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Diagnostic imaging of ventricular septal defect in an Iranian Shall lamb

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Abstract

Ventricular septal defect (VSD) is a common congenital anomaly in large animals, but it is rarely documented in small ruminants. This report described a case involving a three-month-old male lamb of the Iranian Shall breed that was presented to a Veterinary Hospital of University of Tehran with nasal discharge and growth retardation. Clinical examination revealed a heart rate of 120 beats *per* min, a respiratory rate of 50 breaths *per* min, pale mucous membranes and a low-grade pansystolic murmur that was auscultated bilaterally in the thoracic region. Echocardiography revealed a 10.00 mm defect between the right and left ventricles, located in the supraventricular region behind the aortic valve, along with evidence of left-to-right shunting. Imaging studies including echocardiography and radiography indicated increased cardiac volume particularly in the right ventricle. These findings were corroborated by necropsy. This case study aimed to elucidate the imaging findings associated with Ventricular septal defect in an Iranian Shall lamb.

Introduction

Ventricular septal defect (VSD) is the most prevalent congenital structural disorder found in domestic animals. It may occur as an isolated defect or in conjunction with various other cardiac anomalies.[1-4] During fetal development, several structures come together to form the ventricular wall and disruptions in this fusion process can lead to the formation of VSD.[4] Ventricular septal defect can be classified by its location and size.[2] This defect commonly occurs in the subaortic region at the pars membranacea resulting in blood flowing from the left ventricle into the right ventricle.[5] This abnormal flow can lead to pulmonary hypertension and enlargement of both the atrium and ventricle. The shunt is expected to reverse when pulmonary pressure exceeds systemic pressure. However, the turbulence of blood flow plays a significant role in causing endocardial damage emphasizing the need for prompt intervention. Such damage increases the risk of bacterial endocarditis.[1,3] The VSD is the most prevalent congenital structural disorder in domestic animals. Additionally, diminished support from the aortic valve may result in aortic insufficiency.[4] Minor defects are sometimes asymptomatic and may be discovered incidentally during a thorough cardiac examination or at abattoir necropsy.[5] Clinical signs in affected animals may include a pansystolic murmur, growth retardation, dyspnea, exercise intolerance and cyanosis.[3,4] In cases of larger defects, a precordial thrill may be palpable on both sides of the chest,[5] which can be life-threatening. Diagnosing a VSD is crucial for determining the future management and use of the animal. Safe techniques, such as echocardiography, are available for this purpose.[4] This method, often used alongside Doppler color flow mapping,

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has become the standard for identifying cardiac defects. While magnetic resonance imaging and computed tomography (CT) can also be employed to diagnose VSD, they are generally less accessible and more expensive than echocardiography.[6] The exact cause of VSD remains unknown. However, several factors have been proposed as potential etiologies including maternal viral infections, metabolic dysfunction, fetal anoxia, and exposure to toxins, nutritional deficiencies during early pregnancy, genetic predisposition, and drug treatments administered to pregnant animals, along with other unidentified factors.[3,5] The VSD occurs sporadically across various sheep breeds,[3] and there are limited published reports on this condition in sheep. This report aimed to present the imaging findings of VSD in an Iranian Shall lamb.

Case Description

In the summer of 2022, a 3-month-old male lamb of the Iranian Shall breed, weighing 10.00 kg, was presented with nasal serous discharge and signs of growth retardation. Clinical examination revealed a heart rate of 120 beats *per* min (sinus tachycardia; Fig. 1), a respiratory rate of 50 breaths *per* min and the presence of pulmonary crackles and hyperpnea. The body temperature was recorded at 38.90 ˚C. Examination of the mucous membranes showed pallor, although the lamb maintained a normal appetite. A low-grade pan systolic murmur (3/6) was detected during auscultation and was audible on both sides of the thorax with the point of maximum intensity located at the third right intercostal space approximately 2.00 – 3.00 cm above the sternum. Palpation of the thorax revealed no thrills on either side. Consequently, the patient was referred for echocardiography and CT scan to further evaluate the identified murmur.

Complete blood count, blood gases analysis and concentration of electrolytes. Blood samples were collected from the jugular vein and ear artery of five healthy lambs aged 3 to 4 months to conduct a complete blood count, blood gas saturation analysis using a Blood Gas Analyzer (OPTI CCA-TS; Opti Medical Systems Inc., Roswell, USA), and to measure electrolyte concentrations. A total of 5.00 mL of blood was taken for these analyses in order to establish reference ranges. In the affected lamb, hematocrit and red blood cell (RBC) count were significantly decreased, measuring 11.00% and 2.75 × 10^6 μL^{-1}, respectively. Additionally, mean corpuscular hemoglobin was increased to 44.70 pg (Table 1). The partial pressure of carbon dioxide was increased markedly to 30.00 mm Hg, while the partial pressure of oxygen was reduced to 55.00 mm Hg and oxygen saturation was dropped to 87.00%. However, serum sodium, potassium and chloride concentrations remained within the normal range (Table 2).

Table 1. Hematological characteristics of the affected lamb.

Parameters	Affected lamb values	Normal values
Hematocrit (%)	11.00	35.80 ± 2.58
RBC (× 10^6 μL^{-1})	2.75	14.12 ± 1.75
Hemoglobin (g dL^{-1})	12.30	11.62 ± 0.85
MCV (fL)	40.70	39.80 ± 1.60
MCH (pg)	44.70	9.08 ± 1.53
WBC (× 10^3 μL^{-1})	12.00	9.77 ± 1.36

RBC: Red blood cell, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, and WBC: White blood cell.

Table 2. Mean values of blood gas analysis, electrolyte levels, and acid-base balance parameters in the affected lamb.

Parameters	Affected lamb values	Normal values
pH	7.55	7.58 ± 0.08
PO_2 (mm Hg)	55.00	71.16 ± 1.19
PCO_2 (mm Hg)	30.00	24.40 ± 1.46
SpO_2 (%)	87.00	93.20 ± 3.10
HCO_3^- act (mmol L^{-1})	22.10	23.02 ± 1.14
Na (mmol L^{-1})	145	148 ± 2.14
K (mmol L^{-1})	4.50	4.68 ± 0.30
Cl (mmol L^{-1})	103	104 ± 2.10

PO_2: Partial pressure of oxygen, PCO_2: Partial pressure of carbon dioxide, and SpO_2: Peripheral oxygen saturation.

Imaging report: echocardiography (GE Vivid 7, General Electric, Boston, USA; multifrequency phased array probe). In the five-chamber long-axis view, a defect measuring approximately 10.00 mm was observed in the supraventricular region, located between the left and right ventricles, posterior to the aortic valve (Fig. 2). This defect was associated with a significant left-to-right shunt, evidenced by a turbulent jet with a maximum velocity of 1.64 m sec^{-1} and a maximum pressure gradient of 10.78 mmHg. Consequently, there was dilation of the right ventricle with the right ventricular outflow tract demonstrating a maximum velocity of 2.04 m sec^{-1} and a maximum pressure gradient of 16.58 mmHg as measured in the short-axis five-chamber view.

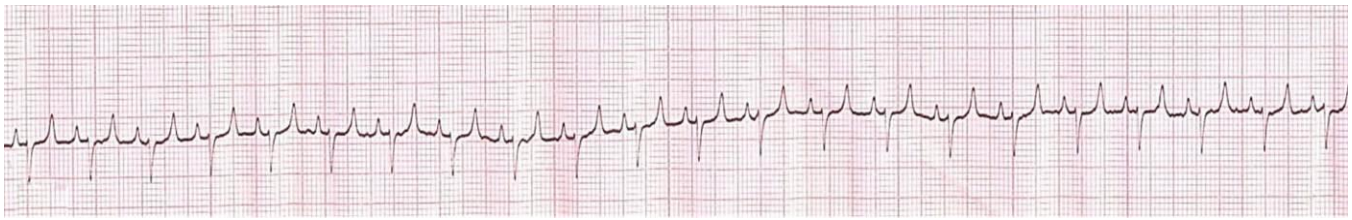

Fig. 1. Electrocardiogram demonstrating sinus tachycardia.

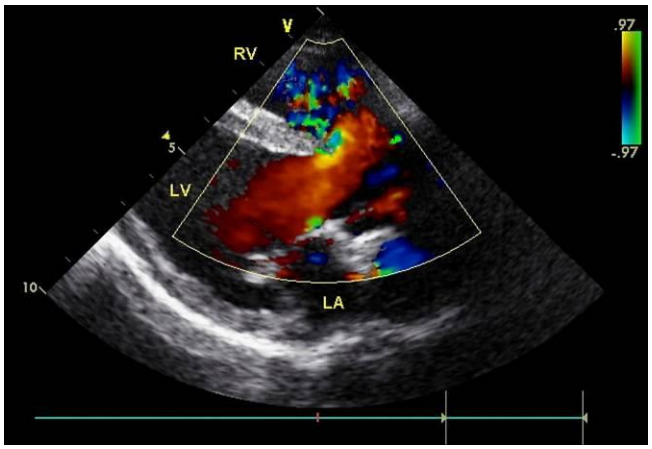

Fig. 2. Right parasternal long-axis five-chamber view illustrating a significant left-to-right shunt, accompanied by severe turbulence at the ventricular defect site. LA: Left atrium, LV: Left ventricle, RV: Right ventricle and V: Ventricle.

The M-mode study revealed that the intraventricular septum and left ventricular posterior wall at end-diastole were at elevated levels of 1.05 and 0.82 mm, respectively. However, both intraventricular septum and left ventricular posterior wall measurements during systole remained within normal limits (1.13 and 1.21 mm, respectively). The left ventricular internal diameter was decreased in both end-diastolic and systolic phases, measuring 3.43 and 2.26 mm, respectively. The fractional shortening was also slightly lower than normal at 34.09%. There were no indications of regurgitation in the aortic or mitral valves. Other measured parameters including the left ventricular outflow tract velocity (1.16 m sec^{-1}), left atrial to aortic ratio (1.23), mitral valve ejection/acceleration (E/A) ratio (0.82), and E-point septal separation (0.23) were all within normal ranges. In a subjective ultrasono-graphic examination using SonoSite MicroMaxx (FUJIFILM Sonosite Inc., Bothell, USA), the liver appeared mildly enlarged with prominent portal vein walls and marked dilation of the hepatic veins. These findings were corroborated by the venous phase of a CT scan (Multi-detector CT scan Somatom Spirit 2; Siemens, Munich, Germany) under reconstruction interval: 0.50 - 1.00 mm, X-ray tube potential: 130 kV, tube current: 120 mA, pitch: 1.00, rotation time: 1 sec, slice thickness: 1.00 mm) following the administration of 800 mg kg^{-1} intravenous contrast (Omnipaque; GE Healthcare, Chicago, USA), delivered via an automatic injector. The CT scan revealed diffuse increased attenuation on the Hounsfield scale in the hepatic parenchyma consistent with congestive hepatomegaly (Fig. 3). In both lateral and ventrodorsal views from radiography and CT angiography of the thoracic cavity, there is currently no quantitative index available to assess heart size in ruminant species. However, subjective signs such as increased sternal contact and an enlarged heart size relative to the intrathoracic distance in the ventrodorsal projection were evident (Fig. 4).

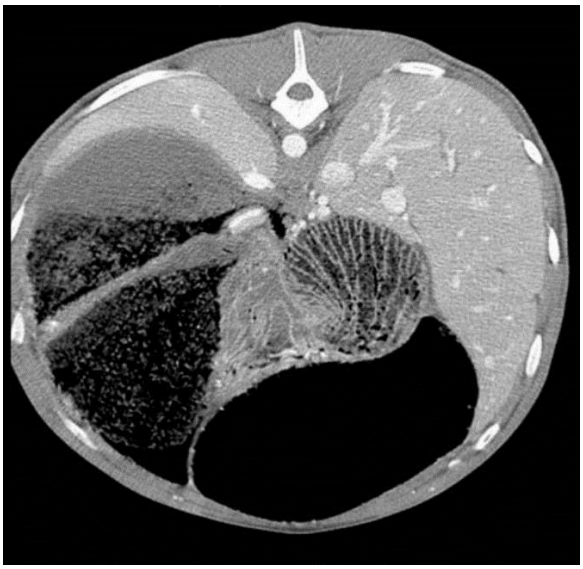

Fig. 3. Transverse computed tomography angiography at the cranial abdominal level reveals markedly increased attenuation of the hepatic parenchyma, accompanied by dilation of the hepatic vasculature.

Additionally, a diffuse, unstructured increase in opacity of the lung lobes and the presence of several peri-bronchial edemas on both sides were noted indicating an alveolo-interstitial lung pattern. This was accompanied by apparent dilation of the pulmonary vessels, particularly the pulmonary arteries, consistent with cardiogenic pulmonary edema (Fig. 5).

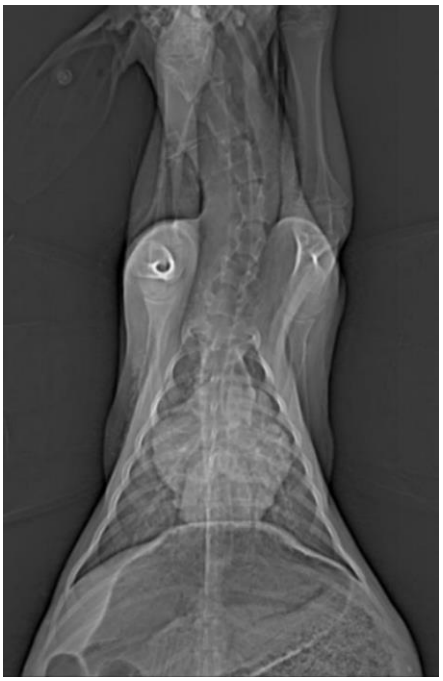

Fig. 4. Ventro-dorsal thoracic radiograph reveals cardio-megaly with an abnormal cardiac silhouette, accompanied by a diffuse increase in pulmonary opacity consistent with cardiogenic edema.

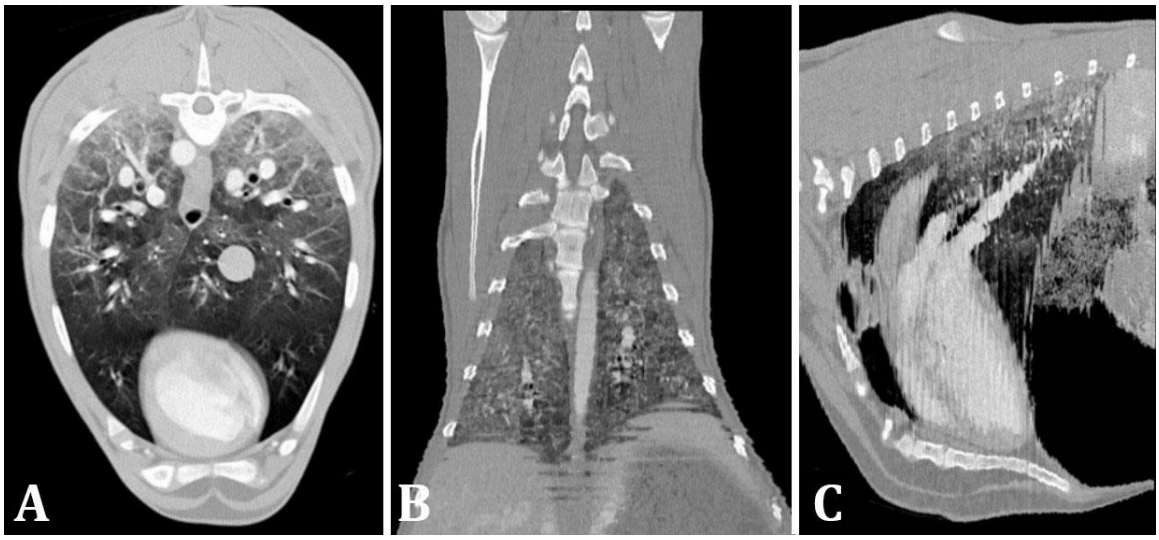

Fig. 5. Computed tomography angiography of the thoracic cavity in**: A)** transverse, **B)** coronal, and **C)** sagittal planes. A prominent mixed alveolar–interstitial lung pattern is evident, consistent with cardiogenic pulmonary edema. Dilation of the main pulmonary vessels is also observed.

Necropsy findings. Due to the observed growth retardation and hyperpnea, the decision was made to perform humane euthanasia followed by necropsy. During the necropsy, a 10.00 mm defect was identified between the left and right ventricles, located posterior to the aortic valve (Fig. 6), which confirmed the findings from the echocardiogram. The edges of the liver were slightly rounded, although its color appeared normal.

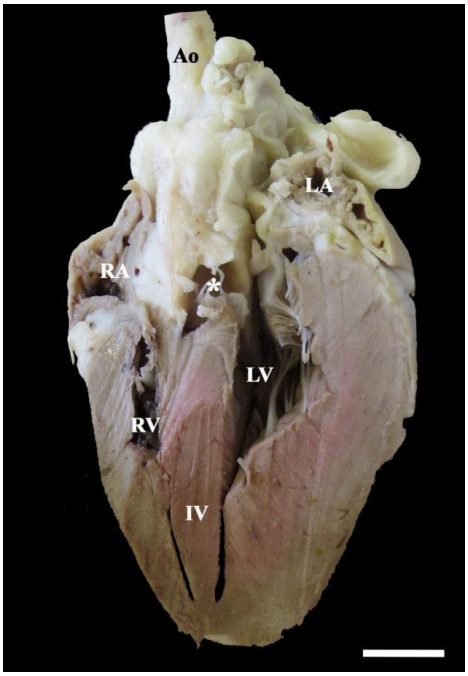

Fig. 6. Anatomical regions of the heart affected by ventricular septal defect (VSD) in a lamb. Ao: Aorta; IV: Interventricular septum; RA: Right atrium; RV: Right ventricle; LA: Left atrium; LV: Left ventricle. The asterisk marks the site of the septal defect, (bar = 2.00 cm).

The lungs appeared macroscopically normal with only a small amount of exudate found in the airways, which likely accounted for the abnormal pulmonary sounds detected during auscultation. The aortic, pulmonary and atrio-ventricular valves were all normal. Other inspected organs were also macroscopically normal showing no anomalies.

Discussion

Ventricular septal defect is a rare cardiac anomaly observed in sheep with its incidence reported in a limited number of cases.[3] This condition manifests due to abnormal development that can affect one, two or all three components of the embryonic heart which includes: 1) the conotruncal ridges, 2) the muscular portion of the septum and 3) the membranous part of the endocardial cushions.[7]

The etiology of VSD, like many congenital heart defects, can be multifactorial. An increase in congenital diseases within a herd may stem from a variety of factors including repeated breeding practices with the same individuals, environmental influences and or a hereditary genetic predisposition.[4] While hereditary and genetic predispositions have been documented in certain bovine breeds,[1] research regarding comparable factors in sheep remains limited.

In the specific case involving the index lamb, it is noteworthy that the herd showed no prior history of congenital disorders, raising questions about the underlying cause of the VSD in this instance. It is critical to further investigate whether environmental stressors or genetic factors could have played a role in the development of this condition especially in light of the lack of documented familial trends. More comprehensive studies are needed to elucidate the potential genetic and environmental contributions to VSD in sheep and to better

understand the overall impact of breeding practices on the health of lambs within affected herds. This knowledge could prove crucial for managing breeding programs and enhancing the health outcomes for future generations.

Ventricular septal defect causes a left-to-right shunt with clinical manifestations that vary depending on the size of the defect. The affected animal may die immediately after birth, exhibit fatal symptoms later in life, particularly after exertion, or the defect may only be diagnosed post-mortem.[3] In some instances, individuals can remain asymptomatic for an extended period. However, the most common associated complication is chronic non-responsive pneumonia which can be identified through clinical evaluation and necropsy.[1]

In the case of the index lamb, clinical signs such as an increased respiratory rate, hyperpnea and pulmonary crackles were observed. These signs occur when blood flow between the two ventricles is doubled resulting in regurgitation of blood into the lungs which subsequently leads to pulmonary hypertension and edema.[1] This process describes the pathway to heart failure. Similar clinical signs have been reported by Peretti *et al.* in a 1-month-old Ile de France lamb with VSD.[3]

Inadequate cardiac output fails to deliver sufficient blood to the tissues (hypoperfusion) resulting in systemic hypoxia and degenerative damage.[3] A decrease in partial pressure of oxygen in the affected lamb can lead to a state of metabolic acidosis. However, this condition does not significantly alter blood pH or HCO_3 levels. Hematocrit and (RBC counts are notably reduced. The shunt formation in VSD can cause RBC damage and hemolysis[8] leading to anemia, pale mucous membranes and stunted growth in the affected lamb. It appears that an increased mean corpuscular hemoglobin may act as a compensatory mechanism allowing each RBC to carry more oxygen.

Echocardiography is the preferred technique for rapid and non-invasive assessment of congenital cardiac diseases. It allows for evaluation of cardiac shape and size, valvular function and precise localization of defects.[4] Echocardiography is instrumental in assessing the relationship between defect size and prognosis.[1] It identified a supraventricular defect located behind the aortic valve accompanied by a left-to-right shunt which was later corroborated during necropsy. As lambs mature, they typically exhibit myocardial hypertrophy and an increase in left ventricular volume as an adaptation for efficient blood circulation.[9] However, in this particular lamb, echocardiographic findings revealed right ventricular dilation. Yadegari *et al.* documented a VSD in a male calf displaying poor weight gain, hyperpnea and pale mucous membranes with the defect located in the ventricular septum below the aortic valve.[4] Furthermore, Buczinski *et al.* reported cardiomegaly secondary to VSD in two heifers, identified through thoracic radiographs.[1] While radiography has limitations in sensitivity for diagnosing cardiovascular issues,[1] it confirmed cardiomegaly in the affected lamb. The prognosis for animals with VSD is generally poor and euthanasia is frequently elected in such cases.[4] Consequently, the precise location of the defect is usually determined through necropsy. Peretti *et al.* also described ascites, increased liver volume, moderate endocarditis and pulmonary edema in the affected lamb,[3] although in our case, only small amounts of exudate were observed in the respiratory airways. Given these considerations, a thorough cardiac examination is essential for animals experiencing growth retardation. However, general practitioners may overlook murmurs during initial physical examinations, particularly if they are of low intensity.

Conflict of interest

The authors declare that there are no conflicts of interest in disclosing this work.

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