

AN UNUSUAL CASE OF ULCERATIVE STOMATITIS AND PROLONGED PREGNANCY IN A BLACK RHINOCEROS

Don Gillespie, DVM*

Cincinnati Zoo, 3400 Vine Street, Cincinnati, Ohio 45220, USA

Michael Burton, VMD

Cheyenne Mountain Zoological Park, 4250 Cheyenne MT Zoo Road, Colorado Springs, Colorado 80906, USA

Sylvie Gosselin, DMV, PhD, Earl Pope, PhD, and Bob Godfrey, PhD

Center for Reproduction of Endangered Wildlife, Cincinnati Zoo, Cincinnati, Ohio 45200, USA

Linda Munson, DVM, PhD

Department of Pathology, National Zoological Park, Washington, D.C. 20008, USA

In late 1988 an 18-yr old black rhinoceros (*Diceros bicornis*) with an 8-yr history of ulcerative stomatitis developed expiratory dyspnea, colic, and generalized weakness. The ulcerative stomatitis was characterized by raised, bleeding ulcers 4-5 cm in diameter which would regress under daily treatment with prednisone starting at 1-2 g orally in bananas and then decreasing over a 1-3 wk period of time. The colic was treated symptomatically utilizing trimethoprim sulfa orally, flunixin meglumine orally, and mineral oil floated on the drinking water with jello powder for flavoring. All medications were accepted, and the animal's appetite increased from half to full feed within 1 wk. The antibiotic regimen was changed after 10 days of trimethoprim sulfa treatment to amoxicillin 20 g once daily in the food.

Blood was obtained for CBC and serum chemistry via the left medial vein while the animal was being fed treats such as bananas. Abnormalities included a severe but responsive macrocytic anemia (PVC=13) and hypophosphatemia (1.2 mg/dl). Fresh blood had been found over this period on the cage floor several times but never in amounts over 200 ml per episode. Hematuria, hemoglobinuria, hemolysis, or melena were never found in numerous stool, urine, or serum samples obtained at this time and until the animal's death.

Because the animal was thought to have bred successfully and was in the last trimester of pregnancy, drugs safe for treatment of pregnant horses were chosen to treat a possible ulcerative stomatitis lesion in the back of the mouth or nasal cavity which did not bleed overtly or possible gastric/duodenal ulcerations. Prednisone was given at 500 mg p.o. s.i.d. for 2 days and then q 48 hr for 3 doses. After this, 250 mg was given p.o. q 48 hr for 5 doses and then 125 mg p.o. q 48 hr for 5 doses. To help prevent secondary fungal infection 5 g thiabendazole p.o. s.i.d. was maintained throughout the steroid treatment. Cimetidine, 1.2 g s.i.d. p.o., and sucralfate, 2 g t.i.d. p.i., were used for ulcer treatment. Ferrous gluconate was used as an iron supplement but caused emesis so the treatment was withdrawn after use for 2 days.

These treatments were initiated about December 15, 1988 and by January 19, 1989 the CBC indicated a rise in the PCV to 17% and serum chemistry showed elevation in the phosphorous level to 5.4 mg/dl (normal = 4.0 - 5.9 mg/dl). Again no other abnormal blood values were seen. appetite, attitude and general

appearance improved during this period.

During March 1989 eruptive ulcerative lesions appeared first where one would expect to find pressure sores, i.e., hocks, stifles, etc. and then spread to ear margins, tail tip, and coronary band areas. Serum testing for autoimmune hemolytic anemia was inconclusive, and skin biopsies taken from multiple sites were negative on indirect immunofluorescent staining with anti-rhino IgG, and anti-rhino serum. Direct histopath and electron microscopy found no evidence of viral lesions but suggested an immune mediated problem. Prednisone therapy was initiated again in the same sequence as in December 1989 and halted the progress of the lesions but failed to cause remission. Localized treatment with hydrogen peroxide and dilute povidone-iodine solution was also used.

By March 21, 1989 blood collection revealed improvement of the PCV to 22.5% while phosphorous remained in the normal range of 4.2 mg/dl. Again no other abnormal blood values were seen.

Pregnancy detection became paramount as day 550 (longest usual recorded normal gestation) was approached during mid-March 1989. Trans-abdominal and trans-rectal ultrasound as well a rectal palpation only revealed a fluid-filled uterus, likely a mucometra. This was contradicted slightly by observation of possible fetal movement in the right lower abdominal quadrant. but in light of ultrasound and palpation finding, of which the animal stood perfectly still while being fed treats, treatment with prostaglandins was initiated twice with only some mucus vulvar discharge as a result.

The animal reached a high point of PCV with 23% and a phosphorous level in the normal range of 4 mg/dl on April 5, 1989. After this the PCV remained 21-23% while the phosphorous level declined to 2-8 mg/dl and then 2.0 mg/dl. The general condition of the animal became worse with weight loss, further skin lesions, epistaxis and first, acute, and then progressive encephalopathy which was thought to be related to hemorrhage first in the brain stem and then further into the CNS. This progression occurred in spite of increased steroid dosage (up to 4 gm p.o. daily), vitamin A injections, vitamin E injections, and H-1 antihistamine blocker agents for the skin lesions.

On June 28, 1989 the animal was found dead. Significant necropsy findings included a uterine torsion of 2-to 3-day duration with a 50 lb normal fetus thought to be at the last part of the last trimester of pregnancy. Possible fulminant septicemia resulted from the uterine torsion. Other findings included severe hematochromatosis, renal cortical fibrosis, failure and resultant metastatic calcification and mild/moderate fungal (*Aspergillus* sp.) pneumonia and dermatitis (*Candida* sp). The fungal infections were regarded as a secondary complication to protracted antibiotic and steroid therapy.

Since this incident another black rhinoceros at the Cincinnati Zoo, 20-yr-old, with no previous incidents of stomatitis, became symptomatic in December 1989 with ulcerative skin and oral lesions and weight loss just prior to her transport back from the Columbus Zoo on breeding loan. She was medically managed similarly as in the first case. Again, as in the first case, PCV dropped from a first blood collection at 27% to a low point of 19%. Phosphorous blood levels dropped at the same point to a low level of 0.9 mg/dl. This time phosphorous supplementation was undertaken by adding 1 lb of bone meal daily to produce. Within 10 days

phosphorous reached a normal level of 5.7 mg/dl. PCV climbed to 34% on May 7, 1990 with a concurrent phosphorous level of 5.3 mg/dl. At 3 points from December 1898 - August 1990, when bone meal supplementation was discontinued, phosphorous levels decreased followed by decrease in PCV. Therefore, bone meal supplementation has been maintained continuously since late July 1990 with a stable PCV of 27-28% and phosphorous level of 4-5.2 mg/dl. Further changes are being monitored by weekly blood collections. As an additional note, the animal has been clinically normal since March 1990 without antibiotic, antifungal or steroid medications. Vitamin E levels have been in the normal captive range since February 1990 through use of TPGS orally and is not postulate to be a factor in the clinical improvement or PCV/phosphorous blood correlations.

Recommendations based on these experiences would include:

- 1) Conditional black rhinos in captivity possible for routine blood collection.
- 2) Diet analysis to include phosphorous supplementation, 1 to 2 lb bone meal orally daily, as needed.
- 3) Further research to explore relationship of phosphorous and PCV levels specifically in terms of relationship to reduced red blood cell survival times (no acute hemolysis) or cause/effect of hematochromatosis.
- 4) Hemolytic episodes in black rhino observed in zoos may represent an acceleration of the previously described events.
- 5) Ultra sound has definite limitations in determining pregnancy in the black rhino, especially with use of portable units which often only offer up to 15 cm penetration. Units which penetrate at least 20-25 cm. are recommended. Detection through saliva, urine, or blood collection must be refined and accelerated in their development.