CARDIAC TRUNCUS ARTERIOSIS IN AN EASTERN BLACK RHINOCEROS (*DICEROS BICORNIS MICHAELI*).

Josep Monné Rodríguez D.V.M., Dipl., E.C.V.P., Julian Chantrey, S. Unwin, Ranieri Verin Dip. , E.C.Z.M, Dipl., E.C.V.P.

Dept. of Veterinary Pathology and Public Health, Institute of Veterinary

Science, Leahurst Campus, University of Liverpool, Chester High Road, CH64 7TE, Neston, United Kingdom (Monné Rodríguez, Chantrey, Verin); North of England Zoological Society, Chester Zoo, United Kingdom (Unwin). Correspondence should be directed to Dr. R. Verin (rverin@liverpool.ac.uk)

Abstract: This brief communication describes a congenital truncus arteriosus diagnosed in a 1 month old Eastern black rhinoceros. Since the first day of life the animal was underweight and from the 22nd day presented with respiratory clinical signs that exacerbated with time leading eventually to collapse and death. Post-mortem examination revealed a single truncus arteriosus originating from the right ventricle giving origin to two separated pulmonary arteries and aorta with the ventricular septum showing a focal and completely communicating defect between the right and left ventricles. Based on the macroscopical examination and current human classifications the truncus arteriosus was classified as type III or A2. According to the authors knowledge this is the first description of a persistent truncus arteriosus in an Eastern black rhino.

Keywords: Truncus arteriosus, congenital, histopathology, Eastern black rhinoceros (*Diceros bicornis Michaeli)*.

**BRIEF COMMUNICATION**

A one-month and three-day old female Eastern black rhinoceros (*Diceros bicornis michaeli*) born in captivity at Chester Zoo (UK), was presented at one month old and below the average expected weight for an animal of that age. The calf was born to a dam in good body condition with no evidence underlying disease. The pregnancy was unremarkable. The birth occurred without complications and the calf stood one hour after been delivered and started suckling one hour later. Both dam and calf were kept in separate enclosures. Apart from mild stiffness of the hind limbs the calf did not show symptoms of illness until the 22nd day after birth when it was observed sneezing. From that day onwards the sneezing persisted and progressively exacerbated. Two days before death the calf was reported with sudden onset of lethargy, laboured breathing with hyperpnoea (60-70 bpm) and thermography imaging revealed cold spots. Separation from the dam was decided for supplementary milk replacement and to perform further physical investigation. The calf did not oppose any resistance to be caught, the rectal temperature was 35.1 ºC and despite the animal showed initial interest for the milk it did not show suckling reflex. The respiratory distress got worsen and eventually the calf collapsed with no response to resuscitation attempts.

At post mortem examination, the carcass presented in poor body condition as indicated by the moderately reduced muscular bulk and markedly prominent bony landmarks (especially dorsal processes of the vertebrae and ischiatic and iliac tuberosities of the pelvis). The oral mucosa and conjunctiva showed diffuse moderate reddening interpreted as congestion. The thoracic cavity contained abundant (approximately 0.5 L) transparent to mildly red tinged fluid (hydrothorax). The heart silhouette was severely enlarged and rounded occupying more than 50% of the thoracic space (Fig. 1A). The pericardium was diffusely severely distended and contained markedly increased amount of fluid (hydropericardium). Close examination of the heart revealed marked anatomical abnormalities (Fig. 1B). The base of the heart exhibited a single arterial trunk originating from the right ventricle. Approximately 3 cm distally from its origin, two independent pulmonary arteries originated from each side of the trunk (left and right), subsequently giving origin to the aorta (Fig. 1C). The cranial and caudal vena cava opened normally into the right atrium and left atrium was communicating with the lungs through 4 severely engorged pulmonary veins (two left and two right). The left and right ventricles communicated through a large 5x4 cm in diameter round defect in the upper aspect of interventricular septum immediately beneath the single arterial trunk (Fig. 1D). The base of this arterial trunk exhibited a single valve characterized by three irregular and thickened leaflets from which a narrow fibrous sheet was in continuity with the mitral valve through the septal defect. Other associated macroscopical changes consisted of diffuse severe congestion and pulmonary atelectasis (caused by the compression by the heart) as well as marked enlargement and congestion of the liver with moderately rounded edges.

Relevant samples of thoracic and abdominal organs were included in 10% buffered formalin and processed for histopathological investigation with Haemotoxylin and Eosin stain (H&E) for general examinations. Histopathology revealed moderate degeneration and multifocal single myofibre acute necrosis (Fig. 1E)The lungs showed diffuse severe congestion dominated by thickening of the alveolar septa due to marked capillary engorgement, intralveolar edema admixed with extravasated erythrocytes, and numerous activated alveolar macrophages with intracytoplasmic erythrocytes or golden brown pigment which was confirmed as hemosiderin by means of Perls’ Prussian blue stain (haemosiderophages-heart failure cells) (Fig 1F). The liver showed diffuse congestion with abundant presence of hemosiderin in Kupffer’s cells.

Based on macroscopical examination this abnormality was diagnosed as persistent truncus arteriosus, which is defined as a single great artery which gives origin to systemic, pulmonary and coronary arteries.5 Truncus arteriosus has been described in domestic animals including dogs, cats, horses, calves and lambs.3-5,10 In human medicine truncus arteriosus accounts for <1% of congenital heart defects and can be further classified into different subtypes using two different score systems which can be also applied to veterinary medicine.7 Thus, based on the anatomical location of the associated arteries, truncus arteriosus can be subclassified as I-IV using 1949 Collet and Edwards classification1, or types A1 to A4 according to the 1965 Van Praaghs system.11 The current case showed independent pulmonary arteries originating from the common trunk which are part of type III or type A2 truncus arteriosus subclassifications. Moreover, ventricular septal defects are almost always present, as in this case.5

Congenital heart abnormalities in black rhinoceros are seldom described in the literature with no more than four cases reported including atrial and ventricular septal defects, patent ductus arteriosus and valvular abnormalities with one case also showing cleft palate.6 This subject did not exhibit other congenital abnormalities apart from the cardiac defect. The congenital origin is well known in human medicine and is due to an incomplete separation of the embryonic truncus arteriosus into the two outflow vessels (pulmonary artery and aorta).9 The underlying cause of truncus arteriosus in humans is usually unknown and only limited studies about the inheritance risks are published.8

Hemolytic anemia is an important cause of death in captive rhinoceros, even though the histological examination revealed haemosiderophages in the lungs and liver in this subject, these changes were interpreted as linked to the cardiomyopathy as truncus arteriosus often results in a left-right shunt that leads to pulmonary hypertension.2

To our knowledge this is the first report describing type III truncus arteriosus in an Eastern black rhinoceros (*Diceros bicornis michaeli*). This abnormality, based on human cardiovascular reports, has a likely congenital origin and could potentially have a risk of inheritance in captive populations of Eastern black rhinoceros.

**LITERATURE CITED**

1 Collett RW, Edwards JE. Persistent truncus arteriosus; a classification according to anatomic types. *Surg Clin North Am.* 1949;29(4):1245-1270.

2 Dennis PM, Funk JA, Rajala-Schultz PJ, et al. A review of some of the health issues of captive black rhinoceroses (Diceros bicornis). *J Zoo Wildl Med.* 2007;38(4):509-517.

3 Haist V, von Altrock A, Beineke A. Persistent truncus arteriosus with dissecting aneurysm and subsequent cardiac tamponade in a lamb. *J Vet Diagn Invest.* 2009;21(4):543-546.

4 Jesty SA, Wilkins PA, Palmer JA, Reef VB. Persistent truncus arteriosus in two Standarbred foals. *Equine Vet Educ.* 2007;19(6):307-3011.

5 Kittleson MD, Kienle RD: Small Animal Cardiovascular Medicine.St. Louis, MO, 1998

6 Lewis S, Duncan M, Houck ML, Bloch R, Haefele H. Congenital Cleft Palate and Cardiac Septal Defects in a Neonatal Southern Black Rhinoceros (Diceros Bicornis Minor). *J Zoo Wildl Med.* 2016;47(3):876-878.

7 Marelli AJ, Ionescu-Ittu R, Mackie AS, Guo L, Dendukuri N, Kaouache M. Lifetime prevalence of congenital heart disease in the general population from 2000 to 2010. *Circulation.* 2014;130(9):749-756.

8 Nourzad G, Baghershiroodi M. A case report of truncus arteriosus communis and genetic counseling. *ARYA Atheroscler.* 2013;9(4):254-259.

9 Sadler TW: Langman's Medical Embryology, 12th ed. Lippincott Williams & Wilkins, Wolters Kluwer business, Philadelphia, PA, 2012

10 Schwarzwald C, Gerspach C, Glaus T, Scharf G, Jenni R. Persistent truncus arteriosus and patent foramen ovale in a Simmentaler x Braunvieh calf. *Vet Rec.* 2003;152(11):329-333.

11 Van Praagh R, Van Praagh S. The anatomy of common aorticopulmonary trunk (truncus arteriosus communis) and its embryologic implications. A study of 57 necropsy cases. *Am J Cardiol.* 1965;16(3):406-425.

Figure 1. A) The thoracic cavity showed markedly enlarged and rounded cardiac silhouette. B) Drawing of heart illustrating the anatomical defects observed: from the vena cava (VC) the blood flows to the right ventricle (RV) and subsequently into the common truncus arteriosus (TA). Once in the truncus arteriosus part of the blood goes to the lungs through one right and one left pulmonary artery (RPA and LPA) while the rest of the blood continues flowing to the aorta. From the pulmonary circulation the blood returns to the heart through two right and two left pulmonary veins (RPV and LPV). There is blood exchange between LV and RV through a complete interventricular septal defect (arrows). C) Originating from the right ventricle there is a single truncus arteriosus that gives origin to two pulmonary arteries in each side of the truncus (black arrows). The interventricular septum exhibits a focal defect (white arrow). D) The left ventricle does not communicate with the aorta but is connected to the right ventricle through a complete ventricular septal defect (black arrow). E) Hematoxylin & Eosin (H&E) stain of the left ventricle showing diffuse myocardial degeneration (white arrow) and single cell necrosis (black arrow). F) The pulmonary parenchyma reveals diffuse severe congestion with numerous heart failure cells within the alveolar lumen ( H&E -black arrows) which stain positive with Perls’ Prussian blue stain revealing iron deposits (black arrow - inset).