*Journal of Zoo and Wildlife Medicine* 47(3): 876–878, 2016


# CONGENITAL CLEFT PALATE AND CARDIAC SEPTAL DEFECTS IN A NEONATAL SOUTHERN BLACK RHINOCEROS (*DICEROS BICORNIS MINOR*)

**Stephany Lewis, D.V.M., Mary Duncan, B.V.M.S., Ph.D., Dipl. A.C.V.P., M.R.C.V.S., Marlys L. Houck, C.G.(A.S.C.P.)CM, Rebecca Bloch, D.V.M., and Holly Haefele, D.V.M.**

*Abstract:* A female Southern black rhinoceros (*Diceros bicornis minor*) calf died unexpectedly at less than 12 hr of age, after an uncomplicated birth and uneventful early postpartum period. Gross necropsy revealed a 15-cm full thickness cleft palate, a patent foramen ovale, and four septal defects ranging from 0.3 to 1 cm in diameter. Histologic findings did not reveal any significant abnormalities. Karyotyping did not indicate any significant numerical or structural chromosomal abnormalities.

*Key words:* Atrial septal defect, black rhinoceros, cleft palate, congenital cardiac disease, *Diceros bicornis,* ventricular septal defect.

## BRIEF COMMUNICATION

A female Southern black rhinoceros (*Diceros bicornis minor*) was born without complication as the first calf of a 12-yr-old dam. The pair was housed separately from the other rhinoceros at Fossil Rim Wildlife Center, Glen Rose, Texas. The dam was born to a female imported from Zimbabwe, and housed at Fossil Rim since 1995. Good maternal care was apparent, and the calf appeared outwardly normal. Approximately 8 hr after birth, the calf was ambulatory, moving with the dam, and attempting to nurse. At less than 12 hr of age, the calf was witnessed pacing behind the dam and within minutes became laterally recumbent with increased respiratory effort. The dam was immediately separated from the calf, but the calf was already dead on retrieval.

A necropsy was performed within 3 hr of death. The calf was in good postmortem condition and good body condition, with ample retroperitoneal fat, and a body weight of 32.02 kg. Gross examination revealed small superficial abrasions on the right forelimb and both hind limbs, but no other evidence of external trauma. There was mild subcutaneous petechiation of the ventral thorax and subcutaneous edema with mild bruising on the ventral chin. Oral examination revealed a complete cleft palate, about 15 cm long, extending through both the soft and hard palate, just caudal to the lip (Fig. 1).

The stomach contained a small amount of clear fluid and a blade of grass. The serosal surface of the small intestines was diffusely mildly to moderately hyperemic, particularly from the distal jejunum to ileum. The duodenal contents consisted of mucus primarily, but there was increasingly solid yellow feces throughout the jejunum and colon.

The lungs appeared heavy and congested with clear fluid, and there was a small amount of foam in the proximal trachea. Externally the heart was the expected size and shape. There were two 1-cm diameter round communications in the interventricular septum. The first was close to the apex of the heart, and the other approximately 2.5 cm closer to the heart base (Fig. 2). Two additional smaller ventricular septal defects were located between the larger defects, 0.3 and 0.5 cm in diameter. An incomplete closure of the foramen ovale created a 1.5-cm diameter atrial septal defect (Fig. 2). All other examined tissues were grossly within expected limits.

Samples were fixed in 10% buffered formalin and processed routinely for light microscopy. Tissue sections were stained with hematoxylin-eosin. Histologic diagnoses were acute multifocal skeletal muscle degeneration and mild corneal vascularization, which were not considered significant to the cause of death. Remaining findings were considered typical for a neonate.

From the Fossil Rim Wildlife Center, 2155 County Road 2008, Glen Rose, Texas 76043, USA (Lewis, Haefele, Bloch); the Saint Louis Zoo, One Government Drive, St. Louis, Missouri 63110, USA (Duncan); and the San Diego Zoo Institute for Conservation Research Genetics Division, 15600 San Pasqual Valley Road, Escondido, California 92027, USA (Houck). Present addresses (Lewis): Michigan State University College of Veterinary Medicine, 784 Wilson Road, East Lansing, Michigan 48824, USA; (Bloch): Banfield Pet Hospital, 2550 Timber Drive, Garner, North Carolina 27529, USA. Correspondence should be directed to Dr. Holly Haefele (hollyh@fossilrim.org).

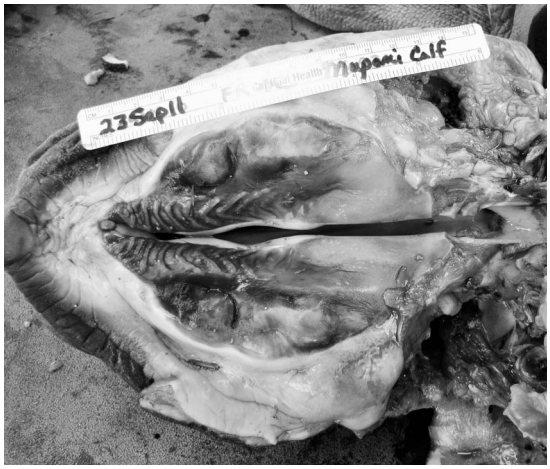


**Figure 1.** Black rhinoceros calf with cleft palate at gross necropsy examination.

A skin biopsy sample was submitted to the San Diego Zoo Institute for Conservation Research Genetics Division for cell culture and karyotyping. A fibroblast cell line was established from the biopsy and accessioned into the Frozen Zoo® cell repository (Lab no. 18843-8530). Standard and G-band karyotype analyses indicated a normal 2n = 84, XX female chromosomal constitution; however, the high diploid number in this species made it difficult to confidently align and analyze chromosomes beyond the 20 largest autosomal pairs and the X chromosomes. Pair 2 was polymorphic for the presence or absence of the p-arm (small arm) in all cells analyzed. Size polymorphisms involving short arm additions have been reported in black, white, Indian, and Sumatran rhinos, but they have not been linked to any phenotype abnormalities.[4,5] No other numerical or structural chromosomal differences or abnormalities were seen; however, the level of resolution provided by karyotyping may not identify microdeletions or small mutations.

Based on gross findings, multiple ventricular septal defects, ostium secundum atrial septal defect, and cleft palate were diagnosed.

Congenital anomalies have been infrequently reported in rhinoceros species. Patent urachus and schistosomus reflexus have both been reported in the Southern white rhinoceros.[1,8] Cardiac disease is rarely reported in rhinoceros. Streptococcal endocarditis has been described in a captive Southern white rhinoceros, and myocardial Purkinje degeneration and necrosis with fibrosis has been reported in free-ranging black rhinoceroses.[6,7]

Cleft palate, or palatoschisis, is a congenital defect caused by failure of embryological closure

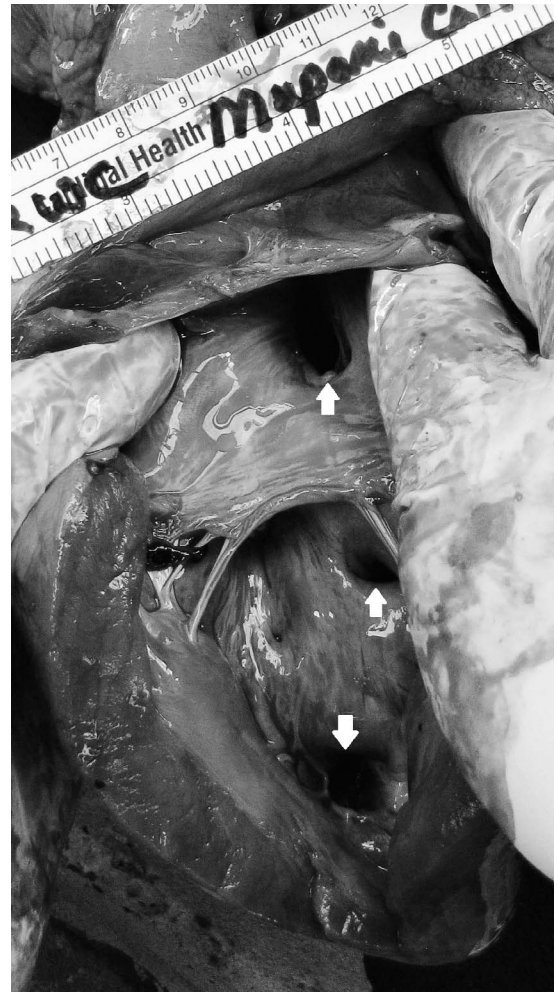

**Figure 2.** Black rhinoceros calf heart with an atrial and multiple ventricular septal defects at gross necropsy examination.

of the palatinel folds at midline. It has a reportedly higher incidence in females, and can often occur with other congenital defects. It typically results in aspiration pneumonia and rhinitis, and has been reported in a great number of species. Surgical repair can be attempted, and has been successful in an American bison.[3,9]

Atrial septal defects have been reported in a number of species.[10,11] Patent foramen ovale is relatively common in cattle, caused by failure of the septum primum, the valve of the foramen ovale, to become adherent to the crista dividends after birth. It is unknown when this closure occurs in rhinoceros neonates, and lack of closure at 12 hr is not necessarily a pathologic lesion. Atrial septal defects may cause left-to-right shunting of

blood, pulmonary overcirculation, and right heart enlargement, but do not always lead to clinical disease. Atrial septal defects are frequently seen with other cardiac anomalies, including ventricular septal defects.[11]

Ventricular septal defects have been reported in many species, including dogs, cats, cattle, horses, primates, camelids, birds, marine mammals, and a blue duiker.[2,10,11,13] They are the most common congenital cardiac defect in horses and domestic ruminants, particularly at the membranous portion of the interventricular septum. The defect is believed to result from failure of fusion of part of the endocardial cushion and muscular ventricular septum, or of the truncal and conal septa. Ventricular septal defects cause left-to-right shunting of blood, resulting in pulmonary overcirculation, left atrial and ventricular enlargement, and occasionally right heart enlargement, depending on their size and location. Volume overload and congestive heart failure result if the defect is large. Definitive antemortem diagnosis of ventricular septal defect requires echocardiography or angiocardiography. Several methods of surgical repair are possible, but the defects are associated with a high mortality rate.[11]

Congenital anomalies are generally idiopathic. Poor maternal nutrition, viral infection, environmental contaminants, drugs, chromosomal abnormalities, and genetics have been implicated in congenital heart disease as well as cleft palate.[2,3,11] At this time, the cause of the congenital abnormalities in this black rhinoceros is unknown.

*Addendum:* Congestive heart failure secondary to multiple congenital heart defects in a six-day-old black rhinoceros (Diceros bicornis) 1996. Reese, KW and Edwards, JL. Proceedings of the American Association of Zoo Veterinarians, Puerto Vallarta, Mexico. Pp. 424–430. This Proceedings article was brought to the authors' attention at the time the report was going to press. The article describes the presence of patent ductus arteriosus, ventricular septal defect and atrial septal defect in a captive born rhinoceros. A personal communication in the article refers to two full sibling rhinoceros calves that died at 10 days with a tricuspid valve defect and at 2.5 mo with a ventricular septal defect respectively.

*Acknowledgments:* The authors would like to thank all the staff and volunteers at Fossil Rim Wildlife Center. We thank Julie Fronczek, Mary-Ann Domingos, and Grace Magee for technical support.

## LITERATURE CITED

1. Bloch RA, Haefele H, Stevens L. Medical management of a patent urachus in a Southern white rhinoceros (*Ceratotherium simum simum*) calf. J Zoo Wildl Med. 2014;45(2):420–422.

2. Evans DE, Tully TN, Strickland KN, Williams JF, Rich GA. Congenital cardiovascular anomalies, including ventricular septal defects, in 2 cockatoos. J Avian Med Surg. 2001;15(2):101–106.

3. Hedlund CS. Surgery of the digestive system. In: Fossum TW (ed.). Small animal surgery. 2nd ed. St. Louis (MO): Mosby; 2002. p. 285–290.

4. Houck ML, Ryder OA, Kumamoto AT, Benirschke K. Cytogenetics of the Rhoncerotidae. Sonderdruck aus Verhandlungbericht des 37. Internationalen Symp über die Erkrankungen der Zootiere, Dresden; 1995. p. 25–32.

5. Houck ML, Ryder OA, Vahala J, Kock RA, Oosterhuis JE. Diploid chromosome number and chromosomal variation in the white rhinoceros (*Ceratotherium simum*). J Hered. 1994;85:30–34.

6. Houszka M, Dzimira S, Krol J, Kandefer-Gola M, Ciaputa R, Sobieraj L, Podkowik M. Streptococcal endocarditis in a captive Southern white rhinoceros (*Ceratotherium simum simum*). J Zoo Wildl Med. 2014; 45(3):635–637.

7. Kock ND. Myocardial Purkinje degeneration and necrosis with fibrosis in free-ranging black rhinoceroses (*Diceros bicornis*) in Zimbabwe. J Wildl Dis. 1996; 32(2):367–369.

8. Lankton JS, VanderHart DJ, Terrell SP. Schistosomus reflexus-like malformation in a Southern white rhinoceros (*Ceratotherium simum simum*). J Zoo Wildl Med. 2014;43(3):708–711.

9. Minter LJ, Karlin WM, Hickey MJ, Byron CR. Surgical repair of a cleft palate in an American bison (*Bison bison*). J Zoo Wildl Med. 2010;41(3):562–566.

10. Powell JWB, Archibald RT, Cross CA, Rotstein DS, Soop VM, McFee WE. Multiple congenital cardiac abnormalities in an Atlantic bottlenose dolphin (*Tursiops truncates*). J Wildl Dis. 2009;45(3):839–842.

11. Reef VB, McGuirk SM. Diseases of the cardiovascular system. In: Smith BP (ed.). Large animal internal medicine. 4th ed. St. Louis (MO): Elsevier; 2009. p. 453–489.

12. Reese KW, Edwards JL. Congestive Heart Failure Secondary to Multiple Congenital Heart Defects in a Six-Day-Old Black Rhinoceros (*Diceros bicornis*). In Annual Conference-American Association of Zoo Veterinarians 1996 (pp. 425–430). American Association of Zoo Veterinarians. Yulee, Florida USA.

13. Suedmeyer WK, Hitchcock LS, Bonagura JD, Kreeger J, Smith T. Ventricular septal defect in a blue duiker (*Cephalophius monticola*). J Zoo Wildl Med. 2000;31(3):408–411.

*Received for publication 26 December 2015*