CONGESTIVE HEART FAILURE SECONDARY TO MULTIPLE CONGENITAL HEART DEFECTS IN A SIX-DAY-OLD BLACK RHINOCEROS (*Diceros bicornis*)

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Abstract

Persistent truncus arteriosus (PTA) is a rare congenital heart defect (CHD) defined by one arterial trunk arising from the ventricular outflow tract which supplies the pulmonic, coronary and systemic circulations. The PTA usually overrides a ventricular septal defect (VSD). A 6-day-old black rhinoceros was diagnosed with PTA, VSD, and atrial septal defect (ASD) which lead to congestive heart failure and death. Persistent truncus arteriosus occurs when the embryonic aorticopulmonary septum fails to divide the truncus arteriosus into an aorta and pulmonary artery. The cause of PTA appears to be multifactorial, although in some breeds of animal and some human families it appears to be due to monogenetic inheritance. The pathophysiology of PTA ultimately leads to pressure and volume overload of the ventricles and congestive heart failure. This report represents the first documented case of PTA in a black rhinoceros.

Resumen

La persistencia del tronco arterial (PTA) es un defecto cardiaco congénito (CHD) poco común definido por un tronco arterial proveniente del tracto ventricular que provisiona a las circulaciones pulmonar, coronaria y sistémica. La PTA usualmente anula un defecto del septo ventricular (VSD). Un rinoceronte negro de seis días fue diagnosticado con PTA, VSD y defecto del septo atrial (ASD) que condujo a una falla cardiaca congestiva y muerte. La persistencia del tronco arterioso ocurre cuando el septo aórtico pulmonar embrionario falla en la división del tronco arterial a la arteria aorta y la arteria pulmonar. La causa de la PTA parece ser multifactorial aunque, en algunas razas de animales y algunas familias humanas, aparentemente es debido a herencia monogenética. La fisiopatología de la PTA a la larga conduce a la sobrecarga de volumen y de presión en los ventrículos y falla cardiaca congestiva. Este reporte representa el primer caso documentado de PTA en rinoceronte negro.

Introduction

Congenital heart defects are not uncommon in animals and man. In man, CHD are the most common form of birth defect.[11] There are several types of CHD including patent ductus arteriosus, atrial and ventricular septal defects, conotruncal abnormalities, valvular abnormalities, and abnormal location of the entire heart (ectopia cordis) which occur at varying rates in animals and man. Clinical signs associated with CHD range from asymptomatic to incompatible with life depending on type and severity of the anomaly.

Persistent truncus arteriosus is a relatively rare CHD and consists of a single arterial trunk arising from the ventricular aspect of the heart. Usually there is an associated ventricular septal defect which allows the trunk to receive blood from both ventricles and supply the coronary, pulmonary, and systemic circulations.[2,3,13,15] Persistent truncus arteriosus was first reported in man in 1798.[18] Since then, PTA has been reported rarely in various species.

In this case, PTA occurred along with VSD, ASD, and hypoplastic pulmonary arteries with secondary right and left ventricular dilation. This combination of congenital cardiac defects has been rarely reported in domestic species and has never been reported in the black rhinoceros.

Case Report

On 18 December 1995, at approximately 1945 hr a male black rhinoceros was born at Caldwell Zoo. The birth, which was monitored via surveillance camera, was uneventful. The calf stood within 2 hr and nursed within 2.5 hr of birth. Initial and subsequent (12 hr post-birth) visual exams of both dam and offspring were within normal limits.

Due to past experience of a rhinoceros birth at the zoo, it was decided to take a "hands off" approach with respect to management of dam and calf. Since repeated visual exams were normal, and calf and dam appeared well-adjusted to each other, initial care provided to the calf was limited to spraying iodine on the umbilical stump two to three times per day. The calf and dam initially did well. The calf was nursing normally, often, and appeared to be gaining weight.

On the afternoon of 23 December 1995, zookeepers noted an increased respiratory rate in the calf. Visual exam revealed a bright, alert and responsive calf who was nursing and following its mother normally. Mucous membranes were noted to be pink. The respiratory rate was 84 breaths per minute and respiratory character was rapid and shallow. Differential diagnoses at that time included: pulmonary hemorrhage secondary to accidental trauma from dam; pneumonia or septicemia secondary to failure of passive transfer; diaphragmatic hernia, congenital or acquired secondary to trauma; and congenital heart or lung disease.

It was decided to monitor the calf overnight and if the condition remained unchanged or worsened, the calf would be separated from its mother in the morning (24 Dec 1995) for physical exam, thoracic radiology, blood work and potential treatment. Zookeepers monitored the dam and calf until 2350 hr and noted nothing unusual. The calf was found dead at 0700 hr 24 December 1995.

Gross necropsy revealed a persistent truncus arteriosus, delineated at its base by three semilunar valves. The PTA was in communication with the right ventricle, but was slightly overriding the high 2.1 x 3.0 cm, oval, VSD. The hypoplastic pulmonary arteries, of equal diameter, branched off of the caudolateral aspect of the truncus 4.0 cm distal to its origin. There was also a multifenestrated, round, 1.4 cm diameter, interatrial septal defect. The left and right ventricular chambers appeared enlarged. The atrioventricular valves and chordae tendinea were unremarkable.

Other lesions included 300 ml of pericardial effusion, a partially foam filled trachea, severe pulmonary edema, and 500-700 ml of peritoneal fluid. Fluid analysis of pericardial and peritoneal fluids were characterized as modified transudates. There were no histopathologic lesions of major

organs. Bacteriology of the above mentioned fluids and lung revealed few contaminant bacteria. All other organ systems were within normal limits. The baby was in good flesh and weighed 47.7 kg (105 lb) at necropsy.

Discussion

Conotruncal abnormalities include a group of CHD in which malformations of the ventricular outflow region occur.[11] Persistent truncus arteriosus, tetralogy of Fallot and subarterial ventricular septal defects are types of conotruncal abnormalities.[11] Persistent truncus arteriosus results from a defect in formation of the aorticopulmonary septum (Fig. 1).[3,7,16,17] The aorticopulmonary septum also participates in the formation of the membranous portion of the ventricular septum, thus it is logical that most PTA occur along with a VSD.[7] The cause of conotruncal abnormalities appears to be multifactorial;[1,2,6,10,11,19] however, there are reports in certain breeds of animals and in some human families of monogenetic inheritance.[4,8,9,11,12]

The consequence or pathophysiology of PTA results in mixing of oxygenated and deoxygenated blood within the atria (if an ASD is present, as in this case) and ventricles (in most cases) (Fig. 2).[5,13,17] Also, the pressures within the ventricles are elevated, especially within the right ventricle, because both ventricles are working against the combined pressures of the systemic and pulmonary circulations.[13] Blood entering the PTA has a relatively low oxygen content and high carbon dioxide content.[13,16] Therefore, the coronary and systemic circulations receive hypo-oxygenated blood. As the animal grows, the increased tissue oxygen demand is not met which sets up a vicious cycle of cardiac compensatory mechanisms, increasing the workload on an abnormal heart. This set of consequences leads to volume and pressure overload of the ventricles and eventually to congestive heart failure.

In conclusion, this is the first report of PTA, a rare form of CHD, in a black rhinoceros. Although there are no published reports of CHD in the black rhinoceros, two cases of full siblings with CHD have been identified (D. Agnew, personal communication). One female died at 10 days of age with a tricuspid valve defect and one male died at roughly 2.5 mo of age with a VSD and *Salmonella* enteritis. Moreover, another full sibling of the above two cases died at 18 days of age with mineralizing cardiomyopathy and mycotic pneumonia; however, these lesions do not appear to be congenital. Therefore, in cases where more than one full sibling is born with a CHD, serious consideration should be given to continued breeding of the parents to each other.

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Figure 1. Schematic diagram demonstrating normal development of the aorticopulmonary or spiral septum (from Latshaw 1987).

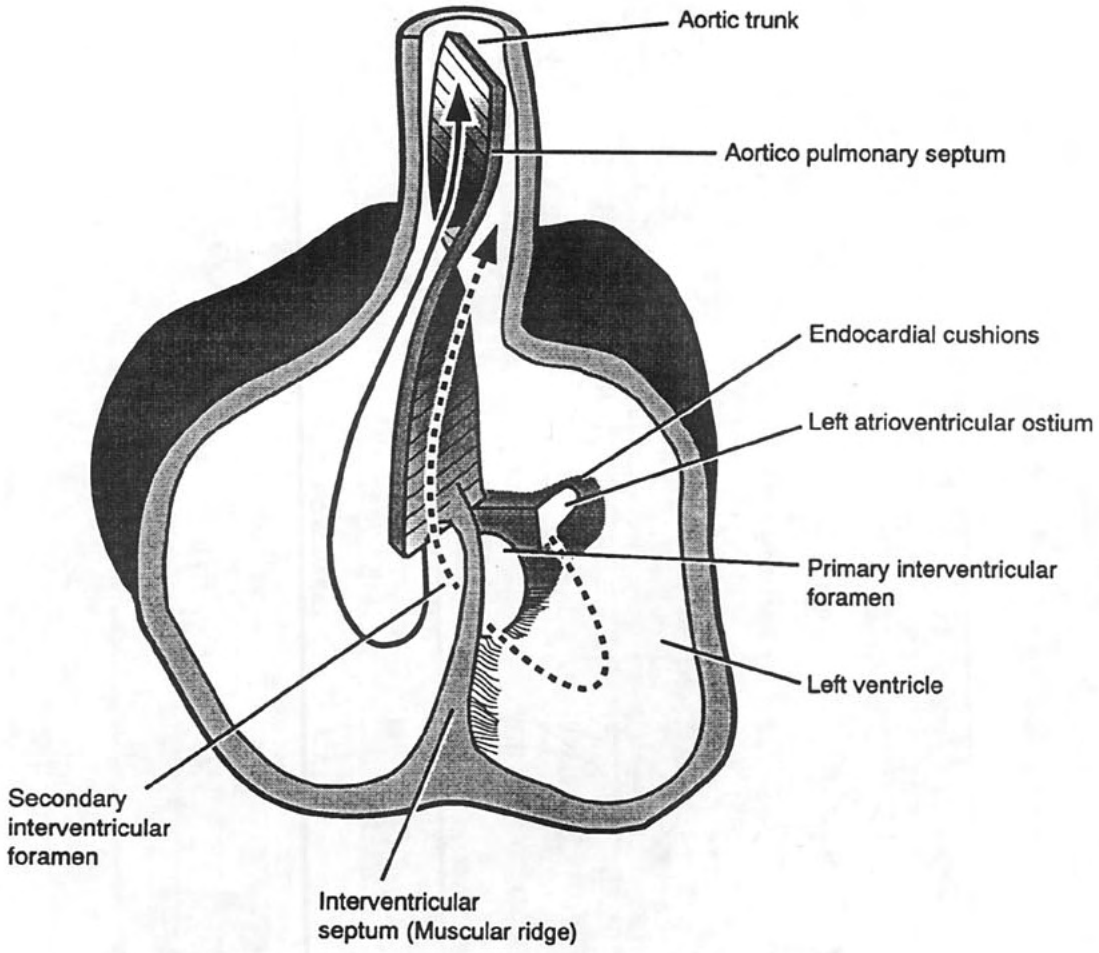

Figure 2. Diagram of the circulatory system in a normal (A) and in persistent truncus arteriosus (B). The luminal shading represents probable relative oxygenation (after Taussig 1960).