Congenital cardiac defect in a pygmy goat (*Capra hircus*)

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Received: 21.05.2010

**Abstract:** Congenital cardiac defects are anatomic conditions present at birth. Few references to such conditions in goats are available in the current scientific literature. This report describes, for the first time, a congenital cardiac disease clinically characterized by polypnea from birth and exercise intolerance in a 2-month-old pygmy goat. An atrioventricular dysplasia known as Ebstein's anomaly, an atrial septal defect, and a mild subaortic stenosis were ultrasonographically diagnosed.

**Key words:** Goat, cardiac defect, Ebstein's anomaly, interatrial septal defect

**Introduction**

Congenital cardiac defects are abnormalities of the cardiac structure that are present at birth. Malformations of the heart can occur in all mammalian species, with the highest prevalence reported in livestock and the lowest in horses (1); there is, however, a lack of information about congenital cardiac defects in goats. Proposed causes include maternal viral infections or metabolic dysfunction, fetal anoxia from placental dysfunction, the use of drugs in pregnant animals, toxins, nutritional deficiency, and genetics. The most common defect in sheep and goats is ventricular septal defect (VSD), but atrial hypoplasia, cardiomegaly, patent ductus arteriosus (PDA), atrial septal defects (ASD), and tetralogy of Fallot have also been reported (2,3,4). Gardner et al. (5) described the occurrence of Ebstein's anomaly (tricuspid valve dysplasia) and atrial septal defect in a male pygmy goat.

The treatment of congenital heart defect is not considered to be economically viable in small ruminants and the inheritability of such diseases make breeding ill-advised when a diagnosis has been made. The present report describes the clinical and instrumental findings in a 2-month-old pygmy goat affected by different cardiac congenital defects.

**Material and methods**

**Case history and examination**

A 2-month-old female pygmy goat (*Capra hircus*) was brought to the Veterinary Teaching Hospital of the School of Veterinary Medical Sciences at the University of Camerino because of the presence of polypnea from birth and exercise intolerance. At the clinical examination the kid was alert, mucus membranes were slightly pale, the respiratory rate was 98/min, the heart rate was 178/min, the arterial pulse was weak and frequent, and the rectal temperature was determined to be 39.3 °C.

During the clinical examination of the cardiovascular system, auscultation revealed a V/VI
systolic murmur with thrill over the tricuspid valve, a IV/VI systolic murmur over the left apex, and a III/VI systolic moderate murmur over the left base. In the auscultation of the thorax, some respiratory crackles and wheezes were revealed in correspondence of the caudal and dorsal lung fields. A hemochrome test was performed but no alteration was revealed. Electrocardiographic examination showed a sinus tachycardia with normal P and T waves and an increase of the QRS amplitude: 0.9 mV with respect to the range of reference (6).

**Analysis**

Echocardiography revealed a dysplastic tricuspid valve displaced toward the apex of the right ventricle (Figure 1), an enlargement of the right atrium and right ventricle, a large left ventricle and atrium (ratio of the left atrium diameter to aortic root diameter: 3.61) (Figure 2), an atrial septal defect, a dysplastic mitral valve, and a mild subaortic stenosis featuring a flow velocity of 2.39 m/s and a systolic pressure gradient of 22.8 mmHg (Figure 3). The Doppler study showed a left-to-right shunt (Figure 4) with
a flow velocity of 2.63 m/s (Figure 5) through the atrial septal defect and a severe mitral (Figure 6) and tricuspid regurgitation (Figure 7) of 4.31 m/s and 3.39 m/s, respectively. M-mode evaluation (Figure 8) allowed us to estimate the ejection fraction (EF = 69%), the shortening fraction (SF = 38%), the left ventricular end-diastolic (LVEDD = 35.9 mm) and end-systolic diameter (LVESD = 22.3 mm), the right ventricular end-diastolic diameter (RVED = 18.1), the interventricular septal thickness in diastole (IVSED = 6.3 mm) and systole (IVSES = 8 mm), and the left ventricular free wall thickness in both diastole (LVWED = 5.4 mm) and systole (LVWES = 7.5 mm).

The goat was hospitalized for 3 days and treated with furosemide (2 mg/kg, twice a day) and enalapril (0.25 mg/kg, once a day), then discharged with the same therapy upon the owner’s request. Another echocardiography was also scheduled, but, unfortunately, the goat was found dead by the owner 2 days later and it was not possible to execute another exam. On the basis of the clinical examination and of the exams already performed, we diagnosed a congenital cardiac disease characterized by an atrioventricular dysplasia (i.e. Ebstein’s anomaly), a mitral dysplasia, an atrial septal defect, and a mild subaortic stenosis. These pathological conditions had never previously been described as occurring simultaneously in a goat.
Results and discussion

The contemporaneous presence of atrioventricular dysplasia and other congenital heart defects, such as septal defect and pulmonary and subaortic stenosis, is common both in humans (7) and in animals (8), but, until now, it has not been described in goats. Only Gardner et al. (5) reported a case of Ebstein’s anomaly and atrial septal defect in a 3-year-old goat. In the present case, several congenital heart defects present in a young pygmy goat were reported.

The kid was affected by mitral and tricuspid dysplasia resulting in atrial systolic regurgitation, demonstrated by an atrial dilation, an increase of the ratio between the left atrium diameter and the aortic root diameter to 3.61 (Figure 2). The mitral and the tricuspid valve regurgitation determines the volume overloading of the left and/or right side of the heart and, subsequently, pulmonary and/or systemic congestion. Chronic volume overload of the ventricle results in eccentric hypertrophy of the involved chamber, which could decrease the contractility of the myocardium and cause the clinical findings of left or right heart failure. In particular, the kid was affected by Ebstein’s anomaly, a specific type of tricuspid valve dysplasia characterized by a congenital apical displacement of the valve in the right ventricle. In humans, Ebstein’s anomaly is present in <1% of all congenital disease (7). In veterinary medicine, there are not many references to the anomaly; Eyster et al. (9) reported Ebstein’s anomaly in 3 dogs and Gardner et al. (5) described a case in a goat. In our report, the Ebstein’s anomaly was clearly shown by an apical displacement of the tricuspid valve in the right ventricle and the septal leaflet was attached to the interventricular septum.

The goat presented the clinical signs of heart failure related to systemic and pulmonary congestion. At the auscultation of the thorax, in fact, some wheezing was observed. Moreover, the ultrasonographic study revealed the presence of an atrial septal defect (1.9 mm) in the middle part of the interatrial septum (Figure 4). ASD is relatively common in humans, but it is rare in animals. The results of 2-dimensional and color Doppler echocardiography showed a shunt through the ASD from the left atrium to the right. It was confirmed by identifying a rate of blood flow across the septum of 2.63 m/s (Figure 5). In addition, the kid was affected by a mild subaortic stenosis with a gradient of pressure of 22.8 mmHg; this condition has never been described before in a goat. This congenital condition obstructed the blood flow to the aorta and caused the weak and frequent pulse that was discovered during the examination of the cardiovascular system.

Even though referential scientific literature about the M-mode parameters of the kid pygmy goat is lacking, it has been possible to make suppositions about the 4-chamber dilation based on a consideration of the reference values for standard-sized adult goats (6,10). Moreover, it was interesting to note that, despite a dilation of the cardiac chambers, there was no alteration of the electrocardiogram, except for an increase of the QRS complex amplitude (0.9 mV, with respect to the range of reference of 0.1-0.7 mV) (6).

It would have been valuable to administer another ultrasonographic examination after the therapy, but the sudden death of the goat prevented it. Our hypothesis is that the heart failure already present at the time of the first echocardiogram became worse, causing the death of the animal. We feel that the present case report is interesting because bibliographic references about echocardiography in the goat are limited and, in recent years, goats have come to be considered not only as livestock, but also as pets. The importance of these animals is not only connected to their zootechnic value, but also to this new consideration of the animal. For this reason, it is important to execute a correct exam of the cardiovascular system in order to ascertain the presence of congenital cardiac defects that could compromise the life of the animal. Furthermore, some authors have referred to the heritable basis of these pathologies in humans and in dogs (7,9). It would be interesting to develop genetic studies in the goat, as well, to avoid the possible transmission of these conditions. Because of the lack of bibliographic references, it is also important to obtain ultrasonographic values of reference in the goat in order to have a correlation and arrive at the correct diagnosis. Smith and Sherman (6) and Moses and Ross (10), in fact, have reported values of references for adult goats, but the values of the kid are absent from the bibliographic references. It would therefore be valuable to also administer echocardiographic examinations to young animals.
References


