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IDIOPATHIC DISTAL ESOPHAGEAL DILATION IN A SOUTHERN BLACK RHINOCEROS (*DICEROS BICORNIS MINOR*)

Robin W. Radcliffe, D.V.M., James Schumacher, D.V.M., M.S., Sandee M. Hartsfield, D.V.M., M.S., Dipl. A.C.V.A., A. M. Merritt, D.V.M., M.S., and Michael J. Murray, D.V.M., M.S., Dipl. A.C.V.I.M.

Abstract: An adult female southern black rhinoceros (*Diceros bicornis minor*) experienced intermittent periods of regurgitation while eating, suggesting an esophageal disorder. Endoscopy for evaluation of the trachea, esophagus, and stomach revealed a 30-mm nasopharyngeal orifice with associated recess located in the caudodorsal pharynx and a 10-cm dilated segment of the distal esophagus that was presumably the cause of regurgitation. Dietary management of esophageal dilation through short-term utilization of a "soft feed" program successfully eliminated the regurgitation. This is the first report of esophageal dysfunction in a rhinoceros.

Key words: black rhinoceros, *Diceros bicornis*, regurgitation, esophageal dilation, endoscopy.

INTRODUCTION

Esophageal dilation with regurgitation has not been reported in any of the five species of rhinoceros. The domestic horse, however, a closely related perissodactylid, may provide a model for the evaluation of this condition in the rhinoceros; a variety of equine esophageal diseases can present with signs of regurgitation.[6–10,12–14,16] This case report details apparent esophageal dysfunction in a female southern black rhinoceros (*Diceros bicornis minor*) and compares and contrasts the condition in the rhinoceros and the horse.

CASE REPORT

A 16-yr-old, wild-caught, female southern black rhinoceros ("Sinampande," studbook #0466, estimated body weight 1,200 kg), transported from Zimbabwe to the United States in April 1992, suddenly began regurgitating on 25 November 1996. The rhinoceros's diet at the time of regurgitation consisted of 25 kg herbivore pellets (20 kg Mazuri ADF-16 No. 5648 and 5 kg Mazuri ADF-25 No. 5649, Purina Mills, St. Louis, Missouri 63166, USA), 2 kg Equine Athlete (Purina Mills), 14 kg alfalfa hay, and occasional sliced apples and yams as treats.

The rhinoceros developed signs of dysphagia; she would eat normally for approximately 15–30 min without incident, then would suddenly drop food from her mouth, extend her neck, and regurgitate approximately 0.5 L of mucus mixed with macerated food (Fig. 1). This behavior was repeated several times at each feeding, interspersed with short periods of normal eating and swallowing. The pH of the regurgitated contents registered 8.0–8.5 with litmus paper on several occasions. Blood was collected without sedation, and with the exception of a low serum phosphorus concentration (2.1 mg/dl), the CBC and serum chemistry values were within normal limits. The volume and frequency of regurgitation increased over the next 7 days.

Treatment included p.o. administration of sulfamethoxazole/trimethoprim, 19.2 g b.i.d. for 30 days (Biocraft Laboratories, Elmwood Park, New Jersey 07407, USA); sucralfate, 2 g b.i.d. for 3 days followed by 3 g t.i.d. for 11 days (Carafate, Marion Merrell Dow, Kansas City, Missouri 64114, USA); phenylbutazone paste, 6 g once (Equiphen, Luitpold Pharmaceuticals, Shirley, New York 11967, USA); ivermectin paste, 1.87%, 224.4 mg once (Eqvalan, Merck & Co., Rahway, New Jersey 07065, USA); and 227 g Medo-Lyte-8X Powder in 56 L of drinking water s.i.d. for 7 days (Fermenta Animal Health Co., Kansas City, Missouri 64153, USA). The diet was supplemented with 454 g steamed bone meal s.i.d. for 30 days (American Protein Corporation, Lytton, Iowa 50561, USA). To reduce esophageal irritation, the amount of alfalfa hay was decreased but not completely eliminated because she was lactating with an 11-mo-old calf.

Ten days after the onset of clinical signs, the rhinoceros was fasted for 30 hr and then immobilized with a combination of 2.3 mg etorphine HCl (M99,

From the Fossil Rim Wildlife Center, Department of Animal Health Services, P.O. Box 2189, Route 1, Box 210, Glen Rose, Texas 76043, USA (Radcliffe); Texas A&M University, College of Veterinary Medicine, College Station, Texas 77843, USA (Schumacher, Hartsfield); the University of Florida, College of Veterinary Medicine, Box 100136 HSC, 2015 SW 16th Avenue, Gainesville, Florida 32610, USA (Merritt); and Marion DuPont Scott Equine Medical Center, Virginia–Maryland Regional College of Veterinary Medicine, Virginia Tech., P.O. Box 1938, Leesburg, Virginia 21077, USA (Murray).

Address correspondence and reprint requests to Robin W. Radcliffe, D.V.M.

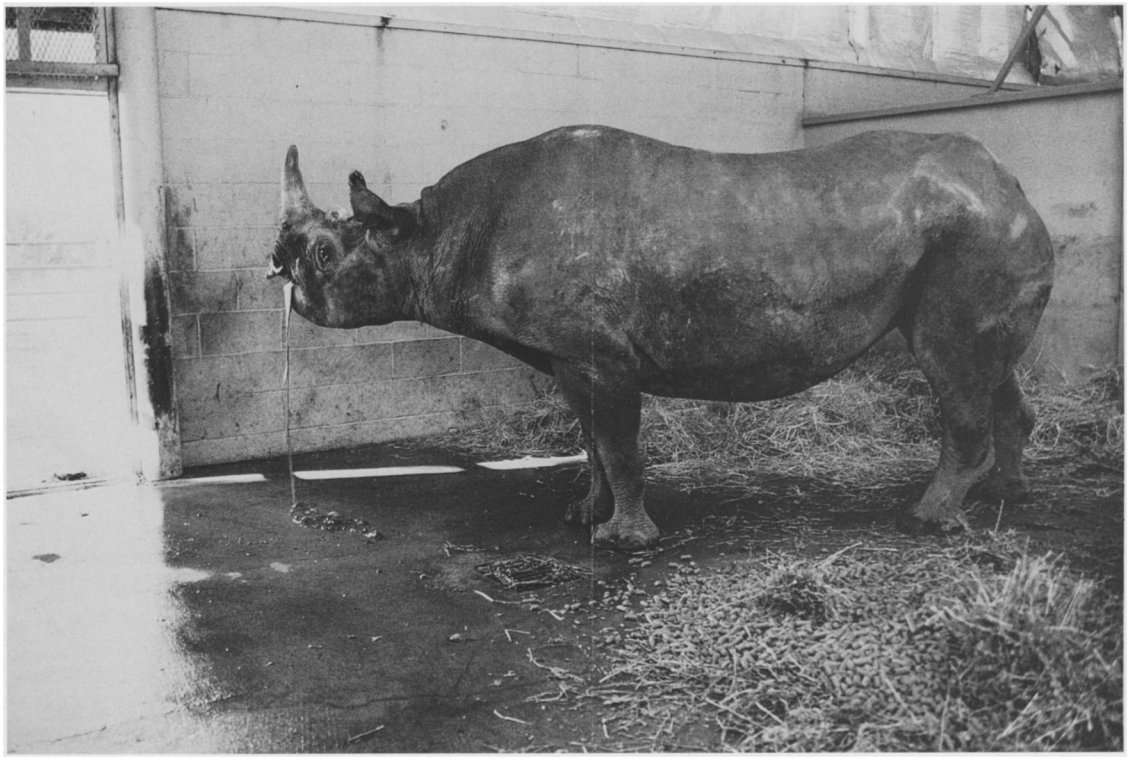

Figure 1. Regurgitation in a southern black rhinoceros (*Diceros bicornis minor*).

Wildlife Pharmaceuticals, Fort Collins, Colorado 80524, USA), 100 mg azaperone (Stresnil, Janssen Pharmaceutica, Mississauga, Ontario L5N 5R9, Canada), and 1,500 USP (TR) units hyaluronidase (Wydase lyophilized, Wyeth Laboratories, Philadelphia, Pennsylvania 19101, USA) mixed in one syringe and delivered i.m. by hand injection. The rhinoceros was positioned in sternal recumbency on a mattress. While the glottis was viewed via an endoscope passed nasally, a stiff nasogastric tube was passed into the trachea through the mouth. This orotracheal tube was used as a "guide" over which a 30-mm-inside-diameter, Murphy-type, cuffed endotracheal tube (Aire-cuf, Bivona, Gary, Indiana 46406, USA) was advanced into the trachea and the cuff inflated.[3]

Anesthesia was maintained with isoflurane (0.5%–2%, AErrane, Ohmeda Caribe, Guayama, Puerto Rico 00784, USA) delivered in oxygen at a flow rate of 7–10 L/min by a large animal gas anesthesia machine (Drager LA, North American Drager Inc., Telford, Pennsylvania 18969, USA). Spontaneous ventilation was maintained throughout the procedure and ranged from 4 to 12 breaths/min. Lactated Ringer's solution (3 L) and 5% dextrose (2.5 L) were administered i.v. Additional medications included 24 g ampicillin sodium i.v. (Amp-Equine, SmithKline Beecham Animal Health, West Chester, Pennsylvania 19380, USA), 7.92 g amikacin sulfate i.v. (Amiglyde-V, Aveco Co. Inc., Fort Dodge, Iowa 50501, USA), 3.6×10^7 IU penicillin G benzathine/procaine i.m. (Crystiben, Solvay Animal Health Inc., Mendota Heights, Minnesota 55120, USA), and 1 g flunixin meglumine i.m. (Banamine, Schering-Plough Animal Health Corp., Kenilworth, New Jersey 07033, USA).

The nasopharynx, cranial trachea, esophagus, and stomach were examined with a 3-m, flexible video-endoscope (Olympus America Inc., Melville, New York 11747, USA).[15] The first notable finding was a diverticulum located in the caudodorsal pharynx. The orifice, measuring approximately 30 mm in diameter and surrounded by intact mucosa, opened into a cavity just dorsal to the glottis (Fig. 2). The second finding, and likely the cause for regurgitation, was an apparent dilation of the lumen of the esophagus. A 10-cm length of esophagus located just proximal to the cardia did not collapse around the endoscope during examination (Fig. 3). The esophageal mucosa in this area appeared thick and wrinkled. The remainder of the esophagus appeared healthy and the lumen collapsed during ob-

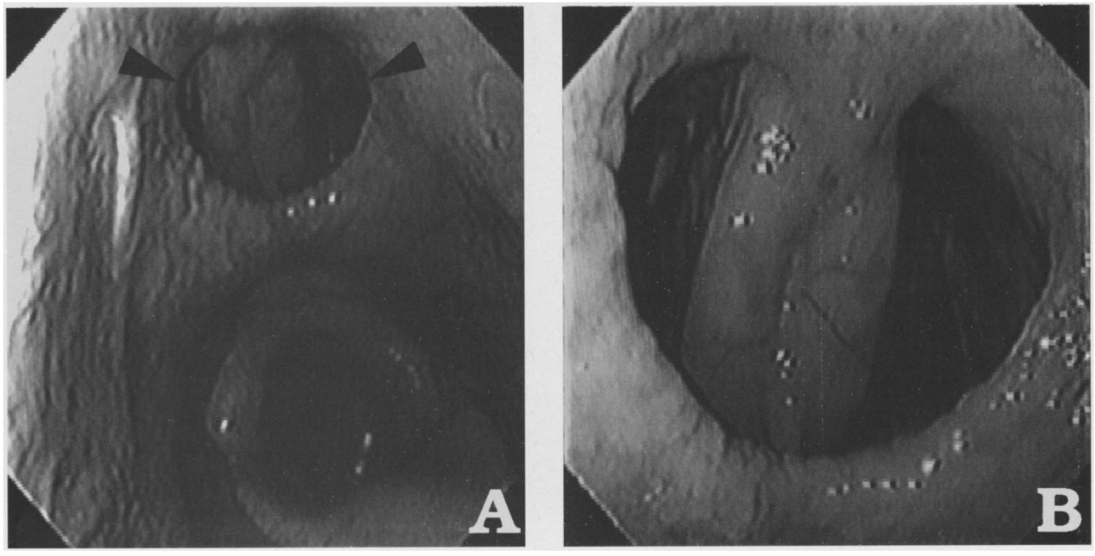

Figure 2. An endoscopic view from the nasal passage illustrating the diverticulum (arrows) in the dorsocaudal pharynx of a female black rhinoceros (**A**). A close-up view of the nasopharyngeal orifice and the associated recess (**B**).

servation, as is characteristic of a normal esophagus during endoscopy.[15] No abnormality of the stomach was found. The trachea was free of debris.

Blood was collected for diagnostic tests, including a CBC, serum chemistries, serum thyroid hormone (T3/T4) concentrations, blood lead levels, and an acetylcholine receptor antibody test. No significant change was noted in the CBC and serum chemistry values. Serum concentrations of thyroxine (T4) measured 1.1 μg/dl, and triiodothyronine (T3) was undetectable. Assays for lead and acetylcholine receptor antibody within the blood were negative.

Sternal recumbency was maintained for 126 min, with shifting of the rhinoceros into opposite semisternal position approximately every 30 min. At the end of the procedure, the etorphine effect was antagonized with 150 mg naltrexone (Trexonil, Wildlife Pharmaceuticals) administered half i.v. and half i.m., and the rhinoceros stood in less than 1 min.

On the basis of recommendations for clinical management of esophageal problems in domestic horses,[14] the diet of the black rhinoceros was changed to a completely "soft" feed consisting of the original herbivore pellets (20 kg Mazuri ADF-16 No. 5648 and 5 kg Mazuri ADF-25 No. 5649), 2 kg Equine Athlete, and 11 kg finely ground alfalfa. Within 24 hr of this dietary change, regurgitation resolved. During this period of nutritional modification, the rhinoceros and her calf developed diarrhea, which may have been related to the lack of coarse fiber in the diet. After 7 days without regurgitation, but with persistent diarrhea, 5 kg of alfalfa cubes was added to the diet. Regurgitation recurred and the alfalfa cubes were discontinued. The "soft" diet was then maintained until 30 days after the immobilization for endoscopy when, because of ongoing problems with diarrhea and weight loss, the diet was rapidly changed to include alfalfa and grass hay. The regurgitation did not return, and the diarrhea resolved.

DISCUSSION

Esophageal problems in domestic animals have been described that cause acute regurgitation.[6–13,16] These problems include obstruction with a foreign body; esophageal stricture; extrinsic esophageal compression, such as from lymphadenopathy or neoplasia; esophageal diverticulum; esophagitis; toxicity, including heavy metals or botulism; neuromuscular disorders; and other causes of motility dysfunction. The alkaline pH of the oral reflux suggested that the material was regurgitant of esophageal origin and not vomitus of gastric origin. In horses, the inability to vomit is thought to be directly related to the high, intrinsic myogenic tone of the lower esophageal sphincter,[9,12] although this has never been proven. Because of the location of the dilation immediately cranial to the cardia, dysfunction of the lower esophageal sphincter may have precipitated esophageal dilation, and thus regurgitation, in this rhinoceros.

Hypothyroidism has been implicated as a possible cause of esophageal dysfunction associated with various musculoskeletal disorders, including me-

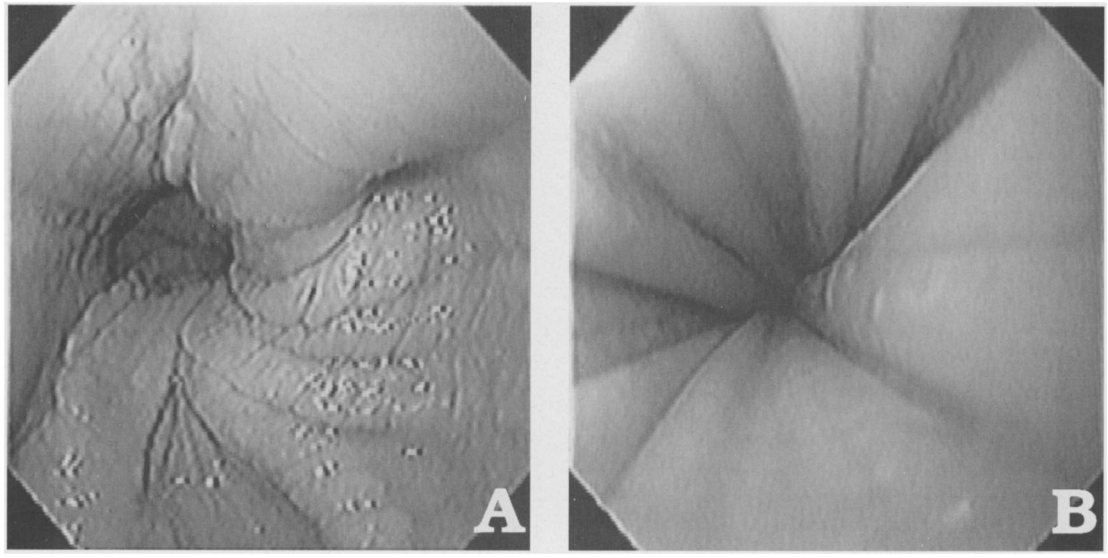

Figure 3. An endoscopic view of a dilated esophageal segment located anterior to the cardia of the stomach in a black rhinoceros. Note the thickened, rugal pattern of the mucosa (**A**). Compare the appearance of normal esophagus cranial to the dilated area (**B**).

gaesophagus in dogs and rhabdomyolysis in horses.[11,12] The T4 concentration in this rhinoceros was normal, and the T3 concentration was low compared with normal ranges in the horse;[13] however, these findings were not significantly different from those in two other healthy black rhinoceroses at this facility. In addition, the diagnosis of hypothyroidism through interpretation of resting T3/T4 levels is complicated, even in domestic species, because these baseline values are influenced by such diverse factors as age, gender, season, anorexia, and administration of pharmacologic agents such as phenylbutazone.[9,12,13]

The absence of mucosal erosions and the yellow-orange, hyperkeratotic lesions characteristic of inflammation caused by gastric reflux suggested that esophagitis was not a factor in development of this esophageal problem.[9] Because the affected esophageal area appeared rigid, fibrous tissue may have formed in the esophageal wall, perhaps secondary to a previous obstructive event. Mural fibrosis would indicate a chronic course to the esophageal dilation because a minimum of 30–60 days is presumably required for fibrous tissue formation in the equid esophagus.[9] This black rhinoceros may have arrived from the wild with this condition and only later developed signs in captivity.

One of the most severe and life-threatening secondary complications of regurgitation in the horse is aspiration pneumonia. The trachea of this rhinoceros appeared normal during endoscopic evaluation, even after 10 days of regurgitation, suggesting that aspiration and pneumonia had not occurred. The typical clinical presentation of regurgitation in the horse includes dysphagia, ptyalism, and nasal return of food and saliva.[1,10,12,16] In this rhinoceros, regurgitation episodes consisted entirely of oral discharge of mucus and feed, with a lack of nasal involvement (Fig. 1). Presumably, this disparity in clinical signs may be related to anatomic and/or functional differences in the soft palate and nasopharynx between the rhinoceros and the horse.

Large feed particle size was likely a factor in the development of clinical signs because regurgitation recurred with the addition of alfalfa cubes to the diet. Perhaps the abnormal esophageal area began to dilate further as fibrous foods were eaten, and this "ballooning" precipitated the regurgitation. This would account for the observed delay in regurgitation signs for up to 30 min after the onset of eating. Why esophageal rest for 30 days through feeding of a "soft" diet was followed by amelioration of the problem is unclear.

The nasopharyngeal diverticulum (Fig. 2) probably had no relation to the clinical problem. This structure is not present in the horse,[1,2,15] but an epipharyngeal bursa has been described previously in the rhinoceros.[4,5] On the basis of anatomic studies of the Rhinocerotidae family, the epipharyngeal bursa appears to constitute a functional aggregate of mucous glands, lymphoid nodules, and an elaborate network of vessels and sensory nerve end-

ings—a complex representing the epipharyngeal tonsil.[4,5]

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