Case Report

Rattlesnake Envenomation in Three Dairy Goats

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1. Introduction

To the author’s knowledge this is the first reported case series of envenomation of dairy goats by the Northern Pacific Rattlesnake (Crotalus oreganus). Cases, treatment, and outcomes of rattlesnake envenomation have been reported for horses [1], llamas, and alpacas [2, 3] but veterinary literature is lacking in cases of rattlesnake envenomation in dairy goats. The following 3 cases, referred to the University of California-Davis William R. Pritchard Veterinary Medical Teaching Hospital (VMTH), between the years of 1994 and 2014, represent rattlesnake envenomation, where the rattlesnake was identified as the Northern Pacific Rattlesnake (Crotalus oreganus).

2. Case Selection

A retrospective search was done through the William R. Pritchard Veterinary Medical Teaching Hospital’s record database (Veterinary Medical & Administrative Computer System, VMACs) for goat cases that were definitively diagnosed as rattlesnake envenomation. Search functions included a designation of caprine patients and then the term: “rattlesnake,” “bite,” “snakebite,” “envenomation,” and “snake.” Records were then checked for the appearance of a snakebite, and then either a witnessed bite or a witnessed account, by the owner, of a rattlesnake in close proximity to the snake near the time of envenomation. Cases that lacked a clear bite wound or some form of an eyewitness account of a rattlesnake were excluded.

3. Cases

Three cases fit the search criteria; all involved dairy goat breeds.

3.1. Case 1. A 3-year-old, female, open Toggenburg show doe presented to the VMTH after she was bitten on the left front leg shortly after the evening milking. This bite was witnessed by the owner and the snake was killed. The owner administered procaine penicillin (PPG) and flunixin meglumine. The affected limb was iced and the patient presented to the VMTH fewer than 2 hours after the bite occurred.

At presentation the doe was bright, alert, and responsive with a rectal temperature of 102.4°F (39.1°C), a slight tachycardia (96 beats/min; normal: 70–90) with a normal rhythm, and a mild tachypnea (56 breaths/min; normal: 15–30); mucus membranes were pink and moist. The left front
leg was swollen from the distal aspect to the axillary region, with pitting edema observed. A bite was noted on the dorsal surface of the fetlock. The owner noted the initial swelling had decreased after cooling the limb with ice. The remainder of the physical examination was within normal limits.

The doe was initially treated at the hospital with a maintenance rate of Lactated Ringer’s Solution (2.3 mL/kg/hour), amoxicillin (10 mg/kg IM, q 12 hours), methylprednisolone (10 mg, IM, once), and flunixin meglumine (1.1 mg/kg SQ, q 12 hours). She was also treated with 10 mL of an equine origin antivenin IV (Crotalid). Hydrotherapy was utilized on the limb every 12 hours for 15 minutes.

On the second day of hospitalization the patient had normal temperature, pulse, and respiration values, and the leg was less swollen. Amoxicillin, flunixin, LRS, and hydrotherapy were continued. The patient was walked several times during the day to improve lymphatic drainage with the hope of decreasing the swelling of the leg. Normal appetite, urination, and defecation were noted, and the doe produced 50% as much milk as she did the day before the bite.

On the third day the patient showed continued improvement with reduced swelling on the proximal portion of the leg, but with edema still present on the fetlock. Milk production had also increased 0.45 kilograms from the previous day. Exercise and hydrotherapy were continued, and the patient was switched from amoxicillin to ticarcillin and clavulanate due to limitations of pharmacy stock. Flunixin meglumine was discontinued at this time.

On the fourth day of hospitalization, the patient was discharged with instructions to administer sodium ceftiofur (2 mg/kg IM, q 12 hours) and PPG (22,000 IU/kg SQ, q 12 hours) for the next 8 days. Hydrotherapy was encouraged and the owner was instructed to observe the leg twice daily. The doe recovered uneventfully with no apparent complications after discharge. She went on to have a productive show career.

3.2. Case 2. A 10-month-old Alpine Doe was found recumbent by her owner with a swollen right front limb. Two puncture wounds were observed below the elbow, and severe swelling was noted on the right limb and ventral neck. She was administered procaine penicillin G (22,000 units/kg, IM), and flunixin meglumine (1.1 mg/kg, SC) by her owner and transported to the VMTH within two hours of discovery in the pasture.

On physical exam the doe was bright, alert, and responsive, with normal hydration and a weight of 50 kg. Her heart rate was within normal reference intervals, but she was tachypneic (50 breaths/minute), and her mucus membranes were pale with an increased capillary refill time (3 seconds). She was normothermic (39.3°C). Swelling was noted in the ventral neck area. The right forelimb was swollen, purple, and warm to the touch. Necrosis and sloughing of the skin were noted on the lateral aspect of the right forelimb. The swelling extended from carpus to shoulder and the patient was nonambulatory on the limb. Two puncture wounds were present below the elbow of this limb. The patient had normal pupillary light reflexes, menace responses, and no evidence of scleral injection. No stertor was observed, although frothing saliva was noted. Normal rumen contractions and normal urinations were also noted during examination.

Upon arrival, a point-of-care chemistry analysis (iStat) revealed hyperglycemia (253 mg/dL; normal: 50–75), hypokalemia (2.7 mEq/L; normal: 3.5–6.7), increased bicarbonate (37 mEq/L; normal 24–28), hypocalcemia (0.81 mg/dL, ionized Ca; normal >1.0), and hyponatremia (2.4 g/dL; normal: 2.7–3.9). A urinalysis also revealed proteinuria (100–300; normal: negative to trace) and glucosuria (2000+; normal: negative). Packed cell volume was within normal ranges at 33% (normal 22–38) and total protein was decreased at 5.3 g/dL (normal 6.4–7.0). Radiographs revealed soft tissue swelling at the level of the radius and ulna with no evidence of fractures. A complete blood count (CBC) displayed a pronounced neutrophilia (13,341/μL; normal: 1,200–7,200) and thrombocytopenia (146,000/μL, normal: 300,000–600,000).

Treatment was initiated with the placement of an IV catheter and a 3 mL/kg bolus of polyionic fluids (Plasmalyte 148), followed by a maintenance rate of IV fluid therapy [2.3 mL/kg/hr; Plasmalyte 148 with 22 mL/L of calcium, phosphorous, magnesium, potassium solution (CMPK), and 40 mL/L of potassium chloride (KCl) added]. The patient was also treated with a bolus of 2 liters of caprine plasma. Overnight the patient developed an acidosis (pH 7.172) and became obtunded. A repeat CBC showed a significant reduction in platelets (72,000/μL). The patient died the next day.

A necropsy was performed. The subcutaneous tissues and connective tissue planes between muscles along the ventral abdomen and left front leg were expanded by edema and hemorrhage. The muscles of the left front leg were dark red and edematous; these changes were more pronounced from the axillary to the carpal region. Aerobic and anaerobic cultures from the puncture wounds yielded no growth. Mild hemorrhage within the left ventricular free wall and diffuse petechiation of the omentum were noted. The lamina propria of the jejunum was infiltrated by inflammatory cells consisting mainly of lymphocytes, eosinophils, and lesser amounts of plasma cells. Tubular degeneration and glomerulonephritis were noted in the kidneys. Diffusely throughout the liver, hepatocytes were swollen and noted to contain one small, well-defined clear vacuole consistent with lipid accumulation. In sections of lymph node there was expansion of the cortex consistent with reactive follicular and parafollicular hyperplasia. The bone marrow was hyperplastic and was most likely a result of increased tissue demand for inflammatory cells.

The lungs were pink with multiple, dark red, ill-defined, atelectatic foci; all sections of lung floated in formalin. An infection with *Dictyocaulus filaria* was diagnosed based on the presence of inflammatory cell infiltrate within and around the bronchioles, profiles of parasites within the bronchiolar lumens, and thickening of the alveolar septa. The thickening of the alveolar septa and accumulation of blood and fibrin from the lungworm infection most likely led to impaired diffusion of oxygen and subsequent respiratory distress and contributed to the death of this animal along with envenomation.
3.3. Case 3. A 5-month-old La Mancha doe (18 kg) presented severe facial swelling of approximately 24-hour duration. The owners had noticed a rattlesnake in immediate proximity to the goat the day before presentation.

At presentation the doe was quiet, alert, and responsive with a rectal temperature of 103.5°F (39.7°C), and a heart rate of 150 beats/min (normal: 70–90), with a respiratory rate of 56 breaths/min (normal: 15–30). The doe's mucus membranes were not examined due to moderate to severe facial swelling and pain, but vulvar mucus membrane capillary refill time was noted to be 1–2 seconds (within normal limits). Two bites were observed, one on the right nostril and one on the nasal planum. Examination of the face was difficult due to severe bilateral swelling noted from the lips to the poll, and to the base of the neck. Some increased respiratory effort was noted on the initial examination. A moderate amount of edema was noted in the ventral neck region.

The doe was initially treated with dexamethasone sodium phosphate (1 mg/kg IV, once), flunixin meglumine (1 mg/kg IV, q 12 hours), sodium ceftiofur (1 mg/kg IM, q 12 hours), butorphanol (0.02 mg/kg IM, once), and given a subcutaneous (SC) bolus of 300 mL Normosol-R with KCl added (total K: 20 mEq/L). She was also vaccinated with a CDT toxoid (SC) and tetanus antitoxin (1500 units IM) was administered.

On the second day the doe's swelling had slightly reduced, and her activity continued to be reduced as she was observed in sternal recumbency for the majority of the day and observed standing only in the afternoon. She had a mild appetite when offered food. Therapy with sodium ceftiofur, flunixin meglumine, and 300 mL bolus fluids (q 4 hours, SC) was continued. PPG (22,000 IU/kg, IM q 12 hours) and morphine (0.25 mg/kg, IM q 12 hours) were added to the therapeutic regimen.

On the third day more improvement was noted in behavior, although the swelling of the ventral neck persisted. A CBC and chemistry revealed normal CBC parameters in addition to hyperglycemia (98 mg/dL; normal: 50–75), hypoproteinemina (4.3 g/dL; normal: 6.4–7.0), hypokalemia (2.9 mEq/L; normal: 3.5–6.7), and hyponatremia (133 mEq/L; normal: 142–155 mEq/L). Furosemide (1 mg/kg, IV, once) was added to the treatment regimen and SC fluids were discontinued.

On the fourth day the doe was noted to be more physically active and playful. Her swelling had significantly decreased around the head and ventral neck. Her appetite appeared normal, but it was noted that in chewing she had a moderate amount of saliva oozing over her mandible. She had diminished facial nerve motor deficits on the left in the mandibular, auricular, and eyelid muscles. No strabismus or nystagmus was noted OU. The doe was discharged with instructions for continuing sodium ceftiofur (5 mg/kg, IM q 12 hours, for 3–5 additional days), and ophthalmic ointment (q 8 hours, until normal tear production was noted).

4. Discussion

While multiple studies outline the etiology and treatment of rattlesnake envenomation in llamas, alpacas [2, 3], horses [1], small animals, and humans [4], to the author's knowledge, there are no studies or reports in current literature of this disease in dairy goats. Rattlesnakes are a common venomous snake species in North America and therefore represent a potential cause for veterinary emergencies. In the northern region of California, rattlesnake envenomation has been reported to have a 9% and 58% mortality rate in horses and new world cameldids (NWC) [1, 2]. In this region of California the Northern Pacific Rattlesnake (Crotalus oreganus) is the most common rattlesnake species [1], and envenomation typically occurs seasonally, associated with the hibernation of rattlesnakes in the winter. All cases reported here occurred in June, within the rattlesnake season of May through August reported for NWC [2] and the rattlesnake season of March through October reported for horses [1] in the same region as these cases. Two bites occurred in lower limbs, and one occurred in the face. The most common sites of rattlesnake envenomation in NWC and horses have been reported as primarily the face and secondarily the legs [1–3]. Delays in treatment and increased mortality have been noted in horses [1], and it is noteworthy that the two goats in this case report that survived were promptly treated within hours, and the one that died experienced a several-day delay between the bite and treatment, which may be compounded by the concurrent lungworm infestation.

The clinical signs observed in horses and NWCs were noted in these goat cases. These patients displayed respiratory distress, fever, tachypnea, tachycardia [3], hyperthermia, and recumbency [2] as reported in NWC and horses [1]. Facial nerve paralysis has also been reported in horses with rattlesnake envenomation [1] and was noted in one of these cases. Cardiac arrhythmias have been reported in horses following envenomation, including atrial premature contractions, sinus pause, sinus tachycardia, sinus arrest, ventricular premature contractions, sinus arrhythmia, atrial fibrillation [10], as well as third-degree AV Block, among other arrhythmias [5]. No arrhythmias were noted in any of the goats, although no ECGs were performed.

In humans, the prophylactic treatment of rattlesnake bites with antibiotics is controversial with recommendations for [6] and against [7] this practice. A study of 15 rattlesnake venom cultures yielded 58 aerobic and 28 anaerobic bacteria, most commonly Pseudomonas aeruginosa, Proteus, coagulase negative staph, Clostridium, and Bacteroides fragilis [8]. Tissue edema and vascular compromise are common in rattlesnake envenomation and this environment predisposes a patient to infection. All 3 goats in this report were treated with antibiotics. However no uniform treatment was initiated due to different clinicians at time of admission. In 58 cases of rattlesnake envenomation of horses in California, almost all animals received antibiotics [1]. In a study of 12 NWC, all 10 that were alive on presentation were treated with procaine penicillin [2]. In the aforementioned Alpine doe case, culture of the wound at necropsy revealed no growth. This result could have been compromised by the antimicrobial therapy the doe received after envenomation, or could be because no bacteria was transmitted during the bite.

In all cases, anti-inflammatory therapy was used in the forms of flunixin meglumine, dexamethasone, dexamethasone sodium phosphate, or methyl prednisolone. Flunixin
meglumine is a commonly used anti-inflammatory in both NWC [2, 3] and equine [1] envenomation. Corticosteroid treatment for snake envenomation has been linked to increased mortality in dogs [9], but in a recent NWC study, 10 of 27 rattlesnake envenomation cases were treated with corticosteroids (9 dexamethasone and 1 prednisolone) [3]. This study reported that treatment in NWC with either NSAIDs or corticosteroids was not associated with outcome. In a NWC study in the same geographical region as the 3 goat cases, 4 out of 5 animals that survived and 3 out of 7 that died were administered corticosteroids [2]. While controversial in its clinical application, dexamethasone may have a place in the treatment of rattlesnake envenomation as dexamethasone has been shown to inhibit Tumor Necrosis Factor- (TNF-) alpha synthesis [10]. Decreasing TNF-alpha could be beneficial in the management of systemic disease caused by rattlesnake envenomation in dairy goats.

All of these cases received some form of fluid therapy for medical management. Fluid therapies utilized were polyionic fluids and caprine plasma. Polyionic fluids utilized included Lactated Ringer's Solution, Normosol-R, and Plasmalyte 148. Potassium chloride and dextrose were utilized with fluid therapy. Two of the cases utilized furosemide in conjunction with fluid therapy.

Antivenin is another aspect of therapy for rattlesnake envenomation. Out of 58 snake-bitten horses, 9 received antivenin, and none of these animals died [1]. While that study was not designed to evaluate treatment regimens, the authors suggest that early administration of antivenin in the timeframe of envenomation could be beneficial for positive outcome in rattlesnake bites. Antivenin administration has also been associated with reduced morbidity in dogs and cats after snake envenomation [11]. This finding may be supported by the Toggenburg case (case 1). In the same area as the 3 cases in this study, as well as one NWC [2] and an equine [1] study, human snakebites were noted to have a 0% case fatality rate, but antivenin was much more heavily utilized, with some patients receiving as much as 12 vials [4].

In horses a rattlesnake-bite severity scoring (RBSS) system has been suggested [1]. This 12-point system scored patients in with 4 different variables (respiratory system, cardiovascular system, wound appearance, and hemostasis) with 0 being a normal presentation and 3 being a clinically severe presentation. It has been suggested that horses with a RBSS of 8 or higher should be watched especially carefully, as more aggressive treatment may be indicated for a patient with this score [1]. Enough clinical information is present to retrospectively apply this scoring system to the Alpine and the La Mancha cases. The Alpine case would have had a score of 8 with a score of 3 for the respiratory system (signs of respiratory distress), 0 for the cardiovascular system (no abnormalities), 3 for wound appearance (severe swelling spreading to the trunk), and a hemostasis score of 2 (platelets 72,000 μL on the second day). The La Mancha would have had a score of 6 with a score of 1 for the respiratory system (mild signs of respiratory distress evidenced by an increased respiratory effort), 2 for the cardiovascular system (a moderate tachycardia), 3 for wound appearance (severe swelling spreading to the neck), and a hemostasis score of 0 (no abnormalities). Using the previously recorded criteria, the Alpine case would have required more attention or more aggressive therapy. While more evaluations are needed, the outcome of the 2 cases (Case 2: score 8, mortality; Case 3: score 6, survival) suggests that the RBSS system may potentially be applicable to dairy goats.

Limitations of this report include its retrospective nature. Multiple clinicians and students were involved over an extended time frame. As such, cases were not worked up in an identical manner. While one of the bites was directly witnessed, 2 were not, and as such diagnosis of envenomation was based on proximity to a snake on discovery, recent observation of snakes on the premises, and appearance of bite wounds. The small sample size is also a limitation with this study. Long term follow-up (beyond 1 year) was only available for one goat in this study.

5. Conclusions

In conclusion, we report a case series of rattlesnake envenomation in dairy goats, which provides evidence of similar pathophysiology and treatment for rattlesnake envenomation in dairy goats as described for other large animal species. Faster implementation of treatment with respect to the timing of the bite may lead to a more positive treatment response. Similarly, the pathologic changes from coinfection with lungworms, such as *Dictyocaulus filaria*, may predispose a goat to a lesser prognosis in the case of rattlesnake envenomation. While this study was limited to three cases, more research is needed with respect to envenomation from different species of rattlesnake and therapy utilized to manage a snakebite, as well as the application of a bite scoring system to goats. These case descriptions provide basic information about rattlesnake envenomation that may be useful in managing cases of rattlesnake envenomation in dairy goats.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

References


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