LEFT SIDED CONGESTIVE HEART FAILURE DUE TO SEVERE AORTIC DEGENERATION AND INSUFFICIENCY IN A DOG – A CASE REPORT

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Abstract

Aortic degeneration is an uncommon finding in dogs with uncertain etiology, being associated with endocrine disorders which may induce hypercalcemia, hyperkalemia, or with toxicosis, renal disorders and endocarditis. This entity may develop a diastolic volume overload leading to left sided congestive heart failure. The aim of this study is to report and discuss a case of a dog with left sided congestive heart failure due to severe aortic regurgitation associated with myxomatous mitral valve disease and systemic hypertension.

Case presentation

A 16 years old mix breed male dog, weighting 25 kg was referred to the Cardiology Service of the Veterinary Teaching Hospital of Iași with a history of breathlessness, tachypnea, weight and appetite loss for 3 days. Physical examination revealed decreased body condition score, pale mucous membranes, increased respiratory rate and effort and orthopneic position. Lung auscultation revealed wet crackles on both lungs and cardiac auscultation revealed a left apical systolic plateau murmur of 5th grade and a basal left sided diastolic murmur. The rectal temperature was within normal limits.

A five minutes six-lead electrocardiography was performed with the dedicated PolySpectrum 8V ECG device with the dog in sternal recumbence. The ECG trace revealed a sinus rhythm with a HR of 125 bpm, presence of respiratory arrhythmia and 7 left ventricular premature complexes, organized in 2 single VPCs, one couplet and one triplet (fig. 1 A and C). Also, a run of accelerated idioventricular rhythm of four right ventricular complexes with a mean HR of 120 bpm was observed. A left lateral thoracic radiography revealed left sided cardiomegaly with dorsal displacement of the trachea and a perihilar interstitial and alveolar pattern extending into the diaphragmatic lung lobes was observed (fig. 1 B). The diagnosis of a cardiac disease with pulmonary edema was suspected.

Due to the severe condition of the dog, the complete cardiological examination was postponed and oxygen therapy and furosemide were administered as emergency. Diuretic therapy with furosemide 2mg/kg P.O. BID was recommended and recheck was scheduled after 7 days.

At recheck, one week later, the dog was alert, pink mucous membranes, the respiratory effort decreased and cardiac auscultation revealed a left apical systolic plateau murmur of 5th grade and a basal left sided diastolic murmur. Due to the improved general condition, the patient was subjected to a complete cardiological examination consisting of five minutes six leads electrocardiography, thoracic radiography, blood pressure measurement with a veterinary oscillometric device (Vet HDO) and echocardiography (General Electric, Logiq V5) as previously described (Thomas, Gaber et al. 1993; Egner, Carr et al. 2003; Thrall 2013; Tilley and Smith Jr 2016).

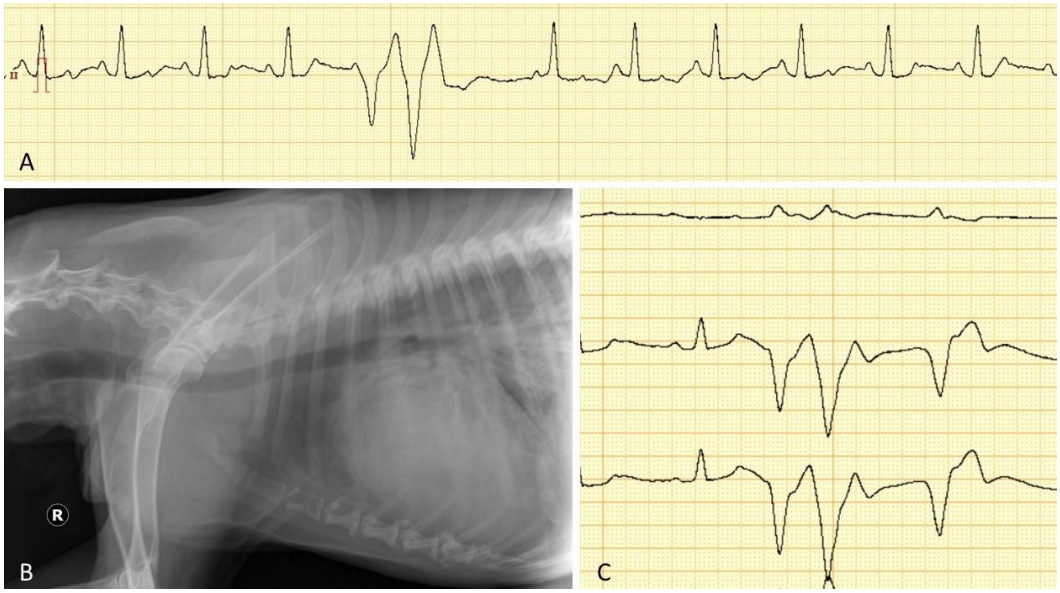

Figure 1 A. Lead II electrocardiography (10 mm/mV, 50 mm/sec) of a dog with severe respiratory distress: sinus rhythm with a coulplet of left ventricular premature complexes visible after the 4th complex; B. right lateral thoracic radiography of the same dog: cardiac enlargement with dorsal displacement of the trachea and a patchy alveolar pulmonary pattern is visible around the perihilar area extended into the caudal lung lobes; C – electrocardiographic trace of the same dog – a triplet composed of 3 successive ventricular complexes is seen;

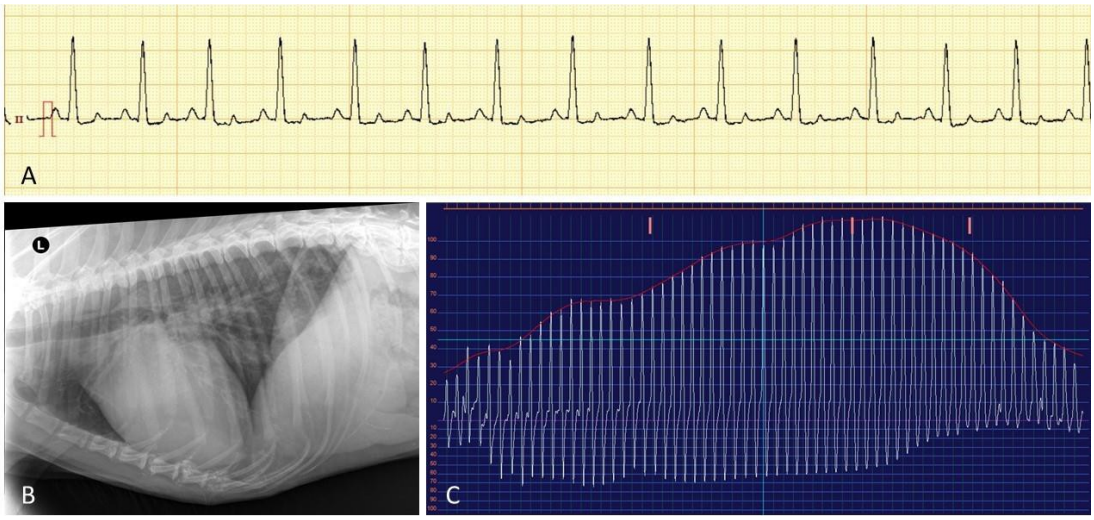

Figure 2. A. Lead 2 electrocardiography (10 mm/mV, 50 mm/sec) of the same dog, one week after oral furosemide monotherapy – HR – 145 bpm, sinus rhythm, absent respiratory arrhythmia and no ventricular premature complexes were present on 5 minutes recording; B. left lateral thoracic radiography of the dog after one week of oral furosemide monotherapy – an improvement on the pulmonary pattern is visible, with resolution of the pulmonary edema. The cardiac enlargement is still present; C. Pulsatile wave graphic of the arterial blood pressure measurement – note the increase in presystolic waves – consistent with presystolic arterial hypertension.

Electrocardiography revealed a sinus rhythm with a heart rate of 145 bpm, with the presence of respiratory arrhythmia, normal mean electrical axis and no ventricular premature complexes on 5 minutes recording (fig.2 A). The blood pressure was measure at the tail with a D2 cuff and the mean value of five measurements was 143/56 (systolic/diastolic pressure) (fig. 2 C). Echocardiography revealed left ventricular eccentric hypertrophy in systole (LVIDs = 26.9 mm; LVIDsn = 1.28) and diastole (LVIDd = 54.6 mm; LVIDdn = 2.11) and atrial dilatation (La/Ao = 1.97). The mitral valve was hyperechoic and thickened, with a mitral regurgitating jet of 4.96 m/s and a pressure gradient of 98.57 mmHg (fig. 3 C). The aortic valve was thickened with hyperechoic aspect and abnormal movement of the leaflets and a diastolic regurgitating jet was present with a velocity of 4.67 m/s and a pressure gradient of 87.13 mmHg (fig. 3 A, B and D). The systolic function of the left ventricle was increased with a shortening fraction of 50.7% and an ejection fraction of 81.5%. The trans-mitral flow interrogation revealed an impaired relaxation pattern with E-wave velocity of 0.7 m/s and deceleration time of 63 milliseconds and a A-wave velocity of 1.11 m/s. The E/A ratio was 0.64. The pulmonary flow was laminar within normal ranges. Subjective evaluation of the right atria and ventricle did not reveal any changes. There were no signs of pulmonary hypertension.

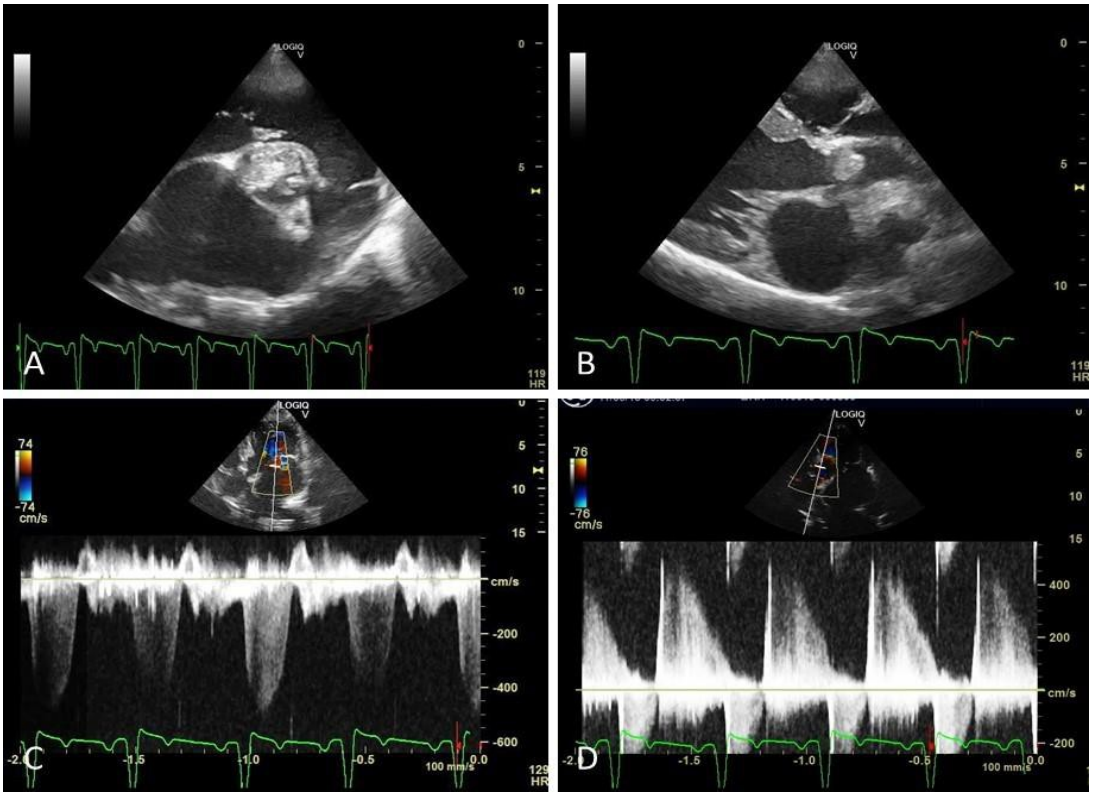

Figure 3 Echocardiography of a dog after one week of therapy with furosemide monotherapy for pulmonary edema: A. Right parasternal shot axis view at the heart base – not the increase in the left atrium and the pulmonary veins and the hyperechoic aspect of the closed aortic valves; B. Right parasternal long axis five chamber view – the aortic valve is thickened and hyperechoic with an abnormal position; C. Continuous Doppler interrogation ofthe mitral regurgitating jet, measuring a maximum velocity of 4.96 m/s with a pressure gradient of 98.57 mmHg; D. Continuous Doppler interrogation of the aortic valve over the regurgitating jet, measuring a maximum velocity of 4.67 m/s with a maximum pressure gradient of mmHg.

Thoracic radiography revealed increased cardiac size with a VHS of 11.9v, dorsal displacement of the trachea and a diffuse interstitial pattern extended into the caudal lung lobes. No alveolar pattern was visible as compared to the thoracic radiography performed one week previously (fig. 2 B). Moreover, severe spondylosis was observed between T12-L3 and L3-S1. Cardiological diagnosis was aortic degeneration associated with myxomatous mitral valve disease and left sided congestive heart failure. Also, the dog was diagnosed with severe spondylosis and arterial hypertension. Therapy was assigned and the dog was released home. One month later the owner requested euthanasia because of the severe pain and ataxia due to spondylosis and necropsy was performed.

Gross pathology of the heart revealed left ventricle and atrial dilation, myxomatous degeneration of the mitral valve and severe degenerative lesions of the aortic leaflets (fig. 4 A and B).

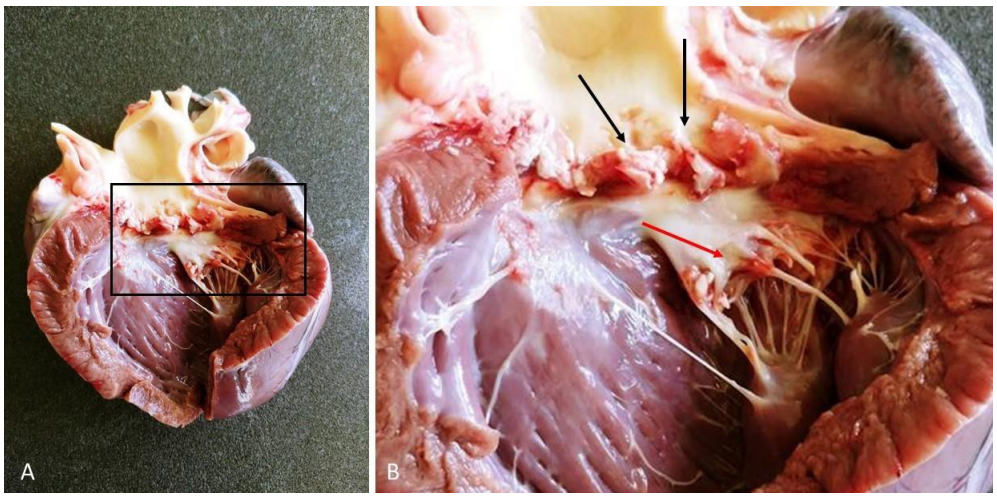

Figure 4 A. Gross pathology of the heart showing left ventricular eccentric hypertrophy; B. Magnification of the same image focusing on the aortic (black arrow) and mitral valve (redarrow) which appear thickened and degenerated.

Histopathological examination revealed mucoid degeneration of the aortic leaflets expressed by the thickening of the leaflets showing a polypoid aspect, defragmentation of the fibrillar structures (elastin and collagen fibers) and increased accumulation of fundamental substance. Aortic wall examination revealed a significant thickening of the intimal layer, lipid accumulation and acid mucopolysaccharides deposits into the media layer, between the leiocytes, with a fibrillar aspect, showing mild basophil coloration. The accumulation of mucopolysaccharides induced a dissociation of the media layer and fragmentation of the myocytes. Aortic calcification was observed, with intimal localization, expressed through the basal layer and endothelial cell mineralization (fig. 5).

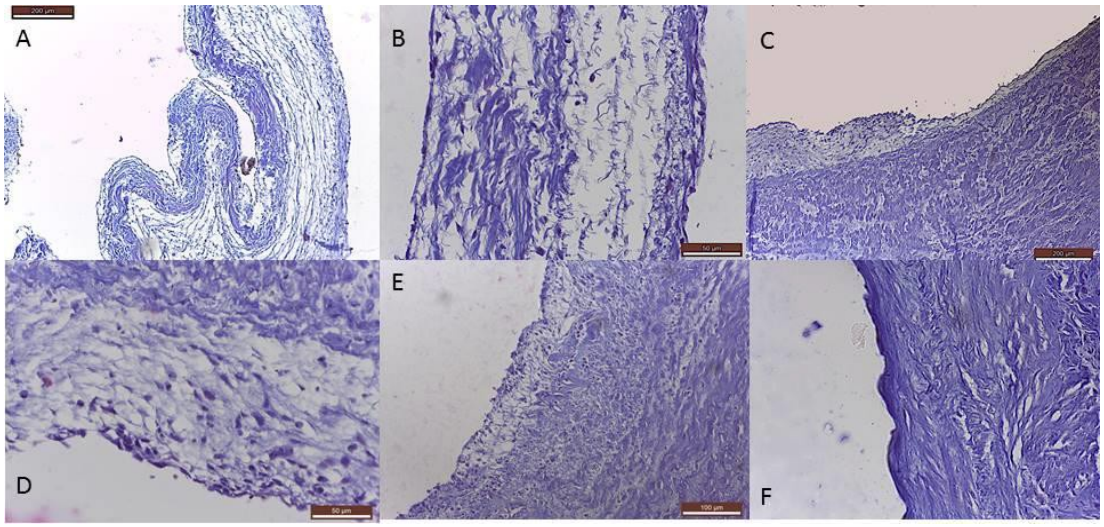

Figure 5 Histopathological images of the lesions: A. Histologic image of the aortic valve – section through proliferation, with mucoid degeneration and fibrosis B. Section through the aortic valve – the fragmentation of the elastic and collagenous fibers within the valvular structure is visible; C. Section through the aortic wall – intimal lipidosis inducing an irregular aspect of the aortic lumen, also note the mucous degeneration of the media layer, hyperproduction of proteoglycans, inducing disorganization of the muscular layer; D. Section through the aortic wall – intimal lipidosis; E. Section through the aortic wall – subendothelial lipid accumulation is visible and degeneration of the media and intimal layers though the synthesis of mucopolysaccharides by the myofibroblasts; F. Section through the aortic wall – Intimal calcification expressed through the mineralization of the basal membrane and the endothelial cells; Trichrome Masson;

Discussions

This paper describes a case of a dog with myxomatous mitral valve disease and severe degeneration of the aortic valve which induced hemodynamic changes and left sided congestive heart failure. The etiology of the aortic degeneration and mineralization is uncertain, however multiple factor are incriminated such as endocrine disorders that may imbalance the calcium concentration in the body, toxics, chronic kidney failure or chronic endocarditis. Endocarditis had been reported, in dogs, with prevalence of 0.9-6.6% (Macdonald 2010), multiple pathogens being involved such as *Staphylococcus spp.*, *Streptococcus spp.*, *Escherichia coli*, *Pseudomonas spp.*, or *Bartonella* (Calvert 1982; Sisson and Thomas 1984; Kelly, Rolain et al. 2006; Sykes, Kittleson et al. 2006; Sykes, Kittleson et al. 2006). Like any inflammatory process, the chronic phase induces fibrosis of the affected tissue and changes in the organ architecture. These structural changes will develop a coaptation deficit of the aortic valve leaflets allowing the blood to return into the left ventricle during the diastole. The early filling of the left ventricle will lead to volume overload and eccentric hypertrophy. These hemodynamic changes will worsen the myxomatous valve disease progression by increasing the amount of blood regurgitating into the left atrium and by filling the left ventricle in early diastole and increasing the pressure during late diastole, leaving the left atrial cavity with a residual volume. These mechanisms together, worsen the left atrial volume and pressure overload leading to dilatation and pulmonary capillary wedge pressure and finally left sided congestive heart failure. Cardiogenic pulmonary edema develops once the left ventricular end-diastolic pressure exceeds 20-25 mmHg (Guyton and Lindsey 1959).

The development of endocarditis and degenerative valvular lesions must have underlying

factors such as bacteriemia and disruption of the endothelial tissue. The most common predisposing factors in dogs are subaortic stenosis which create a turbulent flow. However, the patient presented in this paper did not show any echocardiographic signs of subaortic stenosis, but a mild obstruction of the aortic annulus cannot be ruled out. Other common factors that predispose to bacteriemia is discospondylitis. Our patient had severe spondylosis, which may be possibly developed secondary to intervertebral disc infection.

The presence of the ventricular premature complexes was found only in the first visit, when the dog was dyspneic and had pulmonary edema, however, these complexes disappeared after the pulmonary edema was treated. It is known that both myocardial fibrosis and hypoxia may induce ventricular premature complexes. The mechanism behind the generation of ectopic beats is unidirectional block inside a region with diffuse fibrosis and hypoxia, where micro-reentries are formed (Sachetto, Alonso et al. 2018) and also hypoxemia and respiratory acidosis during prolonged pulmonary edema may trigger abnormal automaticity (Hansel, Solleder et al. 2009).

From the anatomopathological point of view, the intimal lipidosis is the promoter for the aortic ateromatosis, the lipid infiltration inducing lately transformation of the myocytes into fibroblasts, synthesis of the collagen fibers and incorporation of the lipids into the arterial wall. The two dystrophic processes coexist inside the aortic wall. On the other side, these changes lead to arteriosclerosis of the arterial wall. Lipid deposit and proteoglycans synthesis by the myofibroblasts lead to compression of the myocytes and diffuse parietal sclerosis.

Mineralization of the aortic wall was observed at the anatomopathological examination. Causes of the arterial medial mineralization are reported to be associated with carcinogenic plant toxicosis, hypothyroidism, hypercholesterolemia, hypercalcemia, vitamin D toxicosis, primary hyperparathyroidism, and chronic kidney failure with secondary hyperparathyroidism. One study reported the radiographic features of the aortic mineralization in 20 dogs without any clinical signs linked to the radiographic findings. However, these changes have been associated with older dogs, and 13 of 20 had signs related to neoplasia (Douglass, Berry et al. 2003). Another study reported a prevalence of aortic and cardiac mineralization seen on radiography of 0.61%. Similarly, this entity was overrepresented in geriatric dogs and also in Rottweilers. The authors suggested that this pathology may be either age-related degenerative process or chronic disease-related process (Schwarz, Sullivan et al. 2002).

Finally, the cause of the aortic valve degeneration is unclear, however, the hemodynamic changes can be well documented by echocardiography and therapy can be assigned to ensure a better quality of life.

Conclusions

Aortic degeneration may be clinically suspected by the diastolic basal murmur and confirmed through echocardiographic examination. Due to the chronic changes of the valvular structure, the therapy aims to restore the hemodynamic changes and prevent the onset of left sided congestive heart failure.

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