CASE REPORT

ISSN Online 1678-4456

Cor triatriatum sinister in a cat: case report

Cor triatriatum sinister em um gato: relato de caso

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ABSTRACT

Cor triatriatum sinister is an uncommon congenital condition characterized by an aberrant membrane that divides the left atrium into two chambers due to an abnormal incorporation of the pulmonary veins. This anomaly obstructs blood flow to the left atrium, leading to pulmonary hypertension and, consequently, congestive heart failure. This case report presents a cat that was treated in the veterinary cardiology service for dyspnea. An echocardiogram revealed an intra-atrial membrane within the left atrium. Due to the patient´s deteriorating condition, euthanasia was performed, and subsequent necropsy and histopathology examination also confirmed the diagnosis of cor triatriatum sinister.

Keywords: Anomaly. Pulmonary hypertension. Hemodynamic. Cats. CTS.

RESUMO

O cor triatriatum sinister é uma condição congênita rara caracterizada por uma membrana aberrante que divide o átrio esquerdo em duas câmaras devido a uma incorporação anormal das veias pulmonares. Esta anomalia obstrui o fluxo sanguíneo para o átrio esquerdo, resultando em hipertensão pulmonar e, consequentemente, insuficiência cardíaca congestiva. Este relato de caso descreve um paciente felino que foi atendido no serviço de cardiologia veterinária devido a quadro de dispneia. O ecocardiograma revelou a presença de uma membrana intra-atrial no átrio esquerdo. Devido à deterioração da condição do paciente, foi realizada a eutanásia e a necropsia subsequente, bem como o exame histopatológico, que confirmaram o diagnóstico do cor triatriatum sinister.

Palavras chaves: Anomalia. Hipertensão pulmonar. Hemodinâmico. Gatos. CTS.

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Received: August 13, 2024
Approved: June 30, 2025

How to cite: León FAM, Fernandes TR, Souza GG, Siqueira RC, Félix M, Andreatti Filho RL, Hataka A. Cor triatriatum heart in a cat: case report. Braz J Vet Res Anim Sci. 2025;62: e227921. https://doi.org/10.11606/issn.1678-4456.bjvras.2024.227921.

Cor triatriatum (CT) results from the anomalous incorporation of the pulmonary vein into the atrium, leading to the formation of a fibromuscular membrane that separates the atrium into two chambers. The low (apical) chamber communicates with the left ventricle through the mitral valve, while the upper chamber receives the pulmonary venous inflow. It is termed CT sinister (CTS) when located in the left atrium. (García-Fuertes et al., 2021), and CT dexter (CTD) when in the right atrium (Jha & Makhija, 2017).

This pathology can be found either as an isolated anomaly or, less frequently, associated with another cardiovascular anomaly (Almeida et al., 2012).

Several theories have been proposed to explain this malformation, but the embryological basis remains incompletely understood (Butt et al., 2024). The most accepted theory, known as malincorporation, suggests that the common pulmonary vein fails to consolidate properly into the left atrium, leading to the formation of two chambers separated by a narrow opening. However, this theory does not fully describe the existence of the fossa ovalis and atrial muscle fibers within the proximal chambers, where only a venous wall should be present. Another hypothesis, malseptation, proposes that anomalous growth of the septum primum affects the atrial septum. Additionally, the entrapment theory proposes that the left horn of the embryonic sinus venosus traps the common pulmonary vein, impeding its proper incorporation into the left atrium (Nassar & Hamdan, 2011) (Kumar et al., 2022).

The diameter of the perforation is in inverse variation to the severity of the lesion. Obstruction of pulmonary venous return leads to elevated pulmonary pressures and fluctuating degrees of secondary pulmonary arterial hypertension. In early stages of the disease, increment pulmonary venous pressure serves as a passive response to preserving a tolerable gradient for the left atrial filling (Heaney & Bulmer, 2004).

Diagnosis can be made using bidimensional echocardiography, but it can also be performed with computed tomography, magnetic resonance imaging, and three-dimensional (3D) echocardiography (Champion et al., 2014). However, these methods are expensive, infrequently available in veterinary clinics, and require general anesthesia to be conducted (Almeida et al., 2012). Treatment of CTD consists of balloon dilatation or surgical removal of the atrial membrane (Borenstein et al., 2015).

A 9-month-old male Maine Coon cat was referred to the Specialized Veterinary Cardiology Service with signs of acute pulmonary edema. Physical examination revealed pale mucous membranes, a rectal temperature of 36.2°C, systolic arterial pressure of 70 mmHg, and oxygen saturation (SO2): 85%. Cardiopulmonary auscultation revealed bilateral pulmonary rattles and a systolic murmur at the mitral focus grade 4/6.

Thoracic radiography revealed a globular appearance of the heart and lung parenchyma showing an alveolar pattern, indicative of cardiogenic edema and/or pneumonia (Figures 1A,B).

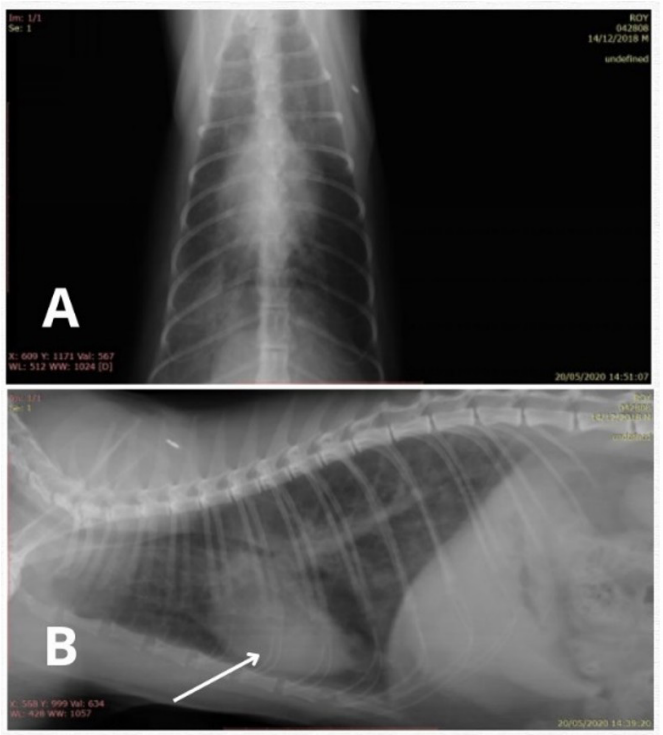

Figure 1 – **(A)** Ventrodorsal and **(B)** Lateral thoracic radiographs of the patient, heart with a globular appearance (arrow) and lung parenchyma exhibiting an alveolar pattern. **Source:** Personal archive.

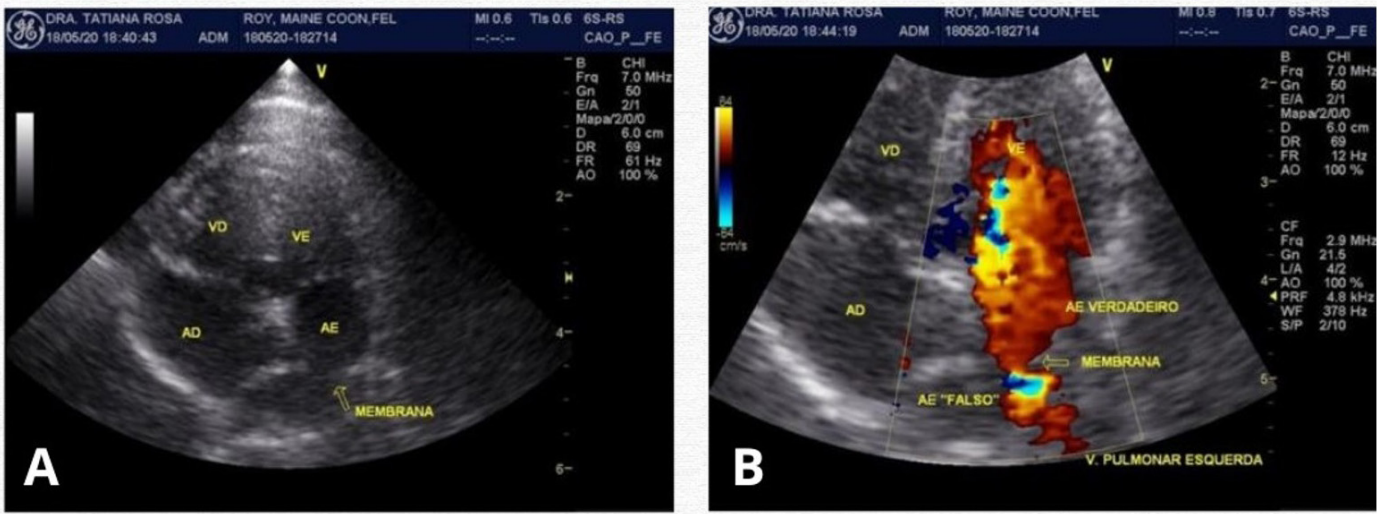

Figure 2 – **(A)** Longitudinal echocardiographic view in apical 4-chamber cut (left parasternal window), arrow indicating left intra-atrial membrane. **(B)** Echocardiographic image apical 4-chamber view, color Doppler method demonstrating turbulent systolic flow within the left atrium, due to increased velocity across the intra-atrial membrane. **Source:** Personal archive.

Echocardiography revealed a membrane 0.17 cm in length, dividing the left atrium into two chambers, in addition to a reduction of the systolic and diastolic internal diameters of the left ventricle, and signs of post-capillary intermediate probability of pulmonary hypertension. According to Reinero et al. (2020), three echo signs in anatomic sites of pulmonary hypertension classify as high probability of PH such as: right atrial and ventricular enlargement, peak tricuspid regurgitation velocity not measurable, the right pulmonary artery distensibility index (RPAD) was 24% (reference value: above 30%), enlargement of pulmonary artery (AP/Ao relation: 1,3) and enlargement of caudal vena cava (Figures 2A,B).

The animal was promptly treated with an emergency protocol, including oxygen therapy (1.5 L/ min), sedation with butorphanol 0.2 mg/kg, and furosemide 1mg/kg TID (IV). The animal was hospitalized for 2 days.

The post-hospitalization prescription included pimobendan 0.62 mg BID, clopidogrel (18.75 mg/animal) SID, furosemide 10 mg BID, and sildenafil citrate 1 mg/kg.

Despite the administration of medication, the animal continued to exhibit dyspnea, pulmonary edema, and pleural effusion. Due to the worsening condition, euthanasia was elected.

Necropsy findings revealed that the oral mucosa was cyanotic, along with hydropericardium, pulmonary edema, and an accentuated lobular hepatic pattern. No macroscopic alterations were observed in the external examination of the heart. However, upon examination, the left atrium section of the heart showed that an abnormal septum had formed a thick, white membrane dividing the chamber, which was equivalent to CTS (Figure 3).

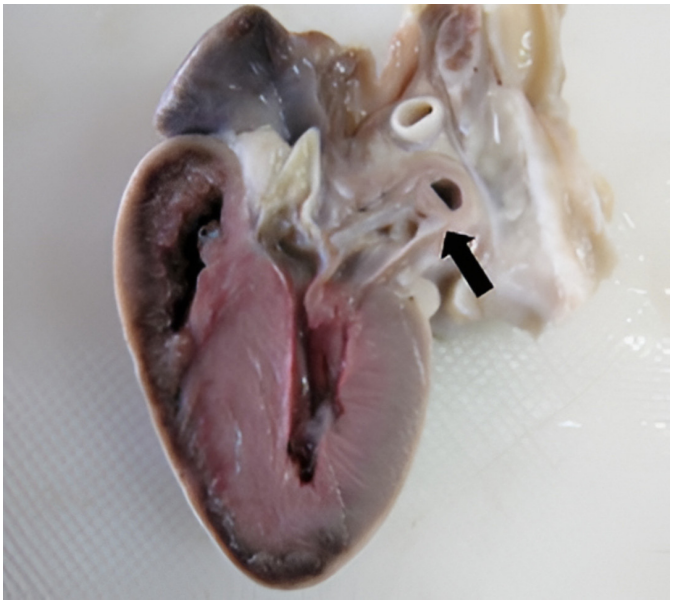

Figure 3 – Longitudinal section of the left heart chamber showing a thick intra-atrial membrane (arrow) in the left atrium, dividing it into two chambers. **Source:** Personal archive.

Collected fragments were fixed in a 10% buffered formalin solution, dehydrated, diaphanized, bathed, and embedded in histological paraffin. Cuts of 4 micrometers were stained with hematoxylin-eosin and Masson's trichrome. The histological examination revealed cardiac muscle tissue separated by a membrane composed of fibrous and muscular tissue, and the muscle cells were localized only at the periphery of the membrane. Masson´s trichome stain was used to highlight this membrane (Figure 4).

CTS is an abnormality characterized by an anomalous membrane that splits the left atrium into two compartments. These compartments include the true left atrium and the

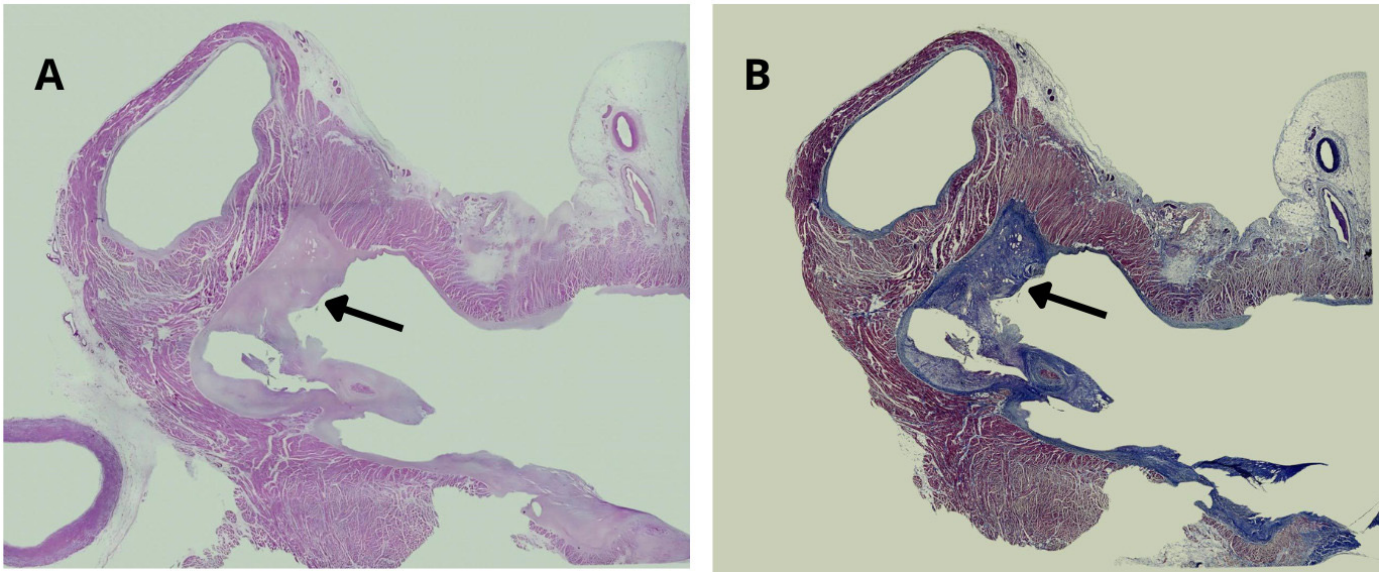

Figure 4 – **(A)** H&E staining shows the cardiac muscle separated by a membrane (arrow). **(B)** Masson's Trichrome staining, with blue (arrow) highlighting the membrane that divides the atrium. Submacro magnification.

accessorial atrial chamber. The membrane itself can vary significantly in size and shape, resembling either a diaphragm or a funnel shape. It may be entirely intact (imperforate) or contain one or more fenestrations of varying diameters (Castagna et al., 2019).

In humans, the CTS has a prevalence of 0.1% among congenital heart diseases. Symptomatic cases are typically diagnosed in childhood and are linked with a high mortality rate. Critically obstructed cases in adults are very rare (Narayanapillai, 2016).

In animals, this anomaly has been observed in dogs, although it is more commonly reported in cats (Champion et al., 2014). Recent studies in animals have reported that in dogs, CTS represents 4.59% of cases among congenital cardiac diseases (Kumar et al., 2022).

Most case reports have described this congenital abnormality in cats under one year of age, which is consistent with our finding in a 9-month-old cat. However, an exceptional case has been reported by Morey & Mueller (2020) in an 11-year-old cat.

CT results in impediment of venous flow through the atrium. In CTD, a membrane forms in the right atrium between the caudal vena cava and the tricuspid valve, obstructing venous return from the caudal vena cava and leading to ascites, while not affecting flow from the cranial vena cava. In CTS, the membrane impedes venous flow from the pulmonary veins, resulting in pulmonary edema (Orton, 2012).

Suppose the interconnection among pulmonary veins and the systemic venous system fails (I-Ping & Tung, 2022). In that case, this means that any lesion that restricts pulmonary venous return will produce elevated pulmonary venous pressures and varying degrees of secondary (post-capillary) pulmonary arterial hypertension (Heaney & Bulmer, 2004). Determining the actual prevalence in veterinary medicine is challenging, as it does not always present with a heart murmur (I-Ping & Tung, 2022). Depending on the severity of the pulmonary hypertension, cats could exhibit signs of both left and right-sided heart failure (Heaney & Bulmer, 2004).

In human medicine, CTS classifications can be categorized into the shape and position of the proximal chamber, patterns of pulmonary venous drainage, among other factors (Koie et al., 2000), as well as the quantity and morphology of fenestrations in the anomalous membrane (Castagna et al., 2019).

The differential diagnosis of CTS includes supravalvular mitral stenosis. The key distinction between these abnormalities is the location of the membrane in association with the auricle. In the CTS, the membrane is placed high in the intersection between the left auricle and atrium, as observed in the echocardiogram of the cat reported here, while in the supravulvar mitral stenosis, the membrane is located under the intersection of the atria, above the mitral valve (Henze et al., 2023).

The breed of the patient described here is very predisposed to hypertrophic cardiomyopathy (HCM), so it has to be eliminated in the differential diagnosis of a cardiac disease in these animals. This disease has been described in various species, particularly in cats, associated with a high risk of sudden death, congestive heart failure, and aortic thromboembolism. Diagnosis is performed by an echocardiogram, revealing hypertrophy of the interventricular septum and the left ventricular free wall. In 1999, HCM was identified as a hereditary disorder in a family of Maine Coon cats, following an autosomal dominant inheritance

pattern with complete penetrance. Later in 2005, the cause of the mutation in this breed was discovered in the MYBPC3 gene. This mutation leads to the substitution of alanine with proline at codon 31, resulting in an aberrant protein (Mary et al., 2010)

Previously, cardiac catheterization was the standard method for diagnosing CTS before the advent of echocardiography. Angiography could establish the diagnosis by demonstrating differential filling of the two distinct atrial chambers. Today, echocardiography is the preferred noninvasive technique, as it can precisely delineate the morphology of the membrane and associated cardiac lesions. On a four-chamber view, echocardiography typically illustrates distal to the mitral valve and the left auricle a thin linear structure that divides the atrium. The superior part of the membrane is frequently parallel to the aortic wall. Additionally, one or more foramina may be visible within the membrane, often showing increased flow velocity on color Doppler imaging (Nassar & Hamdan, 2011).

Although some authors consider these techniques unreliable for distinguishing triatriatum from other causes of left ventricular inflow obstruction in humans, and angiography may worsen the condition in ill infants, they conclude that the best technique for the diagnosis is echocardiography (Ostman-Smith et al., 1984).

The use of computed tomography, nuclear medicine lung perfusion imaging, and cardiac magnetic resonance can serve as alternative diagnostic methods, but they were not performed in this case. However, employing these techniques in veterinary medicine could signify a risk to the patient due to the need for anesthesia (Kriström et al., 2023).

The indication for repair depends on the degree of obstruction (Rudienė et al., 2019). The definitive treatment is the complete surgical excision of the membrane (Keene & Tou, 2015).

Transcatheter treatment is often preferred over open-chest surgery due to its lower invasiveness, reduced tissue trauma, and greater cost-effectiveness. However, when a cardiopulmonary bypass (CPB) approach is used, it provides a secure and controlled method with minimal risk of cardiac rupture. This approach allows for excellent visualization and direct excision of the membrane, followed by spontaneous restoration of normal blood flow (Borenstein et al., 2015).

Surgical resection and balloon dilation have proven effective for CTD in dogs and cats with CTS. These techniques represent a cost-effective approach for managing this malformation in small animals (Castagna et al., 2019). Additionally, a hybrid technique involving the insertion of an inflatable balloon through the defect, following a thoracotomy to access the left atrium, has been successfully performed in one cat with CTS (Stern et al., 2013).

The choice of the method should consider the animal's weight, anatomy, concurrent cardiac conditions, the technical proficiency of the surgical team, and the available sources. Cardiac surgery in cats is particularly challenging due to their small size. The lack of neonatal oxygenators and feline blood products makes CPB less accessible in cats compared to dogs. Nonetheless, many of these challenges can be managed with an appropriate CPB strategy and a pediatric cardiac surgery team (Borenstein et al., 2015).

The cutting balloon allows for scoring of the perforation, facilitating subsequent standard balloon dilatation to a much larger diameter. However, an interventional approach to the cat´s left atrium is challenging due to the requirement for large venous and arterial access to introduce balloon catheters. To overcome these limitations, a combined surgical and interventional approach, guided by transesophageal echocardiography, proved to be an optimal strategy, as it enabled the use of a cutting balloon followed by standard dilation without using inflow occlusion, cardiac bypass, or atriotomy (Stern et al., 2013).

The principal benefits of balloon expandable stents and cut-balloon consist of their capacity to reach the desired diameter and avoid the foreshortening issues often encountered with some self-expanding metallic stents. However, potential complications of this procedure include the risk of balloon entrapment within the stent during removal, stent stenosis due to muscular ingrowth, and movement of the balloon during inflation, which can lead to suboptimal placement of the stent (Barncord et al., 2016). The combination of diuretics (furosemide), ACE inhibitors (benazepril), and inodilator (pimobendan) is the preferred treatment for managing symptoms of CTS, as used and prescribed for the patient in this report. Diuretics help reduce preload and alleviate congestion resulting from cardiac dysfunction. While these medications can be used individually, they activate the renin-angiotensin-aldosterone system (RAAS) and should be combined with ACE inhibitors to enhance therapeutic efficacy (Kumar et al., 2022).

During necropsy, as observed in this case report, a membrane dividing the left atrium, distinct from the mitral valve, was observed. Microscopically, the membrane exhibits a fibromuscular pattern. Additional findings include pulmonary edema and significant thickening of the pulmonary artery wall, indicative of pulmonary arterial hypertension. Passive congestion in the liver and degeneration of renal tubules are also noted (Champion et al., 2014).

The case reported here is compatible with Cor Triatriatum sinister, a serious anomaly that is detrimental to the quality of life of affected animals, and which should be considered by the pathologist during the necropsy of young animals. The importance of complementary examinations, as carried out antemortem in this case, should be emphasized, as they were fundamental to understanding the hemodynamic consequences of the CTS process.

Conflict of Interest

The authors declare no conflict of interest in the writing of the manuscript or in the decision to publish this article.

Ethics Statement

All procedures involving the handling of animals were carried out under the approval of the Ethics Committee on the Use of Animals of the School of Veterinary Medicine and Animal Science of the São Paulo State University, with the protocol code number: CEUA 000.191.

Acknowledgements

The authors acknowledge the Department of Veterinary Clinical Sciences, School of Veterinary Medicine and Animal Science, São Paulo State University (UNESP), Botucatu, São Paulo, Brazil for the structure and material support.

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Financial Support: The authors declare that no financial support was received for this study.