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DISEASE IN WILDLIFE OR EXOTIC SPECIES

Cardiac Truncus Arteriosus in an Eastern Black Rhinoceros (*Diceros bicornis michaeli*)

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Summary

This case report describes congenital truncus arteriosus in a 1-month-old Eastern black rhinoceros (*Diceros bicornis michaeli*). From the first day of life the animal was underweight and from the 22nd day of life displayed respiratory signs that exacerbated with time leading eventually to collapse and death. Post-mortem examination revealed a single truncus arteriosus originating from the right ventricle leading to two separated pulmonary arteries and the aorta, with the ventricular septum showing a focal communicating defect. Based on the gross examination and current human classifications, the truncus arteriosus was classified as type III or A2. This is the first description of persistent truncus arteriosus in an Eastern black rhinoceros.

Keywords: black rhinoceros; congenital cardiac defect; truncus arteriosus

Congenital cardiac abnormalities in black rhinoceros (*Diceros bicornis michaeli*) are described rarely, with no more than four cases reported including animals with atrial and ventricular septal defects, patent ductus arteriosus and valvular abnormalities, with one case also showing a cleft palate (Lewis *et al.*, 2016). Persistent truncus arteriosus (PTA) is defined as a single great artery, which gives origin to systemic, pulmonary and coronary arteries (Kittleson, 1998) and has been described in domestic animals including dogs, cats, horses, calves and lambs (Kittleson, 1998; Schwarzwald *et al.*, 2003; Jesty *et al.*, 2007; Haist *et al.*, 2009). In human medicine, truncus arteriosus accounts for <1% of congenital heart defects and it is known to have a congenital origin due to an incomplete separation of the embryonic truncus arteriosus into the two outflow vessels (i.e. pulmonary artery and aorta) (Sadler, 2012). However, the underlying cause is usually unknown and few studies about the inheritance risks are published (Nourzad and Baghershiroodi, 2013). Truncus arteriosus can be further classified into different subtypes using two different scoring systems, which can be also applied to animals (Kittleson, 1998). Based on the anatomical location of the associated arteries, truncus arteriosus can be subclassified as types I–IV using the classification of Collett and Edwards (1949) or types A1 to A4 according to Van Praagh and Van Praagh (1965). The current report describes a type III or type A2 truncus arteriosus in a 1-month-old Eastern black rhinoceros.

A 1-month and 3-day old female Eastern black rhinoceros, born in captivity at Chester Zoo (UK), was markedly under the average expected weight for an animal of that age in captivity (personal communication, Chester zoo curator). The calf was born to a 17-year-old multiparous dam in good bodily condition with no evidence of underlying disease. The pregnancy, which was the result of natural breeding,

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had been unremarkable. The birth occurred without complications and the calf stood 1 h after delivery and started sucking 1 h later. Both dam and calf were kept away from the other rhinoceroses. Apart from mild stiffness of the hindlimbs, the calf did not show any clinical signs until the 22nd day after birth, when it was observed to be sneezing. From that day onwards, the sneezing persisted and worsened progressively. Two days before death, the calf was reported to show sudden onset lethargy, laboured breathing and hyperpnoea (60–70 breaths per min) and thermographical imaging revealed cold spots consistent with cooling of the surface temperature, which may indicate development of hypothermia. The calf was separated from the dam for supplementary milk replacement and to perform further physical investigation. The calf did not show any resistance; its rectal temperature was 35.1°C and despite having initial interest in the milk, she did not show a sucking reflex. The respiratory distress intensified and eventually the calf collapsed with no response to resuscitation attempts.

At post-mortem examination, the carcass was in poor bodily condition as indicated by moderately reduced muscle bulk and markedly prominent bony landmarks (especially the dorsal processes of the vertebrae and the ischiatic and iliac tuberosities of the pelvis). The oral mucosa and conjunctiva showed diffuse moderate reddening, interpreted as congestion. The thoracic cavity contained approximately 0.5 l of colourless to red-tinged fluid (hydrothorax). The heart was markedly enlarged with a prominent double apex and rounded cardiac profile, occupying >50% of the thoracic space (Fig. 1).

The pericardial cavity was markedly distended due to an increased amount of fluid (i.e. hydropericardium). Detailed examination of the heart revealed marked anatomical abnormalities (Fig. 2). The base of the heart had a single arterial trunk originating from the right ventricle. Approximately 3 cm from its origin, two independent pulmonary arteries originated from each side of the trunk (left and right), subsequently giving rise to the aorta (Fig. 3). The cranial and caudal vena cavae opened normally into the right atrium and the left atrium communicated with the lungs through four markedly engorged pulmonary veins (two left and two right). The left and right ventricles communicated through a 5 × 4 cm round defect in the upper aspect of the interventricular septum immediately beneath the single arterial trunk. The base of this arterial trunk had a single valve characterized by three irregular and thickened leaflets, from which a narrow fibrous sheet communicated with the mitral valve through the septal defect. Other associated gross changes were diffuse severe

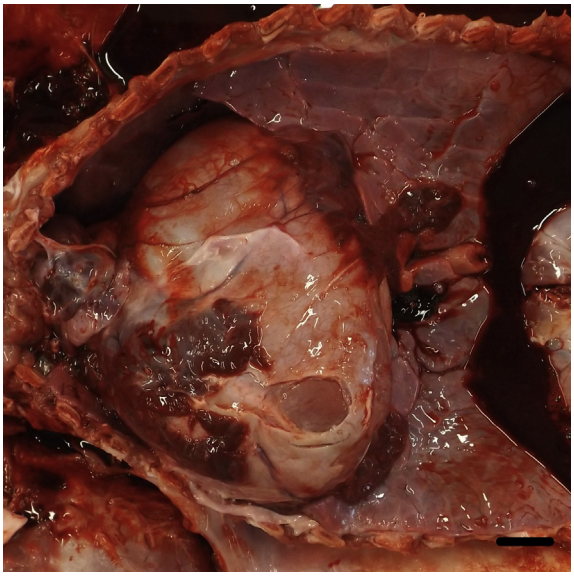

Fig. 1. The thoracic cavity showed a markedly enlarged and rounded heart. Bar, 5 cm.

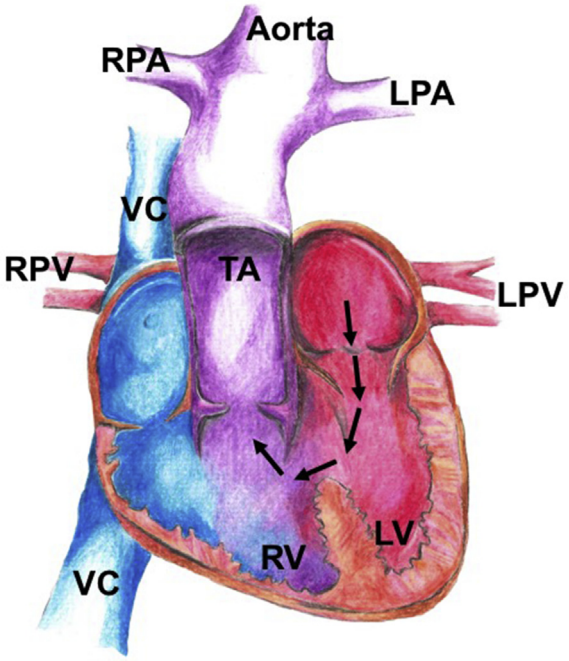

Fig. 2. Drawing of heart illustrating the anatomical defects observed: from the vena cava (VC) the blood flows to the right ventricle (RV) and subsequently into the common truncus arteriosus (TA). Once in the truncus arteriosus, part of the blood goes to the lungs through one right and one left pulmonary 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 the LV and RV through a complete interventricular septal defect (arrows).

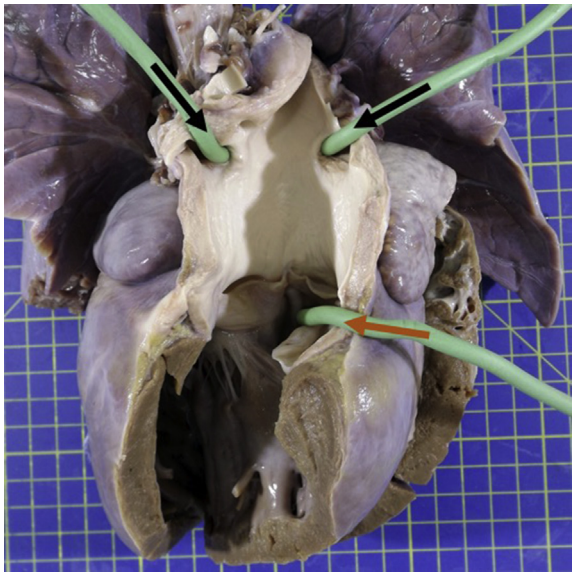

Fig. 3. Originating from the right ventricle is a single truncus arteriosus that gives rise to two pulmonary arteries on each side of the truncus (black arrows). The interventricular septum shows a focal defect (orange arrow). Grid squares, 1 cm.

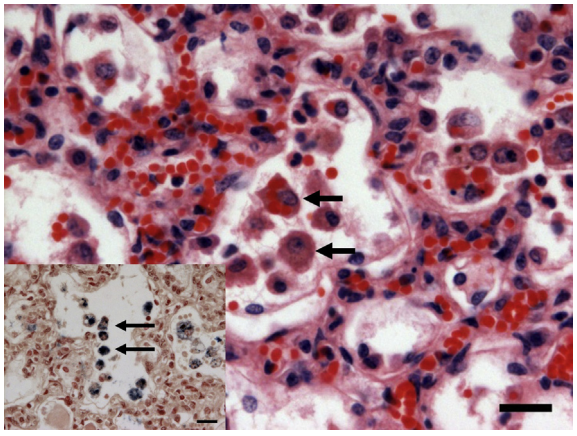

Fig. 4. Section of lung showing diffuse severe congestion with numerous 'heart failure cells' within the alveolar lumen (black arrows). HE. Bar, 20 µm. Inset: these cells stain positively with Perls' Prussian blue stain, revealing iron deposits (black arrows).

pulmonary congestion and atelectasis (caused by compression by the heart) as well as marked enlargement and congestion of the liver, which had moderately rounded edges.

Samples of thoracic and abdominal organs were collected into 10% neutral buffered formalin, processed routinely and embedded in paraffin wax. Sections were stained with haematoxylin and eosin stain (HE). Microscopical examination revealed diffuse moderate myocardiocyte degeneration and multifocal acute necrosis of single myofibres. The lungs showed diffuse severe congestion, with thickening of the alveolar septa due to marked capillary engorgement, intra-alveolar oedema admixed with extravasated erythrocytes and numerous activated alveolar macrophages with intracytoplasmic erythrocytes or golden–brown pigment, confirmed as haemosiderin by Perls' Prussian blue stain (Fig. 4). The liver showed diffuse congestion with abundant presence of haemosiderin in Kupffer cells.

This congenital cardiovascular anomaly is the result of an incomplete separation of the embryonic truncus arteriosus, which gives origin to the proximal ascending aorta and pulmonary artery. Interaction between secondary heart field (SHF) and neural crest cells during early embryogenic gestation regulates the formation of the pulmonary outflows, and therefore, insults to the SHF and/or neural crest cells, may result in PTA (Sadler, 2012). Other congenital defects with similar underlying mechanisms include tetralogy of Fallot, pulmonary stenosis and transposition of great vessels. The morphological appearance of PTA may vary depending on the position of the pulmonary arteries and ascending aorta with respect to the common pulmonary trunk. In the current case, based on gross examination, the presence of independent pulmonary arteries originating from each side of the common trunk was consistent with type III or type A2 truncus arteriosus (Collett and Edwards, 1949; Van Praagh and Van Praagh, 1965). Moreover, the current case also showed a ventricular septal defect, which is almost always present in this type of lesion (Kittleson, 1998). Different to previous reports describing congenital cardiac defects in rhinoceroses, the present case did not show other congenital abnormalities apart from the cardiac defect (Lewis *et al.*, 2016). This abnormality, based on human studies, has a likely congenital origin and could potentially have a risk of inheritance in the relatively small captive population of Eastern black rhinoceroses; however, infectious and toxic causes have also been described as potential risk factors for such abnormalities (Gilbert-Barness and Debich-Spicer, 2004). To our knowledge this is the first report describing type III truncus arteriosus in an Eastern black rhinoceros.

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Conflict of Interest Statement

The authors declare no potential conflicts of interest with respect to the research, authorship and/or publication of this article. None of the authors of this paper have financial or personal relationships with other people or organisations that could inappropriately influence or bias the content of the paper.

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