



Congestive heart failure in a young cat with excessive moderator bands (false tendons) in the left ventricle

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ABSTRACT: *Left ventricular false tendons are fibrous or fibromuscular bands that transverse the ventricular cavity and have no attachment to the mitral valve in many species. In cats it is considered a congenital defect that is rarely related to clinical disease and death in adult cats. A 45 days-old mixed breed cat had a history of inappetence since birth. At the physical exam the patient was lethargic and presented restrictive dyspnoea. At necropsy, there were marked ascites, hydrothorax, hepatomegaly with enhanced lobular pattern (nutmeg liver), and the lungs were markedly diminished (compressive pulmonary atelectasis). The heart was enlarged due to marked dilation of the cardiac chambers. Moreover, multiple slightly whitish and irregular cord-like structures were connecting the posterior papillary muscle to the interventricular septum (excessive moderator bands /left ventricular false tendons) at the left ventricle. Microscopically, these structures were characterized by a marked proliferation of fibrous connective tissue intermixed with Purkinje cells and well-differentiated cardiomyocytes lined by a single layer of endocardium. This study described a case of excessive moderator bands (left ventricular false tendons) in a young cat associated with congestive heart failure and death.*

Key words: *feline, cardiomyopathy, congenital, cardiovascular diseases.*

Insuficiência cardíaca congestiva em um gato jovem com bandas moderadoras excessivas (falsos tendões) em ventrículo esquerdo

RESUMO: *Falsos tendões são bandas fibrosas ou fibromusculares que atravessam a cavidade do ventrículo esquerdo, sem ligação com a valva mitral, em várias espécies. Nos gatos, a alteração é considerada um defeito congênito que raramente está relacionada com alterações clínicas e morte em gatos adultos. Um gato de 45 dias de idade, sem raça definida e com histórico de inapetência desde o nascimento foi encaminhado para atendimento veterinário. Ao exame físico apresentou letargia e dispnéia restritiva. À necropsia, havia acentuada ascite, hidrotórax, hepatomegalia com evidência do padrão lobular (fígado noz-moscada) e os pulmões apresentavam-se acentuadamente diminuídos (atelectasia pulmonar compressiva). O coração apresentava acentuada dilatação das câmaras cardíacas. Em ventrículo esquerdo, havia numerosas estruturas brancas semelhantes a cordas conectando o músculo papilar ao septo interventricular (bandas moderadoras excessivas/falsos tendões). Microscopicamente, estas estruturas apresentavam acentuada proliferação de tecido conjuntivo fibroso, além de células de Purkinje e cardiomiócitos bem diferenciados revestidas por uma única camada de endocárdio. O objetivo deste trabalho é descrever um caso de bandas moderadoras excessivas (falsos tendões) em ventrículo esquerdo de um gato jovem com insuficiência cardíaca congestiva e morte.*

Palavras-chave: *felino, cardiomiopatia, congênito, doença cardiovascular.*

Cardiomyopathies are cardiac functional and morphological disorders, which are classified as hypertrophic, restrictive, dilated and, rarely, as excessive moderator bands (false tendons) in the left ventricle in cats (ROBINSON & ROBINSON, 2016; ARGENTA et al., 2020). Furthermore, a consensus statement for the classification, diagnosis and management of cardiomyopathies in cats has been recently established, in which a classification system based on cardiac structure and function (phenotype) was defined (FUENTES et al., 2020). These phenotypes are further classified in hypertrophic

cardiomyopathy, restrictive (divided as endomyocardial form and myocardial form), dilated, arrhythmogenic, and nonspecific phenotype (FUENTES et al., 2020). Left ventricular false tendons are fibrous or fibromuscular bands that transverse the ventricular cavity and have no attachment to the mitral valve (PHILIP et al., 2011). Microscopically, the excessive moderator bands consist of Purkinje cells, muscle cells and dense collagen covered by endothelium (KIMURA et al., 2015; ROBINSON & ROBINSON, 2016). In humans, the presence of left ventricular false tendons has been noted in patients undergoing

echocardiography for a variety of cardiac conditions (KERVANCIOGLU et al., 2003). In addition, several studies have indicated that they might have a pathophysiologic role in ventricular geometric anomalies and arrhythmias (MALOUF et al., 1986; LOUKAS et al., 2007). In cats, the left ventricular false tendons are referred to as excessive moderator bands and have been reportedly associated with heart failure and death (LIU et al., 1982). Although, it is considered a congenital defect in cats, clinical disease is a rare condition of adult cats characterized by left heart failure disease (ROBINSON & ROBINSON, 2016), with a decreased diastolic capacity and electrical conduction disorders (CAVALCANTI et al., 2018). This study described the pathological findings of a fatal case of congestive heart failure in a 45 days-old mixed breed cat with excessive moderator bands (false tendons) in the left ventricle.

A 45 days-old mixed breed female cat, which was born as the smallest of the litter, presented clinical signs of lethargy, restrictive dyspnea, and cyanosis. A presumptive diagnosis of left heart failure due to a possible cardiac defect was established. Due to the patient's dyspnea status, imaging tests were not performed. Supportive treatment was employed, but clinical response was poor, and euthanasia was

elect. At the necropsy, there were large amounts of serous and slightly reddish liquid (transudate) within the abdominal, thoracic and pericardial cavities (interpreted as ascites, hydrothorax, and hydropericardium, respectively). The lung was markedly small due to compressive atelectasis. The liver was enlarged and dark red, with an enhanced lobular pattern (nutmeg liver) and on the cut surface it exuded moderate amounts of blood (congestion) (Figure 1). The heart was enlarged, with a round aspect, and had marked dilation of the ventricular chambers. No valvar changes, and/or other cardiac malformations were observed. Within the left ventricle chamber, there were multiple slightly whitish and irregular cord-like structures, measuring approximately three times the diameter of a tendinous cord, which connected the posterior papillary muscle to the interventricular septum (excessive moderator bands) (Figure 2). Samples of heart, lungs, stomach, liver, lymph node, large and small intestine, kidneys, central nervous systems, spleen, urinary bladder, adrenal, thyroid and parathyroid were collected, fixed in 10% neutral buffered formalin, routinely processed for histology, and stained by haematoxylin and eosin (HE). Additionally, heart fragments were stained by Masson's trichrome (MT).

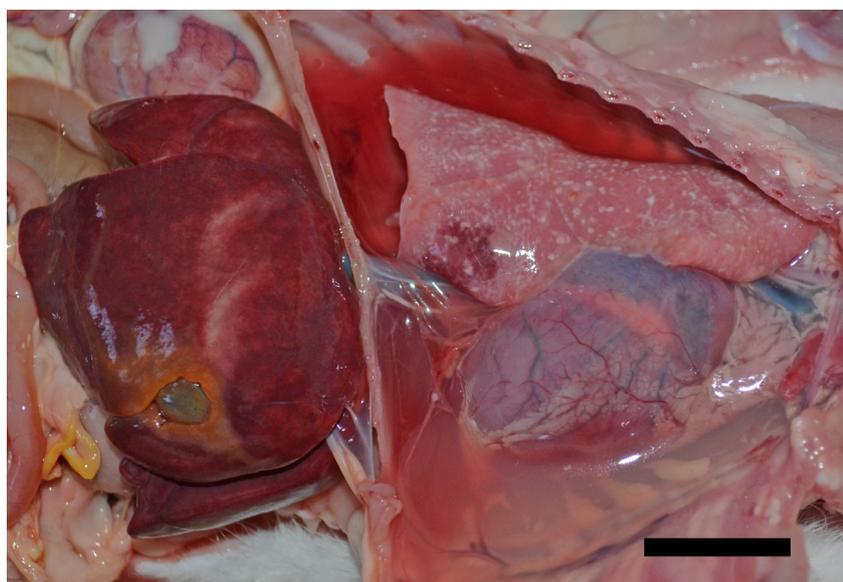
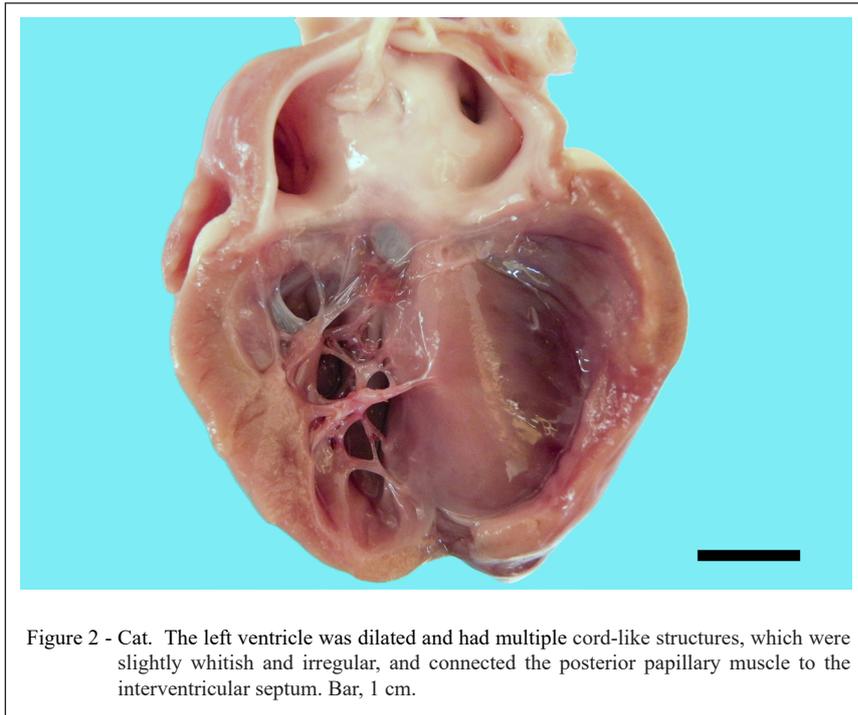


Figure 1 - Cat. Large amounts of serous and slightly reddish liquid fluid in the thoracic cavity (hydrothorax), with marked pulmonary atelectasis. The liver was enlarged, with rounded edges, and a diffuse dark-red color, in addition to marked evidence of the lobular pattern. Bar, 4 cm.



Microscopically, the cord-like structures at the left ventricle consisted of marked proliferation of fibrous connective tissue intermixed with occasional Purkinje cells and multiple well-differentiated cardiomyocytes, which were lined by a single layer of endothelium (Figure 3). The fibrous connective tissue was highlighted by MT stain (Figure 4). The remaining of the myocardium was not affected. The lungs exhibited marked and diffuse alveolar atelectasis and the liver had intense centrilobular congestion.

The diagnosis of congestive heart failure caused by excessive moderator bands (false tendons) in the present case was based on the pathological findings. Excessive moderator bands have been previously associated with heart failure and death in cats. Although, considered a congenital disease, clinical signs are usually observed in senile cats (FERASIN et al., 2003; WRAY et al., 2007; ROBINSON & ROBINSON, 2016; CAVALCANTI et al., 2018), while in this study the condition was diagnosed in a 45 days-old cat. Clinically, the condition is usually characterized by lethargy, anorexia and restrictive dyspnoea in a variable clinical course, in addition to abdominal breathing pattern and muffled sounds at cardiac auscultation (FERASIN et al., 2003; WRAY et al., 2007; CAVALCANTI et al.,

2018), similarly to the present study, in which the cat had inappetence and dyspnoea since birth.

Cats older than eight years-old are predisposed to develop dilated cardiomyopathy as a result of the presence of excessive moderator bands (false tendons), while younger cats (less than four years-old) tend to develop hypertrophic cardiomyopathy under the same conditions (LIU et al., 1982). In the present study; however, the cat presented secondary dilated cardiomyopathy, which may be related to the large amount of false tendons observed at the necropsy. These may have caused a more severe and acute condition, culminating with dyspnea, anorexia and lethargy.

The gross findings of excessive moderator bands related cardiomyopathy in cats included enlarged heart with a round aspect, rounded apex and, in some cases, the left ventricle may be narrow due to the presence of false tendons connecting the ventricle free wall to the ventricular septum (LIU & FOX, 1999). Similar findings were observed in this case, in which the heart was enlarged, with a round aspect and presented marked dilation of the ventricular chambers. Other gross findings included those related to increased hydrostatic pressure within blood vessels, such as ascites, hydrothorax and hydropericardium, in addition to those related with venous congestion,

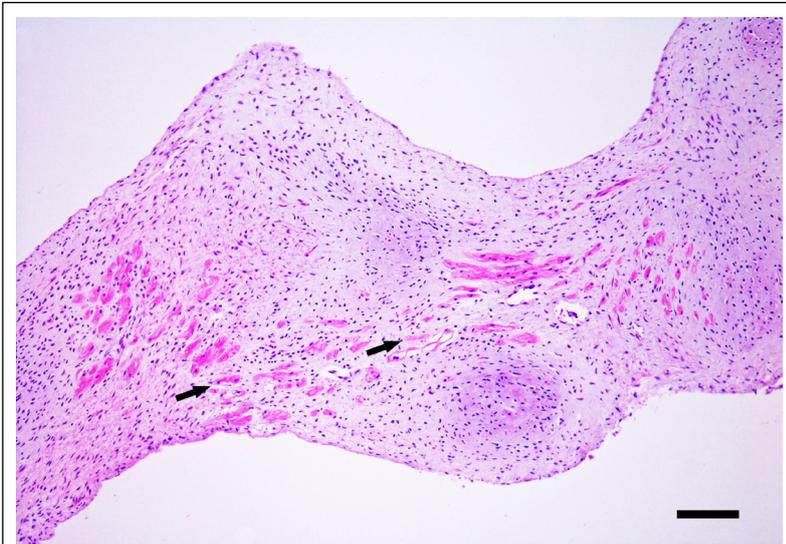


Figure 3 - Cat. Microscopically, the cord-like structures at the left ventricle consisted of marked proliferation of fibrous connective tissue intermixed with occasional Purkinje cells (arrows) and multiple well-differentiated cardiomyocytes, which were lined by a single layer of endothelium. HE. Bar, 140 μ m.

such as enhanced lobular pattern of the liver (nutmeg liver). Even though the false tendons are present in the left ventricle, the gross findings in this case are related to both left and right heart failure. This may be explained by the fact that the cardiovascular system is a closed circuit, in which the failure of

one side will embarrass the other (ROBINSON & ROBINSON, 2016). Left heart failure usually causes pulmonary edema as a result of elevated pressure in the lung vascular bed (postcapillary hypertension) (ROBINSON & ROBINSON, 2016). In the present case; although, lesions were mostly located in the left

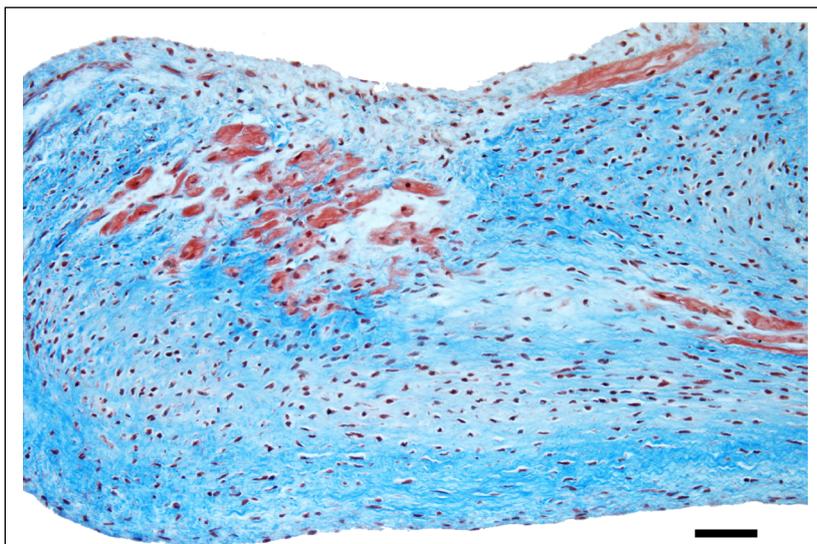


Figure 4 - Cat. The fibrous connective tissue within the excessive moderator band was highlighted (in blue) with Masson's trichrome stain. Bar, 70 μ m.

ventricle, pulmonary edema was not observed. Indeed, the lungs were markedly reduced due to compressive atelectasis caused by hydrothorax. Thoracic effusion is also common in acute heart failure and may reflect the inability of pleural lymphatics to clear sudden increase in pressure-driven pleural fluid formation (ROBINSON & ROBINSON, 2016).

In the present case, it was observed a marked proliferation of fibrous connective tissue, which were highlighted by the MT stain, intermixed with occasional Purkinje cells and multiple well-differentiated cardiomyocytes lined by a single layer of endothelium, similarly to previous studies. Although, previous studies have identified inflammatory and necrotic lesions within excessive moderator bands in cats with congestive cardiac failure (WREY et al., 2007; CAVALCANTI et al., 2018), the present case did not present any of these lesions probably due to the age of the cat.

It is important to consider excessive moderator bands (false tendons) in left ventricle as a differential diagnosis in cases of cardiomyopathy in young cats, with clinical signs of congestive heart failure. The gross, microscopic and histochemical findings are essential to obtain a conclusive diagnosis in these cases.

ACKNOWLEDGEMENTS

This research was supported by Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) and was financed in part by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), Brasil – Finance code 001.

DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflict of interest. The founding sponsors had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, and in the decision to publish the results

AUTHORS' CONTRIBUTIONS

All authors contributed equally for the conception and writing of the manuscript. All authors critically revised the manuscript and approved of the final.

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