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Case Report

Mitral Valve Degeneration in a Saint Bernard Dog-A Case Report

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Abstract

A 7-year-old male Saint Bernard dog weighing 65 kg was presented to the Veterinary Clinical Complex, College of Veterinary Science, Rajendranagar with the history of exercise intolerance, nocturnal cough, dyspnoea at rest in the past one month. On visual examination, the animal assumed abducted elbow posture while standing and sternal recumbency at rest. On clinical examination, there was increase in heart rate and auscultation of the heart revealed cardiac murmurs and further subjected to radiographic examination revealed enlarged left atrium and change in the cardiac silhouette and the lungs were normal; T-Fast revealed increased turbulence and systolic dysfunction and electrocardiographic examination revealed atrial fibrillation and stage two AV block. Further treatment was successfully managed with ACE inhibitors, inodilators, diuretics and antioxidants for about 15 days. On day 8 slight improvement was noticed in cough and on day 15 improvement was observed in exercise intolerance and dyspnoea.

Keywords: Nocturnal Cough; Systolic Dysfunction; Mitral Valve Regurgitation; Mitral Valve Disease; ACE Inhibitors

Introduction

Myxomatous degenerative valvular disease is the most common cardiac disease in dogs [11]. It accounts for about 40% of the cardiovascular diseases in dogs [2]. 58% of the mitral valve degeneration (MVD) cases are due to age related conditions [11]. The degeneration mostly affects the mitral valves, but the tricuspid valves can also be affected. The exact mechanisms of spontaneous MVD are unclear. It is inherited in some dog breeds. Recent evidence suggests a possible role for vasoactive peptide endothelin in the pathogenesis of MVD [8]. Repeated mechanical stress on mitral valve leaflets is also thought to be a cause of myxomatous degeneration. The degenerative mitral valve leaflets had a greater density of endothelin receptors. Furthermore, the density of endothelin receptors was related to the severity of MVD. The pathological changes of MVD include thickened redundant leaflets characterized by glycosaminoglycan deposition, collagen bundle disorganisation and elastic fiber fragmentation [5]. The chordae tendineae are commonly elongated and occasionally rupture [3,6,7]. The clinical signs of dogs affected with MVD include exercise intolerance, nocturnal cough, dyspnoea at rest. The present case report illustrated the diagnosis, treatment and management of MVD in a male Saint Bernard dog.

Case history and clinical examination

A 7-year-old male Saint Bernard dog weighing 65 kg was presented to the Veterinary Clinical Complex, Rajendranagar with the history of exercise intolerance, nocturnal cough, dyspnoea at rest in the past one month. Visual inspection of the dog revealed abducted elbow posture while standing and was in sternal recumbency at rest. On clinical examination the temperature was

recorded as 102^0F, conjunctival and buccal mucus membranes were pink & moist and skin tenting time and capillary refilling time were about 1-2 seconds. On auscultation, the heartbeat was measured to be about 80 beats per minute and slight cardiac murmurs were felt and the murmurs were louder near the apex of the heart. Based on the above findings, the case was suspected to be of cardiac origin and further diagnostic techniques were applied to confirm the case.

Diagnosis

Diagnosis was done based on the radiographic examination, electrocardiogram and T-Fast. Radiographic examination revealed enlarged left atrium and the cardiac silhouette was markedly enlarged in the lateral view of thorax whereas the lungs were normal in the radiograph (Figure 1). Electrocardiogram revealed atrial fibrillation and stage 2 AV block which may be indicative of mitral valve degeneration; there was peaked and hyperacute T wave which indicates myocardial hypoxia and hypokalaemia (Figure 2). T-Fast revealed systolic dysfunction, extreme thickening of mitral valve leaflets and concave inter ventricular septum in B mode (Figure 3). Colour doppler revealed increased turbulence and regurgitation (Figure 4). M mode revealed the ejection fraction to be 32% and fractional shortening to be 20%. All these findings confirmed the case as degenerative mitral valve disease or mitral valve degeneration (MVD).

Treatment and management

The dog was treated with Ramipril (Tab. Cardiopet - 2.5mg), Pimobendan (Tab. Pimocard - 5mg), Furosemide + Spironolactone (Tab. Lasilactone - 50mg), Antioxident (Capsule CoQ-10 - 30mg) for about 15 days, OID, PO. The owner was advised to give a low sodium diet and reduce aggressive exercises for the dog. These drugs worked out effectively and slight improvement started to be noticed in the condition of the animal from day 8 post treatment showing slight reduction in the symptoms such as cough and on day 15 reduction in exercise intolerance and dyspnoea. On day 21, another T-Fast was performed wherein the ejection fraction and fractional shortening were also improved. The medication was continued further daily and the dog is now in good health.

Discussion

One of the most common cardiac diseases in dogs is myxomatous degenerative valvular disease. Dogs with MVD cough at night, have resting dyspnea, and are intolerant to exercise.

The typical cough is dry and more common at night, after physical activity, or during stressful situations. Therefore, diagnosis of MVD should be performed carefully to rule out the exact underlying cause of cough [10]. Notable finding: mitral valve regurgitation is the source of the loudest systolic murmur at the left thoracic apex. If MVD is present, thoracic radiography is the most crucial diagnostic method. However, the cardiac profile is normal in the early stages of MVD; the cardiac silhouette enlarges if mitral regurgitation occurs and has a clinical effect. When it comes to measuring the dimensions of the heart chambers, the ECG is an unreliable tool. Arrhythmias may make it more difficult to diagnose MVD. On rare occasions, atrial fibrillation may occur; this condition often signifies a significant illness with evident atrial dilatation. The mitral valve will be thickened considerably. Assessment of severity of mitral regurgitation can be evaluated quantitatively or semi quantitatively by doppler echocardiography [9]. Myxomatous degeneration essentially has no known cure or medication that can stop its progression. ACE inhibitors delay the development of CHF. These drugs give cardioprotective therapy to postpone CHF [4]. ACE inhibitors delay the progression of CHF. Pimobendane is an Inodilator, calcium sensitizer, and inhibitor of phosphodiesterase 3 that produces vasodilation. Furosemide is a loop diuretic that reduces left ventricular filling pressures, intravascular volume, and edema resolution. Furosemide with ACE inhibitors is a common treatment for advanced DMVD in dogs. Many variables affect the prognosis when it comes to MVD development. In most cases, CHF is a fatal condition once it manifests. Survival is often defined in months, with 8-14 months being the median time frame, even with palliative medical therapy [1].

Conclusion

Canine MVD is the most commonly diagnosed acquired cardiac disease in dogs. Despite being a prevalent disorder that can be encountered in veterinary institutions, the cause of MVD remains unknown. Treating the irregularities in circulation caused by regurgitation of the mitral valve, as well as extending survival time and improving quality of life, have been the goals. A greater knowledge of the pathophysiology of valve degeneration will direct a therapy plan that addresses the lesions themselves and stops the degenerative development, rather than managing the ensuing circulatory problems. This may contribute to the longer and higher quality of life of dogs suffering with MVD.

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