

CASE REPORT



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## Pulmonary atresia with intact ventricular septum and hypoplastic right ventricle in an Arabian foal $\stackrel{\star}{\sim}$

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## **KEYWORDS**

Equine; Congenital heart disease; Cyanotic heart disease; **Abstract** Pulmonary atresia with intact ventricular septum, rudimentary tricuspid valve, hypoplastic right ventricle, and right-to-left atrial shunting were identified in a four-day-old, male Arabian foal with clinical signs of cyanotic heart disease. Pulmonary blood flow was apparently derived from a ductus arteriosus. Echocardiographic evaluation revealed the majority of cardiac abnormalities and also findings compatible with right-sided congestive heart failure. Congenital cardiac

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Patent ductus arteriosus; Pathology defects have a high incidence in this breed, and this is the first description of this combination of congenital cardiac defects. Published by Elsevier B.V.

## Abbreviations

ASD	atrial septal defect
IVS	interventricular septum
LA	left atrium
LV	left ventricle
MV	mitral valve
PA	pulmonary atresia
PDA	patent ductus arteriosus
PFO	patent foramen ovale
RA	right atrium
RV	right ventricle
TV	tricuspid valve

A four-day-old Arabian colt was referred to the University of Minnesota Equine Center for evaluation of lethargy, weakness, and tachycardia. The foal had normal mentation, but was in lateral recumbency and unable to stand or maintain sternal recumbency. An increased respiratory rate (46 breaths per minute) and heart rate (160–220 beats per minute) were noted. The mucous membranes were moist but cyanotic with a capillary refill time of 3 s. Cardiac auscultation revealed a continuous 4/6 left-sided murmur over the cranial heart base. The heart rhythm was regular. Weak arterial pulses and an abnormal pulsation of the jugular vein were noted. Pulmonary auscultation was within normal limits.

Initial laboratory evaluation included complete blood count, serum biochemistry, and arterial blood gas analysis. A decreased white blood cell count of 3.39 103/µL (reference range, × 5.09–11.72  $\times$  103/ $\mu$ L), decreased platelet count of  $101 \times 103/\mu$ L (reference range,  $124-253 \times 103/\mu$ L) and an increased red blood cell count of  $9.69 \times 106/$  $\mu$ L (reference range, 5.90–9.29  $\times$  106/ $\mu$ L) were found. Serum biochemical abnormalities included decreased albumin of 2.8 g/dL (reference range, 2.9–3.9 g/dL), globulin 1.0 g/dL (reference range, 1.9-3.9 g/dL), and total protein of 3.8 g/dL (reference range, 5.9-7.3 g/dL). The arterial blood gas analysis showed a severely decreased PaO2 of 20 mmHg (reference range, 89-115 mmHg), elevated PaCO2 of 51.5 mmHg (reference range, 37–49 mmHg) [1], and a haemoglobin oxygen saturation of 27% (reference range >91%) [2]. The results



**Figure 1** Left apical four chamber view. ASD, atrial septal defect; LA, left atrium; LV, left ventricle; MV, mitral valve; PFO, patent foramen ovale; RA, right atrium.

of the blood gas analysis were suggestive of a rightto-left shunt.

An echocardiogram<sup>d</sup> was performed with the foal in right and left lateral recumbency. Twodimensional M-mode echocardiography and revealed left atrial enlargement [3]. Subjectively, the right atrium was severely enlarged and the interatrial septum was visualised as a thin wall displaced and bowing to the left (Fig. 1, Videos 1 and 2). Right-to-left shunting direction was noted at the level of the interatrial septum by colour flow Doppler. The location of the shunting was compatible with a secundum atrial septal defect (ASD) or a patent foramen ovale. Part of the interatrial septum was mobile and projected into the left atrium. An enlarged left ventricle (LV) with decreased fractional shortening of 22% (reference range 32–45%) [3] and a hypoplastic right ventricle (RV) were found (Figs. 1 and 2; Videos 1 and 2) [3]. The RV was identified echocardiographically by its apical trabeculated morphology. The tricuspid valve was visualized as a rudimentary structure with the leaflets closely tethered to the endocardial surface. A single great vessel originated from the LV, whereas the hypoplastic RV was not connected to the single great artery or to an obvious outflow tract. Neither a pulmonary valve nor a main pulmonary artery could be

<sup>&</sup>lt;sup>d</sup> Vivid 7; M3S 1.5–4 MHz matrix phased-array transducer, GE Healthcare, USA.

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