Atrial septum defect in a pygmy goat: a case report

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ABSTRACT

Congenital heart defects represent developmental anomalies that cause significant anatomical and functional alterations in the heart's structure present at birth. Among these defects, atrial septal defect (ASD) is characterized by an abnormal communication between the right and left atria, typically caused by persistence of the foramen ovale or by an incompletely developed interatrial septum. This structural defect results in a moderate volume overload on the right side of the heart, leading to increased pressure and dilation of the right atrium and ventricle. A 3-day-old Pygmy goat neonate weighing 1.5 kg was admitted to the Large Animal Veterinary Hospital of the Federal University of Lavras (UFLA) presenting with marked apathy, absence of the sucking reflex, permanent lateral recumbency, pale mucous membranes, tachycardia with irregular rhythm, and tachypnea, and a soft systolic murmur most evidente over the right cardiac apex, indicating severe systemic and cardiovascular compromise. Supportive treatment was promptly initiated, including parenteral fluid therapy with isotonic Ringer's lactate solution combined with 50% glucose to address dehydration and hypoglycemia, as well as enteral nutrition administered via a nasogastric tube to ensure adequate caloric intake. Despite intensive medical care and supportive measures, the neonate showed rapid clinical worsening and died seven hours after the onset of treatment. Necropsy examination revealed a globular and enlarged heart, with marked dilation of the right ventricle, presence of a large intracardiac thrombus obstructing the right ventricular outflow tract, and a wide communication between the atria through a persistent foramen ovale (a form of ostium secundum-type ASD). Based on the clinical history, physical examination findings, and necropsy results, a definitive diagnosis of congestive heart failure secondary to ostium secundum atrial septal defect was established. Considering that treatment of congenital cardiac diseases in small ruminants is often economically unfeasible, and given the hereditary nature of this condition, culling of affected animals from the breeding population is generally recommended. The ostium secundum-type ASD in neonate pygmy goats may progress rapidly to congestive heart failure, particularly when complicated by intracardiac thrombosis, and underscores that, despite prompt clinical intervention, necropsy remains essential for definitive diagnosis and for guiding breeding decisions aimed at preventing hereditary cardiac defects.

PAROLE CHIAVE

Cardiomyopathy; Congenital; Congestive heart disease.

INTRODUCTION

Congenital heart defects are developmental anomalies present at birth and compatible with intrauterine life, which lead to anatomical and/or functional alterations in the heart. Although they are uncommon in domestic animals, such conditions have been reported in various species, with the highest prevalence observed in cattle, followed by sheep and goats [8, 3].

Among these defects, Atrial Septal Defect (ASD) refers to the persistence of an opening in the interatrial septum, usually related to the patency of the foramen ovale or incomplete fusion of the septal components. Once present, this communication allows left-to-right shunting due to the atrial pressure gradient, increasing blood flow to the right atrium and right ventricle, potentially leading to volume overload and cardiac remodeling over time [3].

Although many animals with ASD remain asymptomatic, clin-

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ical signs may arise when the shunt is large enough to induce significant hemodynamic changes. Moderate right atrial and ventricular hypertrophy may occur, and in severe cases, reversal of the shunt (right-to-left) may develop, leading to systemic hypoxia and cyanosis [11]. ASD is classified into four types based on its anatomical location: primum ASD, located in the lower part of the septum; secundum ASD, in the region of the fossa ovalis; sinus venosus ASD, near the cranial vena cava; and coronary sinus ASD, involving the wall between the coronary sinus and the left atrium [2, 5].

Although congenital cardiac anomalies are not frequently diagnosed in any particular species, it is likely that many cases go unnoticed due to the absence of clinical signs or limitations in routine diagnostic procedures [3,8, 10].

Although isolated reports of ASD in pygmy goats exist [4], breed predisposition has not yet been well established, as larger case series and epidemiological data [6] have not identified a clear overrepresentation of pygmy goats in congenital cardiac defects. Given the rarity of this condition in goats and the limited literature describing its clinical progression in neonates, this report provides valuable documentation of a congenital cardiac anomaly in a small ruminant. By detailing the clinical presentation, supportive management, and postmortem findings, this case contributes to improving early recognition and understanding of congenital heart defects in neonatal goats, thereby supporting more accurate diagnosis and informed decision-making in breeding and herd health management.

CASE REPORT

A three-day-old Pygmy goat neonate, weighing 1.5 kg, was presented to the Large Animal Veterinary Hospital of the Federal University of Lavras (UFLA) with a history of apathy, exercise intolerance, progressive weakness, and loss of suckling reflex. The parturition was eutocic, and the kid stood up and nursed colostrum normally, only exhibiting the previously described clinical signs at the end of the first day of life. At admission, the animal was in lateral recumbency and reluctant to stand. On physical examination, pale ocular mucous membranes were observed (Figure 1), along with tachycardia (88 beats per minute), cardiac arrhythmia, tachypnea (152 breaths per minute), and a rectal temperature of 37.1°C, as well as a soft systolic cardiac murmur best auscultated over the right cardiac apex. Due to the rapid clinical deterioration and the animal's unstable condition, complementary diagnostic tests (primarily echocardiography or thoracic radiography) could not be performed, and the definitive diagnosis was therefore obtained only at necropsy. Based on the clinical presentation, other potential differential diagnoses could have included congenital cardiac defects such as ventricular septal defect (VSD) [6] or early-onset congestive heart failure [14], as well as non-cardiac causes like nutricional myopathy (e.g., selenium/vitamin E deficiency), particularly in weak, apathic neonates [13].

Figure 1 - Three-day-old Pygmy goat neonate exhibiting pale ocular mucosa at the time of carcass inspection during necropsy.

Laboratory findings

Complete blood count revealed anisocytosis and polychromasia of red blood cells (RBC: 6.55 x 10 /mm³; Ht: 29.8%; Hb: 10.2 g/dL), the presence of metarubricytes (13), Howell-Jolly bodies (+), and basophilic stippling. White blood cell count indicated relative neutrophilia, relative and absolute eosinopenia, reactive lymphocytes, relative and absolute monocytosis, thrombocytosis, platelet anisocytosis, and stress platelets (Table 1).

Treatment and clinical progression

Supportive therapy included parenteral fluid therapy with lactated Ringer's solution (90 mL/kg/h), because it is an isotonic solution containing Na, Cl, K, and Ca, in addition to lactate, which is advantageous in cases where the animal presents metabolic acidosis-an expected condition, as it is the most common scenario in dehydration, continuous intravenous infusion of 50% glucose (1 g/kg) to provide energy to the animal, which had no suckling reflex and had been anorexic for more than 24 hours, and feeding via nasogastric tube every hour goat with milk (20 mL/hour). The animal died seven hours after the beginning of treatment.

Necropsy and histopathological findings

The animal died approximately seven hours after admission and was immediately submitted for post-mortem examination. The overall condition of the carcass showed no cadaveric changes that would indicate any abnormal physiological state. Gross necropsy revealed a markedly enlarged, globose heart (Figure 2A), with significant dilation of the right ventricle reaching the apex, containing a large intracardiac clot, and a wide interatrial communication due to a persistent foramen ovale (Figure 2B). The heart, organs, and carcass weight were not measured at the time of necropsy. Photographs included in this report were taken during necropsy and are original images obtained by the authors. In addition, the animal presented macroscopic alterations related to the cardiac abnormality, such as congestion in the lungs, liver, kidneys, adrenal glands, and brain, with engorged vessels. Histopathology of the lungs revealed marked congestion, diffuse edema, and multifocal atelectasis. No significant macroscopic or microscopic lesions were observed in the other organs. These findings are consistent with an interatrial septal defect leading to congestive heart failure.

DISCUSSION

Atrial septal defect (ASD) is a rare congenital heart condition in goats, with few reports in the literature. However, there are descriptions of ASD in association with other cardiac anomalies such as Ebstein's anomaly and subaortic stenosis, especially in Pygmy goats [8, 4]. The goat in this report was also of the

Table 1 - Hematological parameters of the patient compared with the established reference values for the caprine species.

Parameters	Unit	Observed Value	Reference Value
Red blood cells (RBC)	$10^6/mm^3$	6,55	8-18
Hemoglobin concentration	g/dL	10,2	8-12
Hematocrit (HCT)	%	29,8	38.29 (mean)
Mean corpuscular volume (MCV)	fL	-	16-25
Mean corpuscular hemoglobin (MCH)	pg	-	5.2-8
Mean corpuscular hemoglobin concentration (MCHC)	g/dL	-	30-36
Total leukocyte count	μL	7.900	4.000-13.000
Segmented neutrophils	%		30-48
Lymphocytes	%	18	50-70
Monocytes	%	-	0-4
Eosinophils	%	-	1-8
Basophils	%	-	0-1
Total platelet count	$\times10^3/\mu L$	817.000	300-600

Pygmy breed, reinforcing a possible breed predisposition. Although the etiology of congenital heart defects can include maternal infections, metabolic disorders, toxins, and nutritional imbalances [1], the dam of the affected kid showed no clinical signs or known risk factors during gestation, suggesting a possible sporadic mutation or undetected inbreeding.

Among the causes of congenital cardiac diseases are maternal viral infections, which may result in fetal infection or metabolic dysfunction; fetal anoxia secondary to placental disorders; the use of pharmaceuticals in pregnant females; nutritional deficiencies or excesses; exposure to toxins and physical trauma, as well as heredity [1, 11].

While congenital heart defects in goats can occur sporadically, some authors have discussed the potential hereditary component in other species [10]. Although no history of consanguinity or cardiac abnormalities in the herd was reported, the lack of breeding records limits the ability to rule out genetic involvement.

In the case described by Laus et al [8], a 2-month-old Pygmy goat presented multiple simultaneous anomalies (including Ebstein's anomaly, mitral valve dysplasia, atrial septal defect, and subaortic stenosis), resulting in early signs of cardiac failure and a rapidly fatal outcome, even with therapeutic intervention. In contrast, in a case reported by Gardner et al [4], an adult Pygmy buck exhibited a tricuspid valve anomaly associated with an atrial septal defect-lesions of lesser extent that allowed for a chronic clinical course and prolonged survival. When compared with the present case, it becomes evident that even isolated anomalies may exhibit a more severe clinical behavior than previously described complex malformations, particularly in neonates with low hemodynamic reserve and high vulnerability to the consequences of interatrial septal abnormalities.

Among the most common congenital cardiac anomalies in small ruminants are ventricular septal defect, atrial hypoplasia,

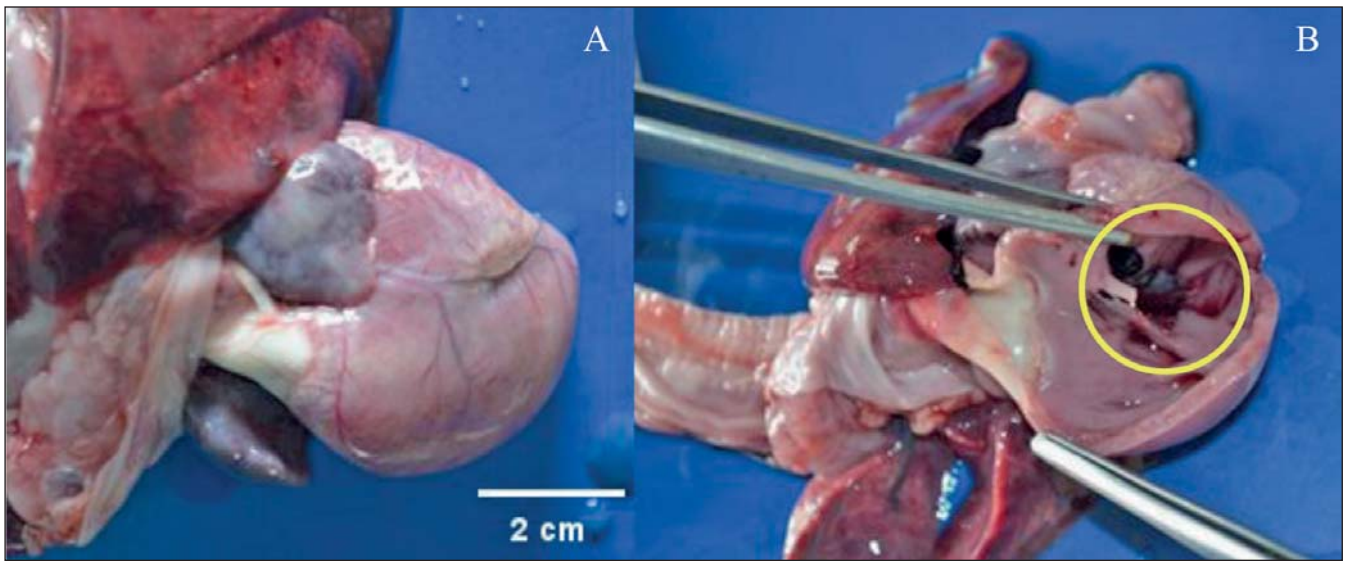

Figure 2 - Necropsy of a three-day-old Pygmy goat neonate - cardiorespiratory system. A. Enlarged heart, particularly the right side, with a globose aspect; the lungs appear congested. B. Wide communication between the atria through a persistent foramen ovale (yellow circle), observed after opening the dilated right ventricle with a thin cardiac wall; congested lungs are also evident.

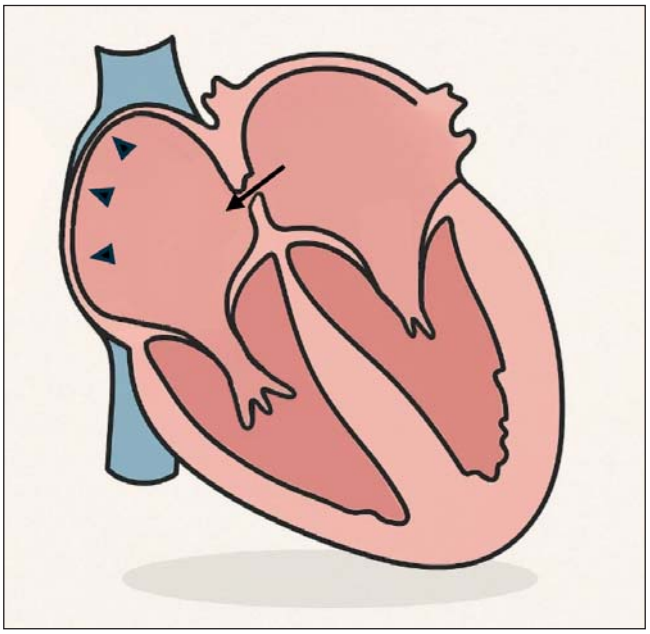

Figure 3 - Schematic representation of the heart from the animal in the present study, highlighting interatrial communication (arrow) and right ventricular dilation (head of arrow). Illustration created by the authors of this study.

cardiomegaly, patent ductus arteriosus, ASD, and tetralogy of Fallot [8]. Notably, the significance of ASD often increases when associated with other anomalies [3]. However, in this case, ASD was the sole defect and was sufficient to cause congestive heart failure, as confirmed at necropsy (Figure 3).

The pathophysiology of congestive heart failure involves increased end-diastolic pressure in the left ventricle, elevated left atrial and pulmonary venous pressures, leading to pulmonary edema and respiratory compromise [3]. In this report, severe pulmonary congestion was confirmed post-mortem, consistent with the literature [8, 11].

Clinical signs of ASD depend on the severity of the defect. While mild cases can remain subclinical, moderate to severe defects result in signs such as exercise intolerance, tachypnea, anorexia, cyanosis, and lethargy [11]. In the reported goat, signs included tachycardia, cardiac arrhythmia, tachypnea, pale mucous membranes, and exercise intolerance-indicating significant cardiovascular compromise. These findings support heart failure as a cause of tachycardia [9], while mucosal pallor suggests poor perfusion.

The presence of a systolic murmur, as observed, is consistent with ASD but is generally attributed not to the atrial shunt itself, but to secondary pulmonary flow turbulence due to right ventricular volume overload [11, 9]. The absence of cyanosis, despite significant cardiac compromise, is likely due to the predominance of a left-to-right shunt, through which the blood reaching the systemic circulation remains relatively oxygenated. In addition, the neonate exhibited minimal physical activity, without physiological exertion sufficient to increase right atrial pressure, reducing the likelihood of shunt reversal and consequent systemic hypoxia [3].

Although the goat died on the third day of life, postnatal closure of the foramen ovale can occur gradually through tissue fusion [7]. However, in this case, a large and persistent opening allowed for significant left-to-right shunting and right-sided volume overload.

The defect was identified as a secundum-type ASD, the most common type in cattle [9], which favors left-to-right shunting due to the greater compliance and thinner wall of the right ventricle. This hemodynamic pattern was confirmed in the case, and the absence of cyanosis is consistent with predominant left-to-right flow.

While echocardiography, the gold standard for diagnosing congenital cardiac anomalies, and thoracic radiography are valuable diagnostic tools [3], they were not performed in this case due to the animal's rapid clinical deterioration and early death. Echocardiography could have demonstrated dilation of the right atrium and ventricle, interatrial shunting, and septal abnormalities, allowing detailed evaluation of the structural and functional impact of the defect. Thoracic radiographs, which can reveal cardiomegaly and vascular changes, might also have indicated hemodynamic overload; however, in this neonate, significant pulmonary artery enlargement would likely not have been present given the rapid progression of the disease. In this case, the definitive diagnosis was made only at necropsy.

Gross pathological examination revealed a globose heart with significant dilation of the right ventricle and atrial communication via the foramen ovale. These findings differ from those previously described in goats, where the heart was characterized as conical and laterally compressed [12]. The altered morphology observed may be explained by a left-to-right shunt leading to volume overload on the right side of the heart, as suggested in earlier studies [8]. Importantly, this volume overload and consequent right ventricular dilation may have played a key role in the formation of the intracardiac thrombus identified at necropsy. The combination of turbulent blood flow across the atrial septal defect, endothelial stress associated with chamber distension, and potential blood flow stasis within the markedly dilated right ventricle creates a pathophysiological milieu conducive to thrombogenesis.

The findings in this report expand current understanding of isolated ASD in goats by confirming that, even without concurrent anomalies, this defect can result in rapid-onset congestive heart failure and death. Further investigation into the genetic and epidemiological patterns in small ruminants, particularly in Pygmy goats, is warranted to clarify potential breed predispositions and improve early diagnosis in clinical practice. Additionally, congenital cardiac anomalies should always be considered in the differential diagnosis of weak or unthrifty neonates, emphasizing the importance of early recognition and clinical vigilance.

CONCLUSIONS

Atrial Septal Defect (ASD) is a rare congenital change that can cause serious circulatory problems and change the anatomy of the heart. Despite the low incidence, there is a need for more studies on ASD and its clinical implications, treatment and prognosis, so that we can increase scientific knowledge and explore other therapeutic and diagnostic approaches in similar cases.

Ethical statement

This study was not submitted for ethics committee review because it is a analysis of medical record of the Large Animal Internal Medicine Service, Veterinary Hospital, Federal University of Lavras - UFLA.

Acknowledgements
To veterinarian for forwarding the studied animal. To the residents for their assistance in treatament and animal care. To the Ministry of Education (MEC) for the scholarship granted to residents.

Author Contributions
All authors contributed to the study conception and design. Methodology and formal analysis and investigation were performed by Laís Maria Viana, Júlia Marília Silva Nascimento, Eligiane Priscila Meurer, Vitória Ferreira Vieira, Franciele de Sá Alves, Marina Resgala Neves and Fábia Fernanda Cardoso de Barros da Conceição. The first draft of the manuscript was written by Laís Maria Viana, Júlia Marília Silva Nascimento, Eligiane Priscila Meurer and Vitória Ferreira Vieira. The review and editing of the manuscript was written by Laís Maria Viana, Júlia Marília Silva Nascimento, Eligiane Priscila Meurer, Vitória Ferreira Vieira, Claudia Dias Monteiro-Toma, Geison Morel Nogueira, Angelica Terezinha Barth Wouters, Adriana de Souza Coutinho, Hugo Shisei Toma. The supervision was performed by Angelica Terezinha Barth Wouters, Adriana de Souza Coutinho, Hugo Shisei Toma. All authors have read and agreed to the published version of the manuscript.

Conflict of interest statement
The authors declare that there are no conflicts of interest.

Funding
This research received no external funding.

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