ULCERATIVE STOMATITIS IN A BLACK RHINOCEROS (DICEROS BICORNIS)

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Introduction

Skin diseases in rhinos are fairly common (1,2,3) and lesions of the mucous membrane have also been described (2). Jones (2) reported a raised vegetative hemorrhagic lesion on the mucosa of the lip of a black rhinoceros that he felt was probably due to a metabolic disease since no primary pathogen could be isolated. The following report describes a similar lesion in a black rhinoceros at the Brookfield Zoo. Two other cases are also briefly described.

History

On October 1, 1980 an adult female black rhinoceros (Diceros bicornis) at the Brookfield Zoo developed slight epistaxis from her right nostril. The animal, estimated to be approximately seven

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years of age, had had no previous medical problems. She had been vaccinated for tetanus[a] in 1978 and 1980 and leptospirosis[b] in 1980.

The epistaxis continued for six days. During this period she was active and had a good appetite. She was treated with 20 mg dexamethasone[c] and 8 cc vitamin K.[d] Two days after the treatment was started, the epistaxis was minimal, but it resumed the following day. She was then treated with epinephrine nose spray and oral dexamethasone powder, 10 mg the first day and 5 mg once daily for the next four days. Nasal cultures grew Aeromonas hydrophila, alpha strep, Staph epidermidis, and Acinetobacter calco var. lwoffi.

On the 15th day of the epistaxis the rhino began pacing excessively in the evening and again on the morning of the 16th. On October 17th she was sedated with 3 mg etorphine[e] and examined. In her right nostril was a highly vascularized mass approximately 1-12" diameter and raised about 1/2" above the nasal mucosa. It was biopsied. A similar-appearing 2" diameter mass was found on the inside of her upper lip. The rhino was bled for a CBC and chemical screen and given intramuscularly both 60 ml of benzathine penicillin[f] and 40 ml doxapram.[g] The sedation was reversed with 6 mg diprenorphine[h] IV and an additional 21 mg IM.

Blood work (Table 1) showed a neutrophilic leukocytosis with a monocytosis and a slight eosinophilia and lymphocytosis. The blood chemistries revealed a slightly decreased serum phosphorus and increased SGOT and LDH.

The biopsy report was as follows: Nonspecific acute and chronic inflammation of the nasal mucosa. The specimen showed acanthosis and elongation of the rete ridges. The underlying subepithelial tissues indicated marked acute inflammation. There was a proliferation of capillaries with dense infiltrations of polymorphonuclear neutrophils, eosinophils and lymphocytes.

On the 21st day of the epistaxis, the rhino was given 1 g prednison[i] once daily for six days then decreased to 500 mg once daily for seven days. She was also given 7.5 g ampicillin[j] twice daily for seven days. The epistaxis stopped within three days after the start of treatment.

However, 18 days after the prednisone was discontinued some blood was seen at her mouth. The animal appeared to be evidencing some pruritis as she would rub her nostril around the stall. The bleeding increased over the next week. She was given 1 g prednisone once daily for three days then 500 mg once daily for four days. The bleeding stopped after two days of treatment.

Nine days after the end of treatment, more bleeding was observed. Five days later, fruits, vegetables and peanut oil were withdrawn from her diet over a three day period. Bleeding continued and five days later she was put back on a gram of prednisone per day.

The animal by this time was trained to allow examination of her mouth and, at the onset of bleeding, a raised mass (2" long, 3/4" wide, 1/4" high) was seen on her upper lip. On the second day of treatment all bleeding stopped. By the third day the raised mass disappeared, leaving a deep ulcer. The wound continued to decrease in size throughout the period of prednisone treatment. Steroids were administered until the ulcer completely healed, one month after the mass was examined.

The rhino's mouth and nostrils continued to be examined daily with no evidence of lesions reappearing. On April 8, 1981, a small elevated bump was observed on the upper lip which disappeared four days later without treatment.

The rhino was fed only grain[k] and alfalfa hay until September 4, 1981, at which time peanut oil was added to her diet. Nine days later, a small raised pale stellate plaque was observed on her right upper lip. It remained unchanged for 21 days. Two days later, two more small reddened raised plaques were observed on the right side of her upper lip. The peanut oil was removed from her diet. Five days later the lesions faded away.

On January 6, 1982, apples were added to her diet and over the next seven months other food items (carrots, hydroponic grass, bananas and bread) were added one at a time to her diet with no ill effect. On October 2, 1982 peanut oil was added to her diet. No reoccurrence of the lesions developed over the next month and a half.

Medical histories were obtained on two other clinical cases of epistaxis in black rhinoceros. One case occurred at the San Diego Zoo, involving a 16-18 year old male, and the other, a 13 year old female at the Detroit Zoo.

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a. Tetanus Toxoid - Fort Dodge, IA 50501.
b. Leptoferm-5 - Norden Laboratories, Lincoln, NE 68501.
c. Azium - Schering Veterinary, Kenilworth, NJ 07033.
d. AquaMEPHYTON - Merck Sharp & Dohme, West Point, PA 19486.
e. M99 - Lemmon Company, Sellersville, PA 18960.
f. Flo-cillin - Bristol Laboratories, Syracuse, NY 13201.
g. Dopram-V - A.H. Robins Company, Richmond, VA 23220.
h. M50/50 - Lemmon Company, Sellersville, PA 18960.
i. Prednisone USP - Pharma-Tek, Inc., Huntington, NY 11743.
j. Polyflex - Bristol Laboratories, Syracuse, NY 13201.
k. Anderson's 703 - The Andersons, Maumee, OH 43537.

The San Diego animal first developed an oral bleeding mass on August 19, 1979. Following histopathological examinations, the mass was described as a chronic granulation tissue with abundance of eosinophils, dilated capillaries and pseudoepitheliomatous hyperplasia of the oral mucosa with surface ulceration and bacterial growth. The animal showed an anemia and a marked neutrophilic leukocytosis with a left shift and an eosinophilia. The serum phosphorus, albumin and proteins were also low.

The animal did not respond to vitamins, antibiotics and/or diet changes to improve the composition of the diet and decrease the roughness of the food. The animal developed loose stool and weight loss. Bleeding did decrease after treatment with 100 mg to 1 g prednisone daily. The bleeding would stop two days after the onset of the treatment, but would reoccur shortly after the animal was taken off steroids. This continued until June 1980 when he deteriorated and died of a systemic mycotic infection.

The Detroit female had chronic skin lesions (sores on her hips and hocks) for fourteen years and epistaxis for four years. In May 1972 she developed multiple oral lesions which were biopsied in 1973 as an eosinophilic granuloma. She was hypoproteinemic and anemic. She was treated with vitamins A, C and E and selenium. It was determined that her diet was low in protein and calcium phosphorus ratio (Ca/P) so the diet was modified to increase the protein composition, improve the Ca/P and the mouth and skin lesions gradually improved over time with no reoccurrence.

Discussion

The three cases described here each have several things in common (chronic oral lesions similar to an eosinophilic granuloma lesions, neutrophilic leukocytosis with eosinophilia, response to steroid treatment, and response to diet changes [Table 2]). One possible etiology considered, which would include these factors, was a nutritional deficiency predisposing the animal to a generalized debilitated condition and an immunological problem. Indeed Jones (2) reported that captive rhinoceros have very low serum vitamin A levels, and administration of large doses of vitamin A (2-4 million units, IM) has produced improvement in some animals with skin lesions.

The diet for the Brookfield animal prior to dietary changes appeared to be nutritionally balanced with an 18.4 percent protein (on a dry matter basis), a Ca/P of 2.3/1 and vitamin levels of 11,300 IU/kg (on a dry matter basis). So another possible etiology considered for her was a dietary allergy which responded to dietary changes and steroids. When peanut oil was added back to the diet of this female, a lesion appeared on her lip and disappeared when the peanut oil was no longer fed. However, when peanut oil was added at a later time, no lesion resulted. Therefore, the dietary allergy theory is still in question.

Stress is another possible cause, but documenting it is difficult. There was a black rhino at the Cincinnati Zoo which showed similar oral lesions which regressed without treatment. The lesions were thought to be associated with a stressful situation (4).

Therefore, in order to treat ulcerative stomatitis in rhinos, one should evaluate for any stressful situations and attempt to eliminate the stressors. In addition to this, dietary changes, treatment with systemic steroids and injectable vitamin A may be considered.

TABLE 1

BLOOD RESULTS

CBC:

WBC	31.80 x $10^3/mm^3$	Neutrophils	69%
RBC	4.81 x $10^6/mm^3$		15,042
Hb	14.30 gms %	Lymphocytes	22%
PCV	38.00%		4,796
MCV	79.00u^3	Monocytes	8%
MCHC	37.00%		1,744
MCH	30.00$u\mu^G$	Eosinophils	1%
T.P.	6.10 gm %		218

SERUM CHEMISTRIES:

Calcium	10.40 mg/dl	SGPT	33.00 IU/l
Phosphorus	3.40 mg/dl	Alkaline Phosphatase	22.00 IU/l
BUN	14.00 mg/dl	LDH	401.00 IU/l
Creatine	1.30 mg/dl	Cholestrol	56.00 mg/l
Uric Acid	0.60 mg/dl	Iron	168.00 mcg/dl
Glucose	121.00 mg/dl	Magnesium	1.81 mcg/l
Albumin	2.50 gm/dl	Sodium	127.00 mmol/l
Globulin	4.10 gm/dl	Potassium	4.50 mmol/l
Total Bilirubin	0.37 mg/dl	Chloride	101.00 mmol/l
Direct Bilirubin	0.17 mg/dl	Triglycerides	68.00 mg/dl
SGOT	137.00 IU/l	G-Glutamyl Transpep.	17.00 u/l

TABLE 2

FACTORS IN COMMON BETWEEN THE THREE CLINICAL CASES

FACTORS	BROOKFIELD	SAN DIEGO	DETROIT
1. Chronic oral ulcers Histo: eosinophilic granuloma	X	X	X
2. Neutrophilic leukocytosis with eosinophilia	X	X	
3. Response to steroid treatment	X	X	
4. Response to diet changes	X	?	X

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