

HEMOLYTIC ANEMIA IN A PRONGHORN (*Antilocapra americana*)

Edward J. Gentz, MS, DVM*

Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853, USA

Lisa A. Harrenstien, DVM

Department of Pathobiology, College of Veterinary Medicine, University of Tennessee, Knoxville, TN 37901, USA

Jeff Baier, DVM

Denver Zoological Gardens, City Park, Denver, CO 80205, USA

James W. Carpenter, MS, DVM, Dipl ACZM

Exotic Animal, Wildlife, and Zoo Animal Medicine Service, Department of Clinical Sciences, College of Veterinary Medicine, Kansas State University, Manhattan, KS 66502, USA

Introduction

Hemolytic anemia is characterized by an accelerated rate of erythrocyte destruction. The disease processes and pathophysiologic mechanisms associated with hemolytic anemia vary considerably. Likewise, the clinical manifestations of hemolytic anemia may vary according to the degree of anemia, rate of erythrocyte destruction, and causative disease or disease agent. This case report documents the successful treatment of what may be the first reported case of hemolytic anemia in a pronghorn.

Presentation

A four-year old, 34 kg, female pronghorn (*Antilocapra americana*) was hospitalized after she was noted to be recumbent in the exhibit she shared with another pronghorn, two white-tailed deer, and two wild turkeys. Physical examination revealed lethargy, depression, moderate dehydration, grade II systolic murmur, prolonged capillary refill time, red-colored urine, and marked icterus of the sclera, gingiva, and vulvar mucous membranes. Hematology revealed a significant anemia (PCV = 22%, normal = 38.3-50.5). Blood serum chemistry analysis revealed hyponatremia (136 mmol/L, normal = 155), hypochloremia (97 mmol/L, normal = 111), hypokalemia (3.9 mmol/L, normal = 5.5), hypocalcemia (7.9 mg/dl, normal = 9.6), and azotemia (urea nitrogen 127 mg/dl, normal = 29-45). Venous blood gas analysis showed a metabolic acidosis (pH 7.33 with decreased pCO₂ @ 34.4 mm Hg and decreased HCO₃ @ 18.2 mmol/L).

A catheter was placed in the right jugular vein and fluids started at a rate of 120 ml/hr (1.5x maintenance). After an initial liter of 5% dextrose, these were changed to lactated Ringer's solution. After 24 hr of fluid therapy, the serum sodium and chloride returned to normal range. However, the serum calcium (6.7 mmol/L) and potassium (3.1 mmol/L) worsened, due to hemodilution. The PCV also decreased to 18% after 24 hr. Subsequently, the fluid rate was decreased to maintenance and 20 meq/L of potassium chloride (KCl) were added daily to the fluid therapy until the serum potassium returned to normal range. Additionally, 25 ml of 23% calcium gluconate were added to the fluid therapy, after which the serum calcium also returned to normal range. Urinalysis of the red-colored urine at the time of presentation revealed 2+ bilirubin, 3+ protein, and 3+ occult blood, but no erythrocytes, indicating the problem was hemoglobinuria, or less likely, myoglobinuria, as the muscles seemed to be normal, rather than hematuria. After 24 hr of fluid therapy, the urine color was a hazy yellow, however, repeat urinalysis still revealed 2+ protein and 2+ occult blood. The initial venous blood gas abnormalities were normalized on subsequent blood gas analysis (pH 7.37, pCO₂ 44.9 mm Hg, HCO₃ 25.6 mmol/L).

The combination of presenting signs of anemia, icterus, and hemoglobinuria were indicative of hemolytic anemia.

Differential Diagnoses

Initial differential diagnoses for the hemolytic hematuria in this pronghorn included intraerythrocytic parasitism (anaplasmosis, babesiosis, eperythrozoonosis, or hemobartonellosis), bacterial infection (leptospirosis, or bacillary hemoglobinuria caused by *Clostridium hemolyticum*), autoimmune hemolytic anemia, or the ingestion of toxic plants causing Heinz body hemolytic anemia (wild onion or red maple leaf poisoning). Other differential diagnoses considered less likely included water intoxication (salt poisoning) seen in calves and copper toxicosis seen in lambs. No potential source of copper toxicity was apparent, and excessive water consumption had not been noted.

Diagnostics

The presence of hemoglobinuria ruled out anaplasmosis as a differential diagnosis, as this disease is characterized by yellow urine and an absence of either hematuria or hemoglobinuria.¹ No other red blood cell parasites were seen on repeated blood smear examinations, ruling out babesiosis, eperythrozoonosis, and hemobartonellosis. Paired leptospirosis titers, utilizing banked serum from the previous year's annual physical exam and stored in an ultra-cold freezer, were negative. Phase contrast microscopy also failed to reveal any leptospire in the urine. Bacillary hemoglobinuria caused by *Clostridium hemolyticum* was considered unlikely, because the pronghorn was currently vaccinated with a multivalent clostridial vaccine, including tetanus. A Coombs' test for autoimmune hemolytic anemia, using bovine reagent, was negative at dilutions 1:2 through 1:128. Additionally, specific examination of repeated blood smears for the presence of spherocytes was negative. Careful inspection of the pronghorn's exhibit found no wild onions, red maple leaves, or other toxic plants capable of causing Heinz body hemolytic anemia.

Therapeutics

In the absence of a definitive diagnosis, it was decided to initiate a broad-spectrum treatment regime. Oxytetracycline (LA 200) was initiated at a dose of 20 mg/kg SQ q48hr to treat for suspected red blood cell parasitism as well as leptospirosis. Additionally, procaine penicillin G was administered (22,000 IU/kg IM BID), as this would be the treatment of choice for *Clostridium hemolyticum*, although generally penicillin and tetracycline are not administered simultaneously. Concurrently, the pronghorn received dexamethasone (12 mg q24hr IV) for suspected autoimmune disease. Intravenous fluid therapy was continued both for general supportive therapy and specifically for possible plant toxicity.

Despite these treatments, the pronghorn's condition continued to deteriorate. When, after 5 days, the PCV reached a nadir of 11%, it was decided to attempt a blood transfusion utilizing her unaffected sibling from the same exhibit. A cross-match between donor and recipient revealed neither major nor minor agglutination. The donor pronghorn was sedated at the zoo with 0.1 mg/kg detomidine and 1.5 units (750 ml) whole blood removed for transfusion. Post-transfusion, the anemic pronghorn's PCV was 21% and she appeared clinically improved. The PCV remained stable for two days, when a CBC revealed a WBC of 26,600, with 24,000 neutrophils. The azotemia resolved at this time (urea nitrogen = 33 mg/dl), indicating hemolysis had ceased. Urinalysis at this time showed no blood or bilirubin in the urine, and only trace protein. Intravenous fluids were discontinued, the dexamethasone was slowly tapered off, the penicillin continued, and the oxytetracycline replaced with 1.1 mg/kg ceftiofur (Naxcel) IM q24hr. Over the next 10 days, the CBC returned to normal and the PCV slowly rose to 31 %, when the pronghorn was returned to exhibit at the zoo. The systolic murmur, suspected to be due to hypovolemia, was no longer present at the time of discharge from the hospital.

Discussion

Hemolytic anemia has, apparently, not been previously reported in pronghorn (E. S. Williams, pers. comm. 1994). Blood samples from hundreds of pronghorns from Texas, Wyoming, and Oregon analyzed for leptospirosis and anaplasmosis were negative.² Anemia in pronghorn fawns secondary to copper deficiency was seen at the Los Angeles Zoo (J. Boehm, pers. comm., 1995).

Wild ruminants such as deer, elk, and bison rarely have clinical anaplasmosis but can be asymptomatic carriers; in California, native deer are considered to be a major source of infection for cattle.¹ Wild ruminants known to be susceptible to bovine babesiosis include white-tailed deer, bison, reindeer, water buffalo, and African buffalo.³ Two sable antelope imported from a zoo in West Germany to South Africa died of babesiosis after displaying hemoglobinuria and anemia.⁴ Bacillary hemoglobinuria caused by *Clostridium hemolyticum* may affect wild ruminants.⁵ Leptospirosis has been reported in white-tailed deer, but may be self-limiting in exotic ruminants.⁶ Hemobartonellosis (eperythrozoonosis), caused by *Eperythrozoon ovis*, has been reported in white-tailed deer, mule deer, elk, eland, and blesbok.⁶ Hemolytic anemia in the black rhinoceros is currently under intense investigation; some, but not all, cases are linked to leptospiral infection. Additionally, decreased serum vitamin E and low ATP levels in black rhino erythrocytes may decrease red blood cell membrane stability and predispose them to lysis.⁷

None of the above diseases was able to be documented in the pronghorn of this report. Additionally, a Coombs' test for autoimmune hemolytic anemia was negative. However, the Coombs' test is not always positive in affected animals. The Coombs' test is also based on species-specific reagents; the bovine reagent used may not be appropriate for use in pronghorn. The Coombs' test was also performed following several corticosteroid treatments, which may inhibit antibody production and lead to a false negative result.⁸ It is also possible that despite a recent vaccination, the hemolytic anemia of this pronghorn was caused by *Clostridium hemolyticum*. A blood culture may have been useful in determining if this was the case. Serum vitamin E and copper levels also may have been useful.

Conclusions

Hemolytic anemia can occur in the pronghorn. In the absence of a definitive diagnosis, the judicious use of broad-spectrum therapeutics, supportive care, and conspecific blood transfusions can contribute to successful case management.

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