Case Report

Cor Triatriatum Sinister in a French Bulldog

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A 3-year-old male French Bulldog was evaluated due to recent history of intolerance to exercise and coughing. The clinical, radiographic, and echocardiographic findings were consistent with cor triatriatum sinister (CTS), a congenital heart anomaly in which the left atrium is subdivided into two compartments by an abnormal fibromuscular membrane. This defect has been rarely recognized in humans and in domestic cats. To the best of our knowledge, this is the first report of the disorder in the canine species.

1. Introduction

Cor triatriatum (CT) literally means “heart with three atria.” It is among the rarest developmental cardiac disorders reported in people [1, 2], dogs [3], and cats [4–6]. Cor triatriatum sinister (CTS) and dexter (CTD) consist of left or right atrium, respectively, divided into two chambers by a fibrous membrane. In CTS, the left auricle has two distinct compartments: one proximal that receives blood from pulmonary veins and one distal that communicates with the left ventricle through mitral valve. The embryogenesis of CTS is still subject of discussion, but the most accepted theory is that it results from lack of normal regression of the fetal pulmonary veins to form the roof of the left atrium [7, 8]. There are one or more small orifices in the separating membrane, allowing communication between both parts of the left auricle [4, 6]. CTS may be found as an isolated defect (classic) or may be associated with other cardiovascular anomalies—the atypical presentation [9]. The pathophysiological consequences of CTS are variable and strongly depend upon the size of the membrane’s orifice. When associated with other cardiovascular defects, it usually represents a serious disorder [9, 10]. If the membrane’s

foramen is large, the disease may show a benign course as seen eventually in adult humans with the classic type [8, 11, 12]. Since 1990s, the transthoracic echocardiography became the first choice imaging technique for the diagnosis of CT due to its high accuracy [1, 9, 10, 13]. This technique is essential not only to establish the diagnosis, but also to assess hemodynamic repercussions and to document eventual associated defects [8]. Other suitable methods for the diagnosis of CTS are cardiac computed tomography and magnetic resonance imaging [12], but they are expensive, rarely available in veterinary clinics, and also need general anesthesia to be performed. From the best of our knowledge, CTS in dogs was not reported before in the veterinary literature. The aim of this study was to describe this peculiar entity that was identified in an adult dog from Rio de Janeiro.

2. Case Presentation

In February 2012, a 3-year-old male French Bulldog was referred for cardiologic evaluation, due to coughing and dyspnea. The dog was on heartworm prophylaxis and had no relevant past medical history. A few days before our

FIGURE 1: Lateral chest radiograph showing mild heart enlargement, signs of pulmonary congestion and oedema (*). The caudal vena cava (==) is calibrous.

evaluation, the patient began to be treated with benazepril and furosemide, showing significant clinical amelioration. The initial treatment has been maintained once the diagnosis of the malformation was made. The dog's owner refused to refer the patient for cardiac surgery, preferring clinical treatment. On physical examination, the dog was in good general physical condition, well active, and weighed 13,1 kg. He was too reactive during all examination but showed no dyspnea or coughing. The cardiac auscultation was normal, the heart rate was 144 beats per minute, the mucous membranes were not cyanotic, and the femoral pulse was regular, strong, and synchronous to the heart beats. The systolic arterial blood pressure measured by the Doppler method was 136 mm Hg. Blood test results (complete blood count, blood urea, creatinine, and fasting glucose) were within normal reference values. The chest radiograph showed a mildly augmented cardiac silhouette, signs of caudal vena cava engorgement, and arterial pulmonary congestion and edema (Figure 1). Also, hemivertebra affecting T3 and T5 to T8 was identified. The electrocardiogram (ECG) was normal (Figure 2). Transthoracic echocardiography revealed a left atrium subdivided by a transverse membrane into two distinct compartments, one proximal and one distal, the hallmark of CTS (Figures 3 and 4). The interatrial septum was seen intact. The left apical four-chamber view documented a continuous forward blood flow across the membrane's orifice (Figure 5). The mitral valve was morphologically normal, and the true left auricle was not enlarged. Mild pulmonary valve regurgitation was detected on the right paraesophageal 4-chamber short-axis view at the level of the cardiac base. The patient has been showing good quality of life since four months ago when furosemide and benazepril were introduced.

3. Discussion

CTS was firstly described in human beings by Church in 1868 [1] and in the domestic cat by Gordon et al. more

FIGURE 2: The ten-lead computed electrocardiographic tracing reveals sinus rhythm, main QRS axis falling between +80 and +90 degrees, heart rate of 150 beats per minute, and no criteria for chamber enlargement. Calibration: 1 cm = 1 mv and velocity of 50 mm per second.

FIGURE 3: The transthoracic two-dimensional echocardiogram (parasternal long-axis view). Note a transverse membrane subdividing the left atrium into two distinct chambers (*), one proximal and one distal, the classical anatomic presentation of CTS. Right ventricle (RV), left ventricle (LV), and aorta (Ao).

FIGURE 4: Left apical four-chamber view in systole, showing a transverse membrane and the two left atrial parts (*LA and **LA). Left ventricle (LV), right atrium (RA), and right ventricle (RV).

FIGURE 5: Transthoracic colour-Doppler echocardiogram (left apical four-chamber view) in diastole. Note a blood flow (green arrow) crossing a central orifice located in the intra-atrial membrane. Right atrium (RA), right ventricle (RV), left atrial chamber (LA), and aorta (AO).

than a century later [14]. Regarding canine species, only the dexter form has been reported [3, 4]. In CTS, the obstructive membrane causes increased pressure in pulmonary venous-capillary circulation, and, in consequence, pulmonary edema or pulmonary hypertension can develop secondarily to vasoconstriction [4]. This hemodynamic disorder may explain the clinical and radiographic changes observed in our case study. CTS may be very difficult to be distinguished from supravulvar mitral stenosis, another rare congenital defect in dogs that has similar physiology. The differentiation between both conditions is based on the level of the obstruction; in supravulvar stenosis the obstructive membrane is distal to left auricle and immediately above the mitral valvular plane, whereas in CTS it is proximal [6, 7, 15], as documented in this paper (Figures 3 and 4). Also, to help differentiation is that, in CTS, the flow crossing the orifice is continuous (Figure 5), while in supravulvar mitral stenosis is diastolic [16]. The peculiar anatomy of CTS can be well delineated by two-dimensional echocardiogram, while

the color-Doppler study permits identifying an increased flow velocity through the membrane's fenestration [12], as observed in our patient (Figures 3, 4, and 5). The electrocardiogram is nonspecific for this anomaly, and, in consequence, a normal ECG does not rule out the defect as reported in adult humans [8, 10–12]. In our patient, the ECG was within normal parameters. We believe that the cardiac condition was not severe enough to produce discernible changes on the ECG surface at the time of examination. In the dog, the pathological findings and natural history of the malformation are unknown but may be identical to that seen in cats [4, 6]. Given that the cardiac defect is obstructive by nature and if the membrane has a small hole, the hemodynamic changes tend to worsen with time. The disease progress may cause enlargement of left proximal atrial chamber, main pulmonary artery, right atrial dilatation, right ventricular dilatation, and eccentric hypertrophy [4, 6]. When heart failure develops, pharmacologic therapy with diuretics and angiotensin converting enzyme inhibitors (ACEIs) is beneficial [4, 17]. Furosemide is a drug of choice for heart failure because it is a potent diuretic that reduces preload and relieves congestion secondarily to cardiac dysfunction. In consequence, it reduces edema formation and dyspnea. However, because furosemide activates the rennin-angiotensin-aldosterone system, a concomitant use of an ACEI is advisable [6]. Benazepril hydrochloride is a long-acting ACEI that counterbalances the adverse effects caused by ACE activity, that is, exacerbated in heart failure. This drug produces significant prolongation of survival time of dogs with heart failure, improves the exercise capacity, and is well tolerated [17]. Although very rare, CTS should be included in the differential diagnosis of dogs with signs of heart failure and suspect congenital heart defect. This malformation is important to be early recognized because it is potentially correctable by surgery when clinically significant.

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