

Case Report

Anomalous Chordae Tendineae Associated with Mitral Valve Dysplasia in a Cat

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Abstract

A rare case of anomalous chordae tendineae associated with mitral valve dysplasia was observed during necropsy in a 5-year-old male Persian cat with a history of sudden death. Grossly, there was thickening of the mitral valve, which was connected to numerous, short and thickened chordae tendineae that were ectopically inserted into the myocardium. In addition, marked left atrial dilation and pulmonary congestion and edema were present.

Keywords: cat, congenital disease, heart

Introduction

Anomalous chordae tendineae (ACT) is a rare idiopathic congenital cardiac abnormality (3,11) in which the chordae may be present in greater numbers and enshrined in ectopic locations such as the free wall of the ventricles, the interventricular septum, and extending from one papillary muscle to another (10). In humans, this is an infrequent condition, and cases have been reported in children and adults (4,8,11,13,15). In veterinary medicine, only one case of ACT has been described in a dog (3). Although the etiology of this condition is unknown, it is believed to be the result of changes during organogenesis (12,14). Chordae tendineae are fibrous filaments that connect each valve cusp to the ventricular cavity. In general, two papillary muscles are present, and chordae tendineae are arranged such that each cusp connects two muscles (2). This arrangement prevents protrusion of the cusps into the atrium during ventricular systole (2). Thus, the main consequence of ACT is the limitation of the movement of the valve leaflets, causing valve prolapse.

This results in incomplete leaflet coaptation, causing blood reflux, heart failure, and death (11).

Mitral valve dysplasia (MD) is defined as an abnormally formed mitral valve (1). Grossly, it is characterized by leaflet thickening and shortening as well as fusion, thickening, or aplasia of the chordae tendineae with dysplastic and malpositioned papillary muscles. MD is the most common congenital heart disease in cats (1), but there have been no reported cases associated with anomalous chordae tendineae.

The aim of this study was to describe a rare case of ACT associated with MD in a cat.

Case report

A 5-year-old castrated male Persian cat was submitted to the Veterinary Hospital's Pathology Department at the Universidade Federal de Minas Gerais (UFMG), Brazil, for necropsy. According to the owner, the cat presented with cyanosis, respiratory distress, and sudden death following bathing in a pet store. Necropsy

revealed a cyanotic tongue and visible mucous membranes. The musculature was moderately hyperemic. There was thickening of the mitral valve, which was connected to numerous, short and thickened chordae tendineae that were ectopically inserted into the myocardium (Figure 1). In addition, the abnormal chordae tendineae had an irregular alignment and insertion into the left ventricular free wall, interventricular septum, and papillary muscles. There was also dilation of the left atrium (Figure 1). The lungs were moderately red due to congestion, and moderate amounts of white and frothy edema fluid oozed out from the parenchyma on the cut surface. The other organs demonstrated no gross changes.

Fragments of several organs were collected, fixed in 10% neutral buffered formalin, routinely processed for histology, and stained with hematoxylin and eosin and Masson's trichrome.

Microscopically, the anomalous chordae were similar to the normal control chordae located in the right ventricle, i.e., composed of connective tissue, including collagenous fibers, few elastic fibers, and fibroblasts stained blue by Masson's trichrome (Figure 2).

The lungs were moderately congested, and the alveolar spaces contained amorphous eosinophilic material characteristic of edema. Multifocal areas of mild alveolar hemorrhage were also observed. Based on the gross and histopathological findings, the diagnosis of ACT associated with MD causing acute left heart failure was confirmed.

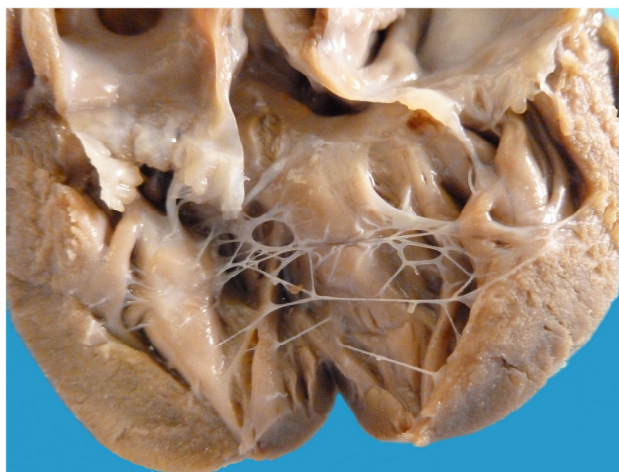


Figure 1. Anomalous chordae tendineae associated with left heart failure in a cat. Anomalous chordae tendineae inserted in the free wall of the left ventricle and interventricular septum and from one papillary muscle to another. The left atrium is intensely dilated.

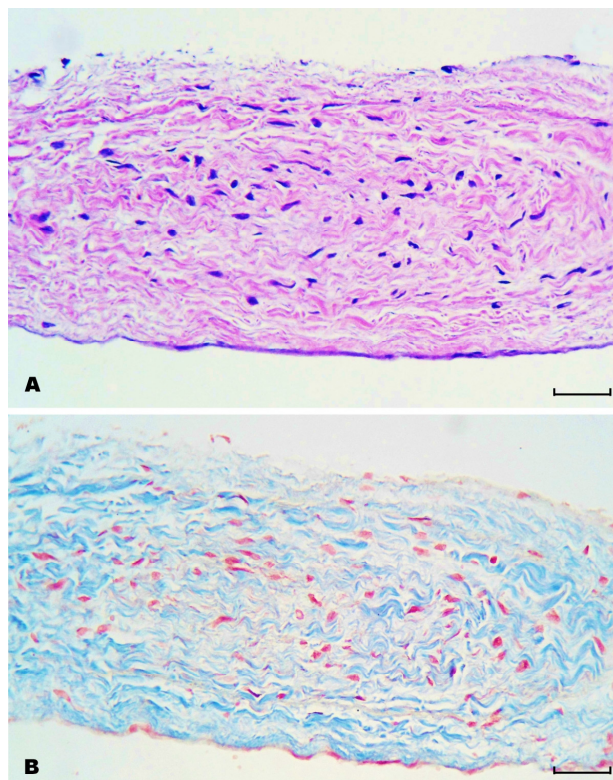


Figure 2. Anomalous chordae tendineae associated with left heart failure in a cat. Anomalous chordae characterized by connective tissue composed of collagenous fibers. HE, (A) Bar = 18.05 μ m, and Masson's trichrome (B) Bar = 16.7 μ m.

Discussion

The animal in this case showed two heart defects, namely MD and ACT. MD is a congenital defect commonly observed in cats and macroscopically diagnosed in this case. The MD diagnosis is based on macroscopic characteristics or ancillary exams, including thoracic radiographs and electrocardiography (1). Conversely, ACT is a rare condition in veterinary medicine, and describing its occurrence in a cat was the primary aim of this report. These congenital changes (MD and ACT) likely caused subclinical valvular insufficiency and consequent acute heart failure (11). The severe left atrial dilation was most likely due to valve prolapse caused by ACT associated with mitral valve dysplasia, resulting in blood reflux into the atrium during ventricular systole. An important cause of atrial dilatation is hypertrophic cardiomyopathy (HCM). HCM is the most common heart disease in cats. It is macroscopically characterized by hypertrophy of the left ventricular wall with narrowing of the left ventricular chamber (6). Myocyte hypertrophy and loss, with disorganization of the cellular architecture, and replacement by fibrous tissue are the main histological features in cases of HCM (7). The cat in this report did not present gross or microscopic changes in the ventricular myocardium, excluding a diagnosis of atrial dilatation caused by HCM.

The presence of left cardiomyopathy associated with extra-cardiac findings of pulmonary congestion and edema indicates that the animal died due to acute cardiopulmonary insufficiency when subjected to the stress caused by the bath. The consequences arising from anomalous chordae tendineae observed in animals, as in this case, have been described in humans with the same congenital anomaly (10,13).

According to some authors (4), ACT may be associated with endocardial fibroelastosis, a congenital heart condition often observed in cats. However, for the animal presented here, the congenital heart defect observed grossly and microscopically in addition to ACT was MD.

The main differential diagnosis of ACT is the presence of excessive moderator bands. Moderator band tissue bundles are composed of muscle and tendon and connect the septum to the ventricular free wall (5). Histologically, moderator bands are characterized by the presence of cardiac muscle fibers, blood vessels, and Purkinje fibers and are directly involved in the conduction of electrical impulses in the heart (5).

In this case, the anomalous structures that were inserted into the free wall of the left ventricle, interventricular septum, and the papillary muscles were histologically characterized by bundles of fibrous connective tissue covered by endocardium, similar to that observed in normal chordae tendineae (9).

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References

1. DARKE PG. Congenital heart disease in dogs and cats. **J. Small Anim. Pract.**, 1989, 30, 599-607.
2. DYCE KM., SACK WO., WENSING CJG. **Tratado de Anatomia Veterinária**. 3 ed. Rio de Janeiro: Elsevier, 2004: 215-55.
3. GREGORI T., GOMEZ OCHOA P., QUINTAVALLA F., MAVROPOULOU A., QUINTAVALLA C. Congenital heart defects in dogs: a double retrospective study on cases from University of Parma and University of Zaragoza. **Ann. Fac. Medic. Vet. Parma**, 2008, 28, 79-90.
4. GUERON M., COHEN W. Anomalous left ventricular chordae tendineae and pre-excitation: Unusual cause of praecordial pansystolic murmur in a baby with fibroelastosis. **Br. Heart J.**, 1972, 34, 966-8.
5. GULYAEVA AS., ROSHCHEVSKAYA IM. Morphology of moderator bands (Septomarginal Trabecula) in porcine heart ventricles. **J. Vet. Med. - Anatomia, Histologia, Embryologia**, 2012, 41, 1-9.
6. KITTLESON MD., KIENLE RD. **Small Animal Cardiovascular Medicine**. St-Louis: Mosby, 1998:347-61.
7. LIU SK., MARON BJ., TILLEY LP. Feline hypertrophic cardiomyopathy: gross anatomic and quantitative histologic features. **Am. J. Pathol.**, 1981, 102, 388-95.
8. MAZZEI V., NASSO G., ANSEMI A., SALAMONE G., MANGANO S., GRASSI R. Correction of discrete subaortic stenosis with abnormal chordae tendineae. **J. Card. Surg.**, 2006, 21, 271-3.
9. MILLINGTON-SANDERS C., MEIR A., LAWRENCE L., STOLINSKI C. Structure of chordae tendineae in the left ventricle of the human heart. **J. Anat.**, 1998, 192, 573-81.
10. ROBERTS WC. Anomalous left ventricular band. An unemphasized cause of a precordial musical murmur. **Am. J. Cardiol.**, 1969, 23, 735-8.
11. TAQATQA AS., BOKOWSKI JW., POLIMENAKOS AC. Congenital anomalous chordae tendinae of the mitral valve: an unusual presentation of mitral insufficiency in children. **J. Card. Surg.**, 2010, 25, 582-5.
12. TAUSSIG HB. World survey of the common cardiac malformations: developmental error or genetic variant? **Am. J. Cardiol.**, 1982, 50, 544-59.
13. VLASSAK I., MUMTAZ M., PETERSSON G., THOMAS JD. Accessory fibrous band causing anterior mitral valve leaflet restriction. **Ann. Thorac. Surg.**, 2002, 74, 592-3.
14. VLEET JFV., FERRANS VJ. Cardiovascular system. MC GAVIN M.D., ZACHARY J.F. **Bases da Patologia em Veterinária**. 5 ed. Mosby-Elsevier, Rio de Janeiro, 2012: 542-91.
15. WATANABE N., WADA N., YOSHIDA K. Uncommon anomalous papillary muscle/chordae tendineae incidentally found in patient with transient ischaemic attack. **J. Heart**, 2005, 9, 1074.