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PATHOLOGY

Biventricular aneurysm in a dog

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ABSTRACT: An aneurysm is defined as a localized dilation of a blood vessel or heart. It may have either a congenital or an acquired origin and occurs in both humans and animals. Cardiac aneurysms are rare, and are characterized by sacculations of the cardiac wall. The present case report describes the macroscopic and microscopic findings of an aneurysm affecting both ventricles of the heart in a dog. A 7-year-old mixed-breed female dog with osteosarcoma on the left pelvic limb was euthanized and submitted to the veterinary pathology laboratory for necropsy. Macroscopically, besides the lesions in the link, the pericardial sac was adhered to the pericardial surface of the heart and there were two sacculations in both ventricles which communicated widely with the respective ventricle. Microscopically, fibrous tissue adhered to the pericardium, atrophy and necrosis of the cardiomyocytes with replacement by adipocytes were observed in addition to hyperplasia of the tunica media of arterioles and thrombosis. A diagnosis of acquired biventricular cardiac aneurysm was made based on the necropsy findings and microscopic lesions in the heart. We emphasize the importance of performing a necropsy and examining tissues histologically for the diagnosis of this rare condition. Ventricular aneurysms should be included in the differential diagnosis of cardiovascular disease in dogs.
Key words: acquired, congenital, dilation, heart.

Aneurisma biventricular em um cão

RESUMO: O aneurisma é uma dilatação localizada em um vaso sanguíneo ou no coração. Pode ter origem congênita ou adquirida e ocorre tanto em humanos quanto em animais. Os aneurismas cardíacos são raros e caracterizados por saculações da parede cardíaca. O presente relato de caso descreve os achados macro e microscópicos de um aneurisma no coração de um cão, que afetava ambos os ventrículos. Uma cadela com sete anos de idade, diagnosticada com osteosarcoma, foi submetida a eutanásia e encaminhada ao laboratório de patologia veterinária para necropsia. Macroscopicamente, além da lesão neoplásica no membro pélvico esquerdo, o saco pericárdico estava aderido à superfície pericárdica do coração e havia duas saculações em ambos os ventrículos que se comunicavam amplamente com o respectivo ventrículo. Microscopicamente, observou-se tecido fibroso aderido ao pericárdio, atrofia e necrose dos cardiomiócitos com substituição por adipócitos, além de hiperplasia da túnica média das arteríolas e trombose. O diagnóstico de aneurisma cardíaco biventricular adquirido foi feito com base nos achados de necropsia e lesões microscópicas no coração. Ressaltamos a importância da necropsia e exame histológico para o diagnóstico dessa rara condição. Os aneurismas ventriculares devem ser incluídos no diagnóstico diferencial de doenças cardiovasculares em cães.
Palavras-chave: adquirida, congênita, coração, dilatação.

INTRODUCTION

Ventricular aneurysms are rare, poorly understood and may develop congestive heart failure. They can be congenital or acquired (JESERICH et al., 2006) and are classified as congenital when the cause of the defect is unknown or other causes are excluded (HERRÁEZ et al., 2011). Acquired ventricular aneurysms derive from areas of vascular smooth muscle weakness (HÉRNANDEZ-RAMÍREZ et al., 2017) and are very common in humans as a sequel to myocardial infarction. In domestic animals, are rare since myocardial infarction is uncommon (GUARDA, 1994). Many patients do not reveal any clinical signs (JESERICH et al., 2006; BOUJOUN et al., 2004). Thus, this study described the pathological characteristics of biventricular aneurysms in a dog.

MATERIALS AND METHODS

A female dog with a history of left pelvic limb swelling, tachypneic, fever and lateral decubitus was referred to the veterinary hospital. Euthanasia was elected due to the diagnosis of osteosarcoma, severe clinical presentation and poor prognosis, followed by necropsy. Samples of organs and tissues from the thoracic and abdominal cavities, and central nervous system were fixed in 10% buffered formalin, routinely processed, and embedded in paraffin. Sections were

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cut to approximately 5 µm thick and stained with hematoxylin and eosin (H&E).

RESULTS

A 7-years old mixed-breed female dog was referred to the veterinary hospital. Radiographs showed the presence of an intramedullary pin inserted in the femur and bone changes compatible with neoplasm. The dog was submitted to the veterinary pathology laboratory at our university for necropsy.

Macroscopically, besides the neoplastic lesion in the left pelvic limb, the pericardial sac was adhered to the epicardial surface at the level of both cardiac ventricles. The pericardial sac was removed and the heart was exposed. There were two sacculations in the cardiac wall arising from each ventricle. This dilatation measured 3 x 3 cm in the left ventricle and was located at the apex of the heart. A fibrous ring with numerous fibrillary projections arising from the pericardium was noted at its base (Figure 1). The cardiac wall was thinner at the lower part of the blind sacculation. The aneurysmal dilation in the right ventricle was located in the free wall of the ventricle immediately beneath the tricuspid valve and measured 3 x 4 cm (Figure 1AB). Its base was also surrounded by a band of fibrous connective tissue and its lumen was filled with a large currant jelly clot. The aneurysm wall was prominently dilated with marked thinning along its entire length. The section of the ventricular cardiac chamber showed a wide communication between the base of the aneurysms and the ventricular lumen. No other gross changes were observed in the heart.

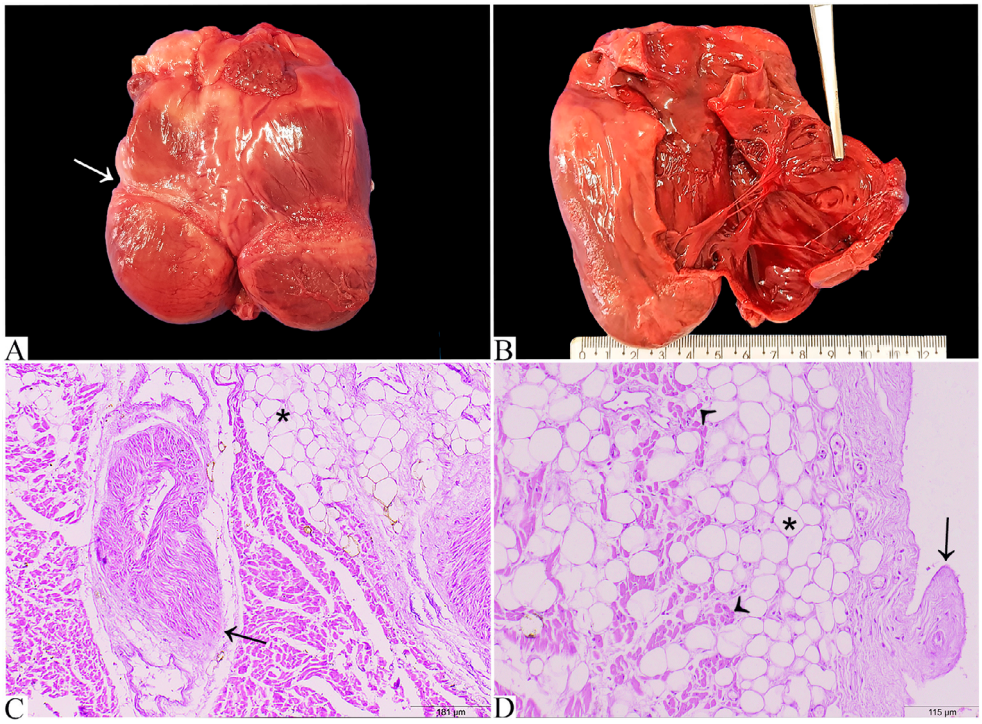

Figure 1 - Heart, biventricular aneurysm, canine, female, 7-year-old, mixed-breed. (A) A sacculation measuring 3 x 4 cm is observed in the free wall of the right ventricle, with a ring of fibrous connective tissue at the base encircling the aneurysm formation (arrow). (B) A left ventricular aneurysm is noted at the cardiac apex, measuring 3 x 3 cm, and occupies the entire length of the cardiac apex. The pericardium is covered by fibrous connective tissue and is encircled at its base by a fibrotic ring. (C) The myocardium, shows moderate hyperplasia of the tunica media of arterioles (arrow) and a moderate number of adipocytes infiltrate and replace cardiomyocytes (asterisk), HE, 8x. (D) The myocardium of the aneurysmal projection shows that a moderate number of adipocytes infiltrate (asterisk) and replace cardiomyocytes with marked atrophy (arrowhead). The pericardium shows moderately multifocal thickening due to fibroplasia. In some foci, the fibrous tissue is projected toward the lumen of the pericardial sac (arrow), HE, 12x.

Mild pulmonary edema associated with moderate diffuse hyperemia and moderate emphysema were also observed especially at the edges of the cranial lung lobes. The liver was moderately swollen with an accentuated lobular pattern. There was a chronic focal wedge-shaped cortical infarct in the right kidney.

Microscopically, there were marked atrophy and cardiomyocyte loss in the cardiac aneurysms. The remaining myocardium was infiltrated and replaced by moderate number of adipocytes (fatty replacement of the myocardium). The pericardium had locally extensive mild to moderate fibrosis. Multifocally, areas of fibroplasia were observed between the cardiomyocytes. Discrete multifocal necrosis of cardiomyocytes and mild to moderate hyperplasia of the tunica media of arterioles were also observed (Figure 1C-D). An arteriole was partially obstructed by a thrombus.

There was diffuse hepatocellular degeneration and centrilobular necrosis associated with marked congestion (nutmeg liver) and moderate cholestasis. The lungs were moderately edematous and there was mild multifocal anthracosis. Radiographic and necropsy findings raised the suspicion of malignancy affecting the left pelvic limb, which was confirmed by histopathology. A definitive diagnosis of osteoblastic osteosarcoma was made based on the typical microscopic features of this neoplasm in HE-stained sections. The gross and microscopic findings in the heart of this dog are compatible with those of an acquired biventricular aneurysm.

DISCUSSION

Ventricular aneurysms are rare and poorly understood, entities that can be congenital or acquired. Aneurysms may develop in any cardiac chamber causing congestive heart failure and can rupture resulting in cavitary effusions (including hemopericardium and hemothorax) (JESERICH et al., 2006).

Acquired ventricular aneurysms (AVA) derive from areas of vascular smooth muscle weakness secondary to ischemia, trauma or infectious and parasitic diseases including Chagas disease due to *Trypanosoma cruzi* (HÉRNANDEZ-RAMÍREZ et al., 2017). AVAs are very common in humans as a sequel to myocardial infarction secondary to atherosclerosis (GUARDA, 1994; GAL et al., 2012). In the present case, the affected myocardial segment is replaced by scar tissue forming a "*loci minoris resistentiae*" (locus of lower resistance), and intraventricular tension thins and stretches out the infarcted heart muscle, which is unable to contract. As a result, there is an expansion of the infarcted area of the myocardium. A thin and weakened layer of muscle forms and grows gradually at each systole (BIASATO et al., 2017). AVAs are even rarer in domestic animals, as myocardial infarction is a relatively rare event (MILLER & GAL., 2017). Lesions of verrucous endocarditis may cause thromboembolism in the coronary arteries resulting in myocardial infarction (GUARDA, 1994). In our case, no endocarditis lesions were observed, but AVA must be considered due to age and a primary osseous tumour. The possibility that osteosarcoma cell emboli or vascular thrombus formed by the tumor growth dislodged from the pelvic limb can be considered as a cause of vascular occlusion resulting in myocardial ischemia, as the kidney also had a chronic infarct.

Cardiac aneurysms are classified as congenital when the cause of the defect is unknown and are extremely rare in animals. In humans, congenital ventricular aneurysms (CVA) occur with a frequency of 0.5 per 100,000 births (HERRÁEZ et al., 2011) and usually affect the left ventricle (JESERICH et al., 2006). Although intrauterine viral infections or coronary vascular abnormalities have been considered as possible causes in some of these cases, most are attributed to intrinsic wall defects that may occur during embryogenesis (JESERICH et al., 2006; MARIJON et al., 2006), similar to those already described in animals with the same condition (GAL et al., 2012; GUARDA et al., 2004).

The terms CVA and congenital ventricular diverticulum (CVD), also referred to as pseudoaneurysm (BOUJON et al., 2004), have been used interchangeably over the years. CVDs are known to be characterized by a close connection to the cardiac chamber and consist of a dilated muscle wall containing all three layers of the heart that present synchronous contractility with the myocardium. Histologically, fibrosis and other hallmarks of tissue injury are absent. CVD is seen at the apex of the heart and is usually associated with other cardiac or extracardiac malformations (MARIJON et al., 2006). In the present case, CVD was ruled out due to the presence of fibrous connective tissue in the aneurysm wall and its wide communication between the ventricles and the aneurysm sac.

A CVA is very similar to an AVA, as it has wide communication with the adjacent ventricle, as well as dyskinesia due to the replacement of the muscle wall by fibrous connective tissue.

Microscopically, fibroplasia may be subendocardial or transmural (MARIJON et al., 2006, GUARDA et al., 2004). Other lesions including replacement of cardiomyocytes by adipose tissue and atrophy and loss of heart muscle fibers have also been reported in this disorder (HÉRNANDEZ-RAMÍREZ et al., 2017; BOUJOUN et al., 2004). These lesions were present in our case. In humans, CVAs may cause clinical signs even during childhood and AVA at a later stage in life, as the primary cause in most cases is atherosclerosis (MARIJON et al., 2006; ZHAI et al., 2013).

Interestingly, histopathology showed arteriosclerosis affecting cardiac arterioles characterized by smooth muscle hyperplasia of the tunica media and the presence of a thrombus partially occluding the lumen of the blood vessel. Arteriosclerosis is a non-specific lesion that affects the arteries and results in the decrease or loss of blood vessel wall elasticity. The condition involves small arteries and has been reported as an important cause of cardiac ischemia in dogs and frequently results in myocardial infarction (WILLIANS, 2003). The presence of this lesion associated with other factors, including the hypothesis of tumor emboli and the patient's advanced age with no clinical signs of chronic heart disease suggests, that the observed aneurysms are is acquired and not congenital.

Ventricular aneurysms can be diagnosed by echocardiography and magnetic resonance imaging. The latter ancillary test may show areas of fibrosis along the aneurysmal dilatation allowing the differentiation between AVA, CVD, and CVA. Electrocardiography is not a useful tool in this clinical scenario as it yields nonspecific changes that do not contribute to the diagnosis of this entity (MARIJON et al., 2006). Many patients do not reveal any clinical signs until the aneurysm reaches large proportions (GUARDA et al., 2004; JESERICH et al., 2006; BOUJOUN et al., 2004). In the present case, the AVA defined was an incidental necropsy finding. An ante mortem diagnosis was not achieved due to a lack of adequate clinical monitoring and appropriate follow-up of the patient and the fact that the owner declined any further diagnostic testing due to financial constraints.

Although clinical signs suggestive of heart disease were not detected during the physical examination of this dog, , the histopathological examination showed hepatocellular congestion and centrilobular necrosis, lesions compatible with right congestive heart insufficiency. Based on these microscopic findings in the liver, we may infer that this animal developed right-sided heart failure due to the presence of an aneurysm in the right ventricle. The wall of the right ventricle is much thinner than that of the left ventricle. Therefore, blood pressure leads to significant distension of the cardiac muscle of the right ventricle causing greater damage to the blood flow on the right side of the heart. It can be evidenced by the absence of hemosiderin-laden macrophages (heart failure cells) in the alveoli, which is a typical microscopic finding in cases of left-sided or congestive heart failure. In our case, Prussian blue-stained lung sections were negative for iron.

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DECLARATION OF CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS' CONTRIBUTIONS

All authors contributed equally to the design and writing of this manuscript. All authors critically reviewed the manuscript and approved the final version.

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