although mild ecchymosis appeared subcutaneously on the dorsum of the foot, the skin and subcutaneous tissue at the 2 puncture sites remained normal.

Because the patient was considered stable, laboratory values were within normal limits (Table), and no further antivenom administration was planned, the patient was moved to a general medical ward 38 hours postbite. At that time, the progression of swelling had halted at the inguinal region. The patient reported to the nurse that the dorsum of her left foot felt numb. She was told that this was likely because of prolonged elevation of the limb during the observation period in the intensive care unit. At 47 hours postbite, the patient was examined by the medical resident and attending internist, who did not believe that the examination was abnormal. She continued to require PCA morphine for discomfort in the bitten leg. Two hours later (25 hours after the last documented dose of CroFab), a visitor called the attending physician to report that the patient could not dorsiflex her foot. The vascular surgeon was consulted again at 54 hours postbite, and compartment pressures were measured for the first time with a pressure-monitoring device (Pressure Sense, Millennium Medical Technologies Inc, Santa Fe, NM). The anterior compartment pressures at 3 different points were >68 mm Hg (the maximum level recordable on the device) and 25 mm Hg in the left lateral compartment. Two hours later, the patient underwent an anterior fasciotomy with compartment decompression; surgery occurred at 56 hours postbite, 18 hours after the patient’s initial complaint of numbness and 7 hours after the communication of loss of movement of the left foot. At the time of fasciotomy, the physician reported substantial amounts of potentially necrotic muscle. Postop-

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### Table. Hematologic and coagulation studies during initial 3 days of hospitalization*

<table>
<thead>
<tr>
<th>Time postbite (h)</th>
<th>1.5</th>
<th>4.2</th>
<th>8.5</th>
<th>13.0</th>
<th>16.3</th>
<th>21.2</th>
<th>29.3</th>
<th>41.8</th>
<th>63.8</th>
<th>70.7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (12—16 g·dL⁻¹)</td>
<td>NA</td>
<td>13.3</td>
<td>13.6</td>
<td>13.9</td>
<td>14.0</td>
<td>13.5</td>
<td>NA</td>
<td>13.5</td>
<td>12.2</td>
<td>NA</td>
</tr>
<tr>
<td>Platelets (130—450 × 10⁶·L⁻¹)</td>
<td>229</td>
<td>208</td>
<td>248</td>
<td>248</td>
<td>235</td>
<td>230</td>
<td>215</td>
<td>189</td>
<td>189</td>
<td>NA</td>
</tr>
<tr>
<td>Prothrombin time (11.0—12.9 s)</td>
<td>12.6</td>
<td>12.6</td>
<td>12.1</td>
<td>12.4</td>
<td>12.7</td>
<td>12.8</td>
<td>13.2</td>
<td>12.5</td>
<td>NA</td>
<td>11.7</td>
</tr>
<tr>
<td>Fibrinogen (150—400 mg·dL⁻¹)</td>
<td>343</td>
<td>316</td>
<td>325</td>
<td>359</td>
<td>371</td>
<td>359</td>
<td>390</td>
<td>515</td>
<td>NA</td>
<td>779</td>
</tr>
</tbody>
</table>

*NA indicates tests were not available for the specific time period.
eratively, there was no recovery of the motor or sensory function of the peroneal nerve, and the patient was not capable of dorsiflexing her left foot. The wound was explored 5 days later and the central two thirds of the anterior compartment muscles was nonviable (as recorded by the surgeon). These were debrided and the wound was closed. Two days later, the wound exhibited signs of infection. The middle third of the fasciotomy site was reopened, the wound was cultured positive for *Bacillus and Enterococcus*, and the patient began a 10-day course of ampicillin (1.5 g IV q6h). The wound was not closed, and it healed by secondary intention. The patient was discharged 12 days after admission and was prescribed wound care and physical therapy for foot drop.

One month postenvenomation, 2 abscesses developed in the vicinity of the bite site; these were opened and debrided as outpatient procedures. With continued physical therapy, foot drop gradually improved during the next 14 months with hypertrophy of the remaining anterior tibialis muscle. At 18 months, adhesions within the compartment limited further muscle development and required surgery to improve range of motion. At 24 months, the patient had recovered dorsiflexion to neutral position, sufficient to permit long-distance running and marathon foot races.

**Discussion**

The pathophysiology of compartment syndrome after trauma results primarily from the increased pressure within the affected compartment. In contrast, cases of venom-induced compartment syndrome include both direct venom effects on tissues as well as decreased perfusion pressures. Elevated pressures with reduced tissue perfusion could potentially exacerbate damage. Subfascial injection resulting in compartment syndrome is unusual for envenomation by pitvipers in North America. In Arizona, where most envenomations are by rattlesnakes, 2 separate studies report that 6 of 164 (4%) and 3 of 236 (1%) cases resulted in fasciotomy for compartment syndrome. The outcome was not reported in the first study; in the second study, all 3 patients did well, though pressure measurements were not performed. However, if not diagnosed and treated early after onset, compartment syndrome may result in nerve damage.

Early clinical symptoms and signs of a pitviper bite may not be sufficient for early diagnosis of compartment syndrome if intracompartamental pressures are not measured. Additionally, pressure monitoring can help eliminate unnecessary fasciotomies. This case is an example of how clinical diagnosis and subsequent treatment can proceed suboptimally if compartment pressures are not measured directly. The initial response in this case was appropriate. The patient did not use specific first aid measures; rather, she remained calm and was transported expeditiously to a hospital, following the guidelines recommended for rattlesnake bites. Antivenom was administered early, the poison center was consulted, and air evacuation happened in a timely manner.

At the initial assessment of the patient, important diagnostic aspects that indicated increased risk of compartment syndrome were 1) the bite location on bare skin over the anterior compartment of the lower leg; 2) the proximity of the skin to the underlying fascia in that area; 3) the lack of tissue reaction near the puncture sites, suggesting a deep penetration of the venom; and 4) the species identity. The mean fang length of adult black-tailed rattlesnakes is 11.7 mm, which increases their capacity to inject venom deeply into the tissue. After initial treatment, the use of high IV doses of morphine to control discomfort in the absence of significant local tissue reaction should also have suggested subfascial injection of venom (Leslie Boyer, Arizona Poison Center, Tuscon, AZ, oral communication, October 2002). These characteristics should have been used to categorize this particular envenomation as severe rather than moderate because of the likelihood of subfascial venom injection, prompting more aggressive antivenom therapy and direct measurement of compartment pressures.

The patient had vascular surgery consultation visits during the first 24 hours, but compartment syndrome did not appear to be present because nerve and vascular function was normal. Nevertheless, all aspects mentioned above should have prompted the use of repeated measurement of subfascial pressures over time, which may have led to more aggressive antivenom therapy and the consideration of earlier surgical decompression. If pressure levels had been marginal (25–30 mm Hg) or even elevated (>30 mm Hg), antivenom treatment could have been resumed and pressures measured again. If pressures had remained elevated, immediate fasciotomy would have then been considered. Clinical and laboratory studies indicate that elevated subfascial pressures after pitviper envenomations can be reduced and even controlled when treated with IV antivenom. Higher doses of antivenom might also have been considered according to the CroFab package insert, which says that the neutralizing capacity for this species of pitviper is lower than for those species whose venoms were used for the antivenom production. However, a recent report indicates that antivenom effectiveness may be less species dependent than previously thought.

The onset of compartment syndrome in this case was unusually delayed, possibly because of early neutralization of venom by IV antivenom. CroFab is excreted
Snakebite and Compartment Syndrome

by the kidneys and has a relatively short half-life; therefore, maintenance doses are usually administered every 6 hours after the initial control of signs and symptoms of envenomation. Because antivenom use was discontinued at 24 hours, venom that had not been neutralized could have induced an increase in subfascial pressure. Given the characteristics of this bite, which met many of the high-risk factors discussed previously, it is not certain that continued antivenom treatment would have reduced tissue damage or effectively controlled compartment syndrome. However, at the very least, if pressure increases had been recognized earlier, this would have allowed for earlier surgical intervention. Surgical decompression of compartment syndrome is most effective and results in fewer complications if it occurs within a ‘golden period’ of 8 to 12 hours post-onset. Delay of diagnosis and treatment in this case likely contributed to the neuropathy and muscle necrosis sustained by the patient.

A recent study in a porcine model found that fasciotomy performed at the time of envenomation resulted in more myonecrosis from the venom than did non-intervention. An editorial summarizes how these findings may effect the future use of fasciotomy in crotaline envenomation. Unfortunately, fasciotomy to prevent neuropathy, an equally severe consequence of increased subfascial pressure, was not discussed in these recent reports. The costs and benefits of surgical decompression remain debatable; nonetheless, measures of compartment pressure can be used to guide decision making in antivenom treatment and, if necessary, timely surgical decompression.

In summary, the location and severity of envenomation can heighten the risk of increased pressures within the affected compartments. In high-risk cases, qualitative monitoring of physical symptoms such as degree of pain, sensation, motor function, and vascular perfusion should be complemented with quantitative measures of compartment pressures.

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