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Case Report

Partial atrioventricular septal defect in an adult sport horse[☆]



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KEYWORDS

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Cleft mitral valve

Abstract A partial atrioventricular septal defect, represented as a large ostium primum atrial septal defect and common (bridging) atrioventricular valve leaflets with cleft septal leaflet of the mitral valve, was diagnosed incidentally in a nine-year-old warmblood gelding used for show jumping. Initial examination findings and a three-year follow-up are documented in this report. The horse was first presented for the evaluation of chronic coughing. A left-sided, grade 4/6 holosystolic (band-shaped) murmur was identified along with a similar right-sided, grade 3/6 heart murmur. Echocardiography revealed a 6.4 cm diameter communication in the ventral atrial septum, considered an ostium primum atrial septal defect, with bidirectional shunting. A hypertrophic septomarginal trabecula, a thickened tricuspid valve, a cleft septal leaflet of the mitral valve, moderate mitral and tricuspid regurgitation likely related to leaflet prolapse, mild aortic regurgitation, and signs of moderate right ventricular volume overload were found as well. Electrocardiography showed no arrhythmias neither at rest nor during treadmill exercise. The

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owner continued to use the horse for show jumping. No exercise intolerance or other signs of disease were noted. Follow-up examination was performed three years after initial presentation. Contrast echocardiography confirmed the presence of right-to-left shunting through the atrial septal defect. Compared with the initial examination, the left ventricular internal diameter on M-mode echocardiography had increased. Occasional ventricular premature depolarizations were noted on the resting and exercise electrocardiogram. This is the first description of a clinically asymptomatic partial atrioventricular septal defect in an adult sport horse.
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A nine-year-old 560 kg warmblood gelding used for show jumping (obstacle height 115–120 cm) was presented to the Equine Clinic of the University of Veterinary and Pharmaceutical Sciences Brno to investigate occasional coughing. No exercise intolerance was noted by the owner. The horse was bright, alert, and in good body condition. Clinical examination on admission revealed a regular heart rhythm of 44 beats per minute (bpm) and a respiratory rate of 24 breaths per minute. A left-sided, holosystolic (band-shaped) grade 4/6 murmur of blowing quality, and caudo-dorsal radiation was detected. On the right side, a similar 3/6 holosystolic murmur was identified. The remainder of the clinical examination, including lung auscultation, was normal.

Upper airway endoscopy showed mild lymphoid hyperplasia of the pharynx and a small amount of tracheal mucus. High neutrophil counts, but no bacteria, were present on endoscopic tracheal wash cytology. Thoracic ultrasonography showed a small number of comet-tail artifacts bilaterally in the cranioventral lung field.

A transthoracic echocardiogram was performed using a 1.5–3.6 MHz transducer^d to investigate the heart murmur. The right parasternal four-chamber view revealed a 6.4 cm defect between the left

and the right atria (Fig. 1, Video 1), directly adjacent to the atrioventricular (AV) valves; it was therefore classified as an ostium primum atrial septal defect (ASD). Mitral and tricuspid valves showed abnormal insertion and appeared to attach to the dorsal interventricular septum at the same level, as opposed to the normal ventral offset of the septal tricuspid insertion. The short-axis image showed bridging (septal) leaflets and the septal leaflet of the mitral valve had a cleft appearance (Fig. 2, Video 2). Both AV valves showed exaggerated motion and prolapse, with valvular regurgitation. The tricuspid valve leaflets also appeared thickened. The aortic valve was found in an unusual position, slightly more to the right. All these findings were considered typical of a partial atrioventricular septal defect (AVSD).

A thick muscular band was present in the right ventricle between the interventricular septum and the ventral margin of the parietal cusp of the tricuspid valve (Fig. 3, Video 3). Subjective assessment of the right heart showed signs of volume overload. In the right parasternal short-axis view, the interventricular septum showed paradoxical motion and septal flattening (Fig. 3). Echocardiographic measurements are reported in Table 1. Left ventricular posterior wall thickness at end-diastole was higher than reference values for a warmblood of this size [1]. Left atrial sizes obtained from a left and right parasternal view were within the reference ranges [2]. Reference values for the right atrial maximal area are not available in the literature, but measurements were made similar as for the left atrium [3] and clearly showed a larger maximal area than for the left atrium. Color flow Doppler showed moderate mitral and tricuspid regurgitation (Video 4) and mild aortic regurgitation. Pulsed-wave Doppler of the pulmonary flow showed a peak velocity within the upper reference limit [4]. Through the ASD, predominant systolic left-to-right shunting was seen with brief end-diastolic and early-systolic right-to-left shunting. Tricuspid regurgitation

Abbreviations

| | |
|------|--------------------------------------|
| ASD | atrial septal defect |
| AV | atrioventricular |
| AVSD | atrioventricular septal defect |
| bpm | beats per minute |
| VPD | ventricular premature depolarization |

^d Vivid i, General Electric Healthcare Europe GMBH, Diegem, Belgium.

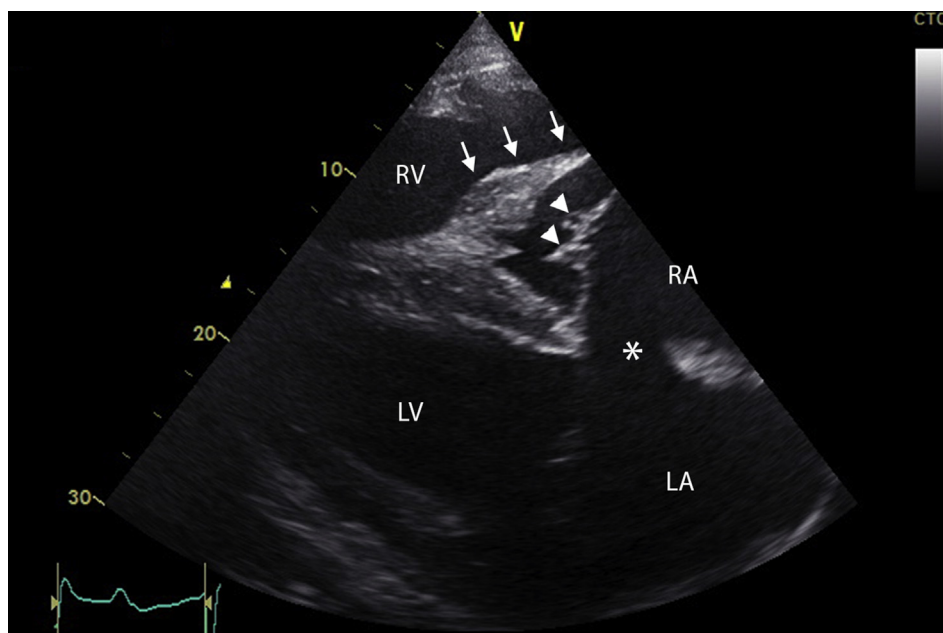


Figure 1 Right parasternal four-chamber view showing the atrial septal defect (*) between right (RA) and left atrium (LA). The septal insertions of the mitral and tricuspid valves are at the same level suggesting a bridging leaflet typical of a partial atrioventricular septal defect. In addition, there is thickening of the tricuspid valve (arrow heads) and a muscular band in the right ventricle (arrows), most likely a hypertrophic septomarginal trabecula. LV, left ventricle; RV, right ventricle.

probably contributed to the right-to-left shunting (Video 4). Continuous-wave Doppler from the right parasternal four-chamber view, dorsal to the tricuspid valve, and through the ASD (Fig. 4) showed a diastolic low-velocity positive flow, which was probably a combination of normal ventricular filling and left-to-right flow through the ASD, and a systolic, higher velocity negative flow (maximal

velocity of 3.2 m/s), which was probably related to mitral and tricuspid regurgitation.

Electrocardiography^e showed sinus rhythm with a heart rate of 44 bpm. The horse was submitted to an incremental standardized exercise test on a high speed treadmill with continuous ECG recording. The warm-up (5 min walk, 5 min trot, 5 min walk)

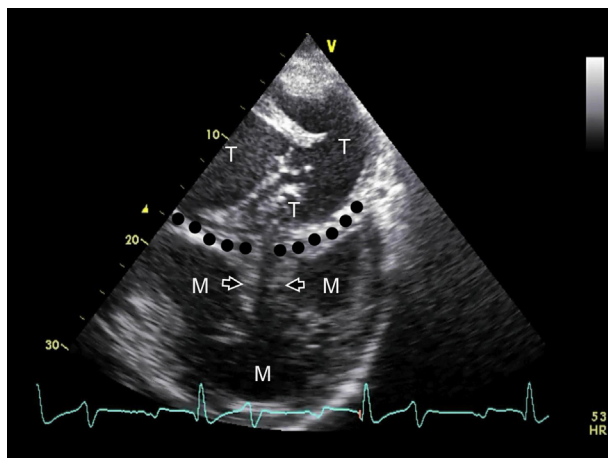


Figure 2 A right parasternal short-axis view shows the mitral (M) and tricuspid (T) valve leaflets at the same level. There are bridging leaflets positioned over the interventricular muscular band (black dots) that give the septal leaflets of the mitral valve a cleft appearance (arrows).

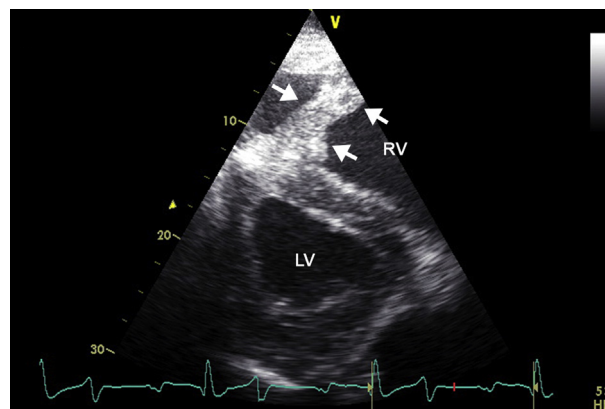


Figure 3 Right parasternal, end-systolic, short-axis view of the right (RV) and left ventricle (LV) showing septal flattening. A thick muscular band (arrows) was present in the RV between the interventricular septum and the ventral margin of the parietal cusp of the tricuspid valve.

^e Televet100 - Telemetric ECG & Holter, Kruuse, Marslev, Germany.

Table 1 M-mode, two-dimensional, continuous- and pulsed-wave Doppler echocardiographic measurements at first presentation (Exam 1) and after three years (Exam 2).

| Measurements | Unit | Exam 1 | Exam2 | Reference ranges | Reference |
|--------------------------|-----------------|--------|-------|------------------|-----------|
| IVSd | cm | 3.5 | 4.0 | 2.4–3.5 | [1] |
| LVIDd | cm | 9.9 | 12.1 | 9.0–13.3 | [1] |
| LVPWd | cm | 3.7 | 3.6 | 1.9–2.9 | [1] |
| IVSs | cm | 4.0 | 4.8 | 3.8–5.2 | [1] |
| LVIDs | cm | 7.2 | 7.9 | 4.9–8.7 | [1] |
| LVPWs | cm | 3.9 | 3.8 | 3.3–4.9 | [1] |
| LV FS | % | 27 | 35 | 24–34 | [25] |
| Aod | cm | 8.7 | 9.2 | 7.1–9.5 | [1] |
| PA | cm | 6.8 | 6.6 | 5.1–7.8 | [1] |
| LA _{LeftPS} | cm | 11.4 | 11.6 | 9.2–13.3 | [1] |
| LA _{RightPS} | cm | 11.2 | NA | 10.5–14.1 | [2] |
| LA _{maxRightPS} | cm ² | 88.6 | NA | 82.1–124.6 | [2] |
| RA _{maxRightPS} | cm ² | 110.4 | NA | NA | NA |
| Vmax PA | m/s | 0.91 | 1.3 | 0.78–1.04 | [4] |

Aod: aortic end-diastolic internal diameter at the level of the sinus of Valsalva; IVSd and IVSs: interventricular septal thickness at end -diastole and in peak systole, respectively; LA_{maxRightPS}: left atrial end-systolic area from the right parasternal four-chamber view (optimized for left atrium); LA_{LeftPS}: left atrial end-systolic internal diameter from the left parasternal long-axis view; LA_{RightPS}: left atrial end-systolic diameter from the right parasternal four-chamber view (optimized for left atrium); LVIDd and LVIDs: left ventricular internal diameter at end-diastole and in peak systole, respectively; LVPWd and LVPWs: left ventricular posterior wall thickness at end-diastole and in peak systole, respectively; LV FS: left ventricular fractional shortening; NA: not available; PA: pulmonary artery internal diameter at end-diastole; RA_{maxRightPS}: right atrial end-systolic area from the right parasternal four-chamber view (optimized for right atrium); Vmax PA: peak velocity of pulmonary artery flow measured using the pulsed-wave Doppler.

was followed by three steps at increasing trotting speeds of 4, 4.5, and 5 m/s. Each speed step lasted for 4 min which included 2 min at 9% inclination followed by 2 min without inclination. One minute of rest was allowed between each step. No cardiac arrhythmias were noticed during exercise.

It was concluded that respiratory signs were caused by mild inflammatory airway disease. Cough resolved with mucolytic treatment and low-dust environment. The partial AVSD was considered clinically silent, and regular cardiac follow-up examinations were recommended together with a reduction in the exercise level. Despite this advice, the owner continued full training and even progressed to a higher jumping class level (obstacle height 125–130 cm). Still no exercise intolerance was noted. Eighteen months after initial presentation, the horse was referred to our clinic for lameness and underwent several uneventful orthopedic surgeries under general anesthesia. The owner declined to perform any cardiac examinations at that time.

Three years after the initial presentation, the horse was admitted to perform a cardiac re-evaluation. At that time, he still did not show any exercise intolerance or other clinical signs.

The body condition had slightly deteriorated compared with that of the previous examination owing to mild muscle atrophy. On clinical

examination, a persistent tachycardia (48–52 bpm) with a regular rhythm at rest was detected. The intensity of the heart murmur had increased with a precordial thrill palpable over the left heart base (grade 5/6). The murmur was blowing in the mitral valve area while buzzing cranial to the left heart base and on the right side (grade 4/6). No other abnormalities were found on clinical examination at rest. Complete blood cell count and plasma biochemistry were normal. Resting cardiac troponin I concentration^f was within reference range (<0.01 ng/mL; ref <0.02). Arterial blood gas analysis^g showed normal PaO₂ at rest (100 mmHg; ref 90–109) and a mildly decreased PaO₂ (69 mmHg; ref >70) [5] immediately after a lunging exercise of moderate intensity (10 min walk, 5 min trot, 5 min canter).

Echocardiography was repeated with similar equipment^h (Table 1). The maximal diameter of the ASD was 6.6 cm. Left ventricular internal diameters were slightly larger but still within the reference range whereas the left ventricular posterior wall thickness at end-diastole remained increased [1]. Other measurements including left

^f Troponin I ultra-sensitive, CLIA, IDEXX Laboratories.

^g Radiometer ABL 800 Flex Analyser, Radiometer Medical ApS, Denmark.

^h Vivid i, GE Medical Systems Israel Ltd., Tirat Carmel, Israel.

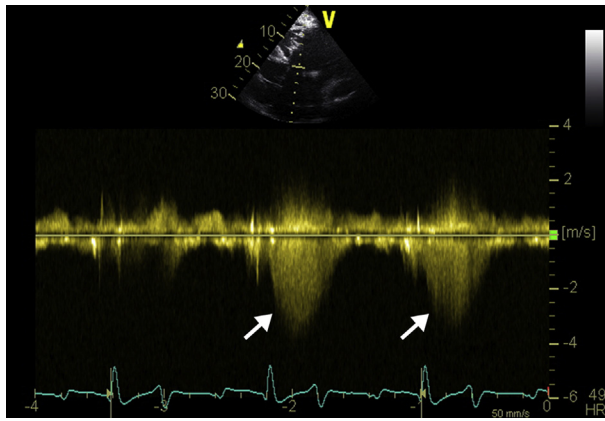


Figure 4 Continuous-wave Doppler recording from the right parasternal four-chamber view. The cursor is placed dorsal to the tricuspid valve and through the atrial septal defect (ASD). The tracing shows a low-velocity positive flow that is most likely due to normal right ventricular filling and possibly left-to-right flow through the ASD. In systole, higher velocity negative flow (arrows) is seen which probably represents mitral and tricuspid regurgitation.

atrial dimensions were within normal limits, except for the pulmonary artery flow which was slightly above reference values [4]. Saline contrast echocardiography confirmed a predominant left-to-right and a small and transient right-to-left shunting through the ASD (Video 5).

The resting ECG^e was recorded for 13 h. The average resting heart rate was 46 bpm. Two ventricular premature depolarizations (VPD) and isolated supraventricular premature depolarizations occurred at rest. The horse was submitted to the same standardized exercise test as three years before. Two VPDs were detected at walk (5 min recording) and one during recovery (2 min recording). After the last follow-up examination, the owner agreed to retire the horse from sport but continued to use it for leisure riding.

Discussion

Atrial septal defects are congenital malformations of the interatrial septum. True atrial septal defects are uncommon in the horse and should not be confused with patent foramen ovale [6]. There are four types of ASD depending on the location of the interatrial communication [7]: (1) ostium secundum ASD – in the region of the fossa ovalis (two thirds of atrial septal defect cases seen in human adults); (2) ostium primum ASD – in the ventral portion of the atrial septum and above the inlet ventricular septum (15% of cases in the human adult population); (3) sinus venosus ASD –

in the upper portion of the atrial septum (10% of cases in the human adult population); and (4) coronary sinus ASD, also called unroofed coronary sinus, and located near the opening of the coronary sinus (rare). In veterinary medicine, the first three types have already been reported [8]. Ostium primum ASD is considered part of the AVSD complex and is ultrasonographically best identified on the right parasternal four-chamber view, also called the inlet view, because an apical view cannot be obtained in adult horses.

Atrioventricular septal defects represent a spectrum of cardiac anomalies resulting from erroneous development of the endocardial cushions. The hallmark feature is a common AV annulus and valve, which has two bridging leaflets that override the ventricular septum. Current classification of the AVSDs includes four main groups [9]: (1) complete AVSD is a combination of ostium primum ASD, non-restrictive inlet ventricular septal defect and one common AV valve orifice; (2) partial AVSD has an isolated atrial (or ventricular) communication, and the common AV valve is divided into two valvular orifices by the attachment of bridging leaflets to the ventricular (or atrial) septum; (3) intermediate/transitional AVSD is a primum ASD with restrictive ventricular septal defect and two valvular orifices; and (4) AVSD with ventricular imbalance describes the situation when one of the ventricles is hypoplastic due to unequal common AV valve commitment.

Congenital cardiac defects in horses represent 3.5% of all congenital abnormalities in horses [10]. In a retrospective study performed on 18 neonatal foals with congenital cardiac defects, an interatrial communication was found in 44% of the cases, but only 11% had a true ASD [11]. Atrial septal defects are most commonly reported as a part of complex congenital abnormalities in foals [11–14]. Complete AVSD has been described in several foals with congestive heart failure [15–18]. To the authors' knowledge, this is the first report of a partial AVSD in a horse. Moreover, this defect was clinically silent in an adult sport horse in full training.

A partial AVSD and several other abnormalities were identified on echocardiography in our patient, but to confirm the full extent of congenital and acquired lesions, a postmortem examination would be necessary. The muscular band found in the right ventricle was probably a hypertrophic septomarginal trabecula with an unusual anatomical position. Hypertrophy of this structure often accompanies congenital defects in humans and can cause some hemodynamic disorders in the right ventricle [19]. Enlargement of the right heart and paradoxical septal movement

were presumably the result of volume overload. Those abnormalities are commonly reported with large ASDs in humans and dogs [7,20,21].

Bidirectional flow through the defect was detected by Doppler and contrast echocardiography. In human patients, a low pressure gradient between left and right atrium can result in bidirectional flow, but most commonly, isolated ASDs result in left-to-right shunting. However, partial AVSD can have significant AV valve regurgitation, and jets of mitral or tricuspid regurgitation can cross the ASD and enter the contralateral atrium. Only a large untreated ASD leading to pulmonary hypertension is likely to produce right-to-left shunting (Eisenmenger's syndrome) accompanied by cyanosis and severe exercise intolerance [20]. In our case, no exercise intolerance was noted by the owner, cyanosis was absent, and arterial partial oxygen pressure was normal. Cardiac catheterization and right heart pressure measurements were unfortunately declined by the owner, but echocardiographic signs of pulmonary hypertension were not present.

Mitral regurgitation caused by a cleft in the anterior leaflet of the mitral valve is almost always present in humans with partial AVSD, and occasionally a cleft in the septal leaflet of the tricuspid valve is the cause of tricuspid regurgitation [22]. Moderate mitral and tricuspid insufficiency was confirmed in our patient, and also a cleft in the mitral valve could be identified (Fig. 2, Videos 2 and 4).

Loud holosystolic murmurs were noticed bilaterally in our patient and must have been the result of tricuspid and mitral regurgitation as an ASD does not produce this type of murmur. During the second exam, the left- and right-sided systolic murmur had increased in intensity. Most likely this represented an increase in AV valve regurgitations, but the difference was difficult to quantify on ultrasound. The increase in AV valve regurgitation could have resulted in more right heart volume overload and subsequently an increase in pulmonary velocity. A typical flow murmur at the left heart base was not detected because of the loud holosystolic murmur.

Some progression was noticed on the echocardiography when comparing both examinations 3 years apart, and this was thought to be due to AV valve regurgitations. It should be noted that each examination was performed by a different clinician which could have contributed to slight differences in the results. However, as the ASD was detected in a nine-year-old sport horse in the absence of clinical signs, progression was expected to be slow.

Human patients with an ASD are susceptible to develop atrial tachycardia or fibrillation. Supraventricular premature depolarizations occurred only on one occasion during the follow-up examination in our case. At that time, several VPDs were noted at rest and exercise. Serum troponin I was normal suggesting that those arrhythmias were not associated with noticeable myocardial damage. However, right-to-left shunting might have contributed to transient myocardial hypoxia and development of ventricular arrhythmias. An increase in tricuspid regurgitation over the three-year period but also an increased right-to-left shunting during exercise might have played a role, but this could not be estimated accurately from the echocardiographic findings.

Most human patients with partial AVSD without surgical repair are symptomatic by early to mid-adulthood and have a poor prognosis, although occasional long-term survival was reported [23]. In the equine species, the prognosis of ASD is usually regarded as unfavorable [6]. Complete AVSD was found only in non-surviving foals, and partial AVSD was not described in a horse [15–18]. Despite a considerably large defect, AV valve regurgitation, and a marked right heart overload, the horse reported here remained twelve years asymptomatic and even performed well as a show jumper. Apart from sport performance, he also underwent several times general anesthesia without any complications.

During the second standardized exercise test, supraventricular and ventricular arrhythmias were found and were of additional concern. It was, however, also not possible to establish if the VPDs during the follow-up were directly related to the AVSD. There is no recommendation for horses with an AVSD in the current literature. Based on the presence of occasional VPDs and taking into account recommendations usually given in cases with other cardiac diseases, this horse should only be ridden or driven with caution by an informed adult [24].

In conclusion, the prognosis of an ASD or AVSD in horses is usually considered poor because it is often associated with additional cardiac abnormalities resulting in congestive heart failure [6,15–18]. However, the case reported here shows that occasionally the prognosis of a partial AVSD can be favorable.

Conflicts of Interest Statement

The authors do not have any conflicts of interest to disclose.

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Supplementary data

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