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CASE REPORT

Eisenmenger ventricular septal defect in a (Humboldt penguin (Spheniscus humboldti)*



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KEYWORDS

Avian; Congenital heart disease; Cardiac anomalies; Echocardiography Abstract The Eisenmenger ventricular septal defect is an uncommon type of ventricular septal defect characterised in humans by a traditionally perimembranous ventricular septal defect, anterior deviation (cranioventral deviation in small animal patients) of the muscular outlet septum causing malalignment relative to the remainder of the muscular septum, and overriding of the aortic valve. This anomaly is reported infrequently in human patients and was identified in a 45-day-old Humboldt Penguin, *Spheniscus humboldti*, with signs of poor growth and a cardiac murmur. This case report describes the findings in this penguin and summarises the anatomy and classification of this cardiac anomaly. To the authors'

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^{*} A unique aspect of the Journal of Veterinary Cardiology is the emphasis of additional web-based images permitting the detailing of procedures and diagnostics. These images can be viewed (by those readers with subscription access) by going to http://www.sciencedirect.com/science/journal/17602734. The issue to be viewed is clicked and the available PDF and image downloading is available via the Summary Plus link. The supplementary material for a given article appears at the end of the page. Downloading the videos may take several minutes. Readers will require at least Quicktime 7 (available free at http://www.apple.com/quicktime/download/) to enjoy the content. Another means to view the material is to go to http://www.doi.org and enter the doi number unique to this paper which is indicated at the end of the manuscript.

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knowledge this is the first report of an Eisenmenger ventricular septal defect in a veterinary patient.

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Abbreviations

Ao aorta AV aortic valve

EVSD Eisenmenger ventricular septal defect

LV left ventricle
PA pulmonary artery
PV pulmonic valve

RIA right innominate artery

RV right ventricle SB septal band TV tricuspid valve

VSD ventricular septal defect

A 45-day-old female Humboldt penguin chick, *Spheniscus humboldti*, was referred to the University of Pennsylvania Matthew J Ryan Veterinary Hospital cardiology service for evaluation of a cardiac murmur. The penguin had a history of poor growth and weighed approximately 2 kg while healthy chicks of that age typically weigh 3–3.5 kg. A heart murmur was originally noted at 4 weeks of age. The murmur increased in intensity with growth and a palpable thrill was noted at the cardiac apex during a routine examination at 6 weeks of age. No medications were administered prior to evaluation.

At presentation, the penguin was bright and alert. Heart rate, temperature, and respiratory rate were within normal limits. Physical examination revealed a grade IV/VI holosystolic murmur with a point of maximal intensity near the keel. The rhythm was regular. A brief awake transthoracic echocardiogram^d was performed with the penguin in a standing position using a traducer with a frequency of 12 MHz. A large defect of the proximal ventricular septum was suspected, and the aortic valve appeared malaligned, though adequate images were difficult to obtain due to patient movement. A more comprehensive echocardiogram was then performed under general anaesthesia. The penguin was administered 0.048 mg/kg atropine sulphate subcutaneously, induced with isoflurane gas, endotracheally

During the echocardiogram, the penguin experienced prolonged aponea, which was responsive to manual stimulation and temporary discontinuation of the isoflurane gas. The initial anaesthetised heart rate was normal at approximately 200 beats per minute though progressive bradycardia was noted during the echocardiogram with a minimum heart rate of approximately 120 beats per minute. The penguin was not responsive to an additional 0.048 mg/kg atropine administered intramuscularly. Respiratory and cardiac arrest developed but resuscitation was not initiated due to concern regarding long-term prognosis and quality of life.

A complete necropsy was performed. There were no significant non-cardiac findings. The heart weighed 23.5 gm or 1.1% of body weight. Postmortem cardiac evaluation revealed the presence of an 8 mm imes 1.5 mm ventricular septal defect (Fig. 2). The aortic valve partially (<50%) straddled the area of the defect. The right ventricular wall was thickened (4 mm) relative to the left ventricular wall (7 mm; Fig. 3). There was a septal band (trabecula septomarginalis) apparent spanning the right ventricle and separating the ventricular defect from the tricuspid valve (Fig. 2). There was a muscular tricuspid valve present (Fig. 2) as well as bilateral innominate arteries, composed of carotid and subclavian arteries, from the aortic arch (Fig. 3), both of which are normal findings in avian species. The relationship of the ventricular septal defect to the tricuspid valve and aortic valve were apparent from the right ventricle (Fig. 4) and the left ventricle (Fig. 5). The left

intubated, and maintained on isoflurane gas. The anaesthetised echocardiogram confirmed the presence of a large proximal ventricular septal defect suspected to be perimembranous. Velocity of flow across the defect could not be assessed. The aortic valve and pulmonic valve could be viewed parallel to one another in a modified long axis image (Fig. 1). There was a moderate jet of pulmonic insufficiency with a maximum velocity of 2.41 m/sec consistent with mild pulmonary arterial hypertension. There was no echocardiographic evidence of valvular or subvalvular pulmonic stenosis (cine loop available in Supplemental material online).

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