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Case Report

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Myocarditis Secondary to Polymyositis in a Dog.

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Abstract

Case Description: An eight-year-old, female, crossbreed dog was referred for investigation of weakness and gait disturbances. Immune-mediated polymyositis was diagnosed in muscle biopsy. The dog was treated with prednisone, azathioprine and mycophenolate. One year later, the dog suffered from congestive heart failure as a consequence of a suspected myocarditis. After six months the dog was euthanized. At post-mortem examination, the heart histopathological examination confirms a myocarditis with a mononuclear cell infiltration and areas of fibrosis, compatible with myocarditis secondary to polymyositis.

To the authors' knowledge, the information reported here provides the second description of myocarditis in dogs with polymyositis, and this association between polymyositis and myocarditis is well recognized in humans.

Clinical Report

An eight-year-old, female, non neutered crossbreed dog was referred to the Veterinary Hospital of University of Cordoba with a year history of episodic weakness and tetraparesia. The patient was treated by its veterinarian with hepatic protectors and gastroenteric food due to elevation of transaminases.

On physical examination, the dog suffered from generalized muscle atrophy, including masseteric and temporomandibular muscle, weighed 6.9 kg with a 2 over 9 in the body condition score, and weakness. The rest of the physiologic parameters were regular. On neurological examination, the dog had ambulatory tetraparesis to a greater degree of weakness in forelimbs and short-strided gait. The postural reactions was abnormal as a consequence of weakness, with a greater level of difficulty in hopping and wheelbarrowing. Finally, the examination of spinal reflexes and cranial nerves was normal.

Haematological examination had no significant abnormalities. Serum biochemical abnormalities include elevated alanine aminotransferase (368 U/L, reference 15-58 U/L), aspartate aminotransferase (273 U/L, reference 16-43 U/L), and creatine kinase (7806 U/L, reference 40-360 U/L), with an unremarkable fasting (1µmol/L, reference 0, 1-10 µmol/L) and post-prandial (2,6 µmol/L, reference <25 µmol/L) bile acids. Furthermore, investigation of abdomen by radiographs and ultrasonography was unremarkable. Urinalysis was unremarkable. Based on the factors listed above, a myopathy was suspected.

The dog was anesthetized for a muscle biopsy. Biopsies were collected from both biceps by an open procedure.

Variability in muscle fibre size, myofibre atrophy and degeneration, and multifocal areas of mixed mononuclear cell infiltration were present in the muscle biopsies. Infiltration cells were composed

of lymphocytes, macrophages, plasma cells and mastocytes. A little number of eosinophils were observed. In addition, perimysial and endomysial fibrosis and evidence of cell regeneration were observed. Organisms were not identified within any of the muscle sections. The histopathological diagnosis was a generalized inflammatory myopathy with perimysial and endomysial fibrosis.

Additionally, further investigation was undertaken for possible causes of the myositis. ELISA technology testing (SNAP 4Dx Plus Test Idexx) for vector-borne infections endemic in Spain (Heartworm disease, Anaplasma phagocytophilum, Anaplasma platys, Borrelia burgdorferi, Ehrlichia canis and Ehirlichia ewingii) was negative. Serology indicated neither any exposure to Leishmania infantum, Toxoplasma species nor to Neospora species. In the absence of any other underlying disease, immune-mediated polymyositis was suspected.

The dog was treated with 2 mg/kg prednisone orally twice daily. The dog got better but still showed resistance to movement, so one month later azathioprine was added to her treatment (2 mg/Kg orally q24h). After five months, mycophenolate was added to her treatment reaching a significant clinical response.

One year later, the dog was attended with a decompensated heart failure. On physical examination, the dog had an elevated heart rate, a systolic heart mumur grade IV/VI and the auscultation of the lung fields revealed increased bronchovesicular sounds and crackles. The findings in thoracic radiographs were a significant generalized enlargement of the heart and perihiliar and caudodorsal alveolar pattern. Echocardiography showed a systolic and diastolic dysfunction, with a right atrial and left ventricular dilation. The thickness of the myocardial walls was decreased. This findings were compatible with myocarditis or end stage of degenerative mitral valve disease. Finally, electrocardiography showed sinus tachycardia.

The dog was hospitalized for 24 hours for supportive care with furosemide and pimobendan. After the resolution of pulmonary edema, furosemide, pimobendane and benazepril were added to its treatment. Approximately 6 months after, the dog presented a severe decompensation of the heart disease and euthanasia was performed. Samples were collected from the heart, quadriceps, masseter and gluteus for microscopic examination according to standard operation procedures: haematoxylin and eosin (H&E) and Pikrosiriusred.

At necropsy, the animal showed a marked bilateral cardiac. Papillary muscles were mildly atrophic and there was a marked diffuse mitral valve myxomatous degeneration, affecting both leaflets and chordae tendineae.

Histologically, lesions in the heart were multifocal to diffuse and polyphasic consisting of: marked myofiber atrophy with severe fiber size variation, multifocal myofiber necrosis with myofibrillar disarrangement and mineralization and severe endomysial and perimysial fibrosis. Intercellular spaces were markedly expanded by numerous fibroblasts immersed in a poorly organized collagenous matrix as well as numerous lymphocytes, plasma cells and rarer macrophages and eosinophils. Lipomatosis cordis, with infiltration of mature adipocytes in-between atrophic angulated myofibers, was also found in subendocardial areas. Mitral valve showed a marked chronic degeneration with myxoid deposits and mineralized necrotic foci.

Skeletal muscles showed similar findings to the heart, joined by regenerative features (activated satellite cells, internalized nuclei, myotube formation). Myopathic atrophy was also marked, with severe fiber size variation, rarer hypertrophy and common fiber splitting and myofibrillar disarray. Both interstitial fibrosis and mononuclear infiltrates were also marked. Lipomatosis was multifocally seen, expanding the perimysium. No specific cytoarchitectural myofiber change was observed and abnormalities were found in both fiber types. No parasitic cysts or evident primary agents could be demonstrated in any sample.

Pathological diagnosis was concurrent with immune-mediated polymyositis accompanied by generalized myocarditis.

Discussion

Inflammatory myopathies refer to a heterogeneous group of disorders characterized by non-suppurative cellular infiltration of skeletal muscle (Engel A. et al, 1994). In dogs, inflammatory myopathies can be classified as focal or generalized based on clinical presentation and muscle biopsy specimens. Generalized inflammatory myopathies have been associated with infectious agents (protozoa [1-8], rickettsia [9,10], and other bacteria [11]), neoplasia (lymphoma [12], round cell tumors13, other malignancies [14,15]) or idiopathic immune-mediated disorder [16].

In the dog, polymyositis seems to have a predilection for a larger breed and mature to older dogs, and Boxers and Newfoundlands are overrepresented [17]. The clinical signs are variable, they may

wax and wane initially. Common clinical signs include generalized weakness, a stiff-stilted gait, generalized and progressive muscle atrophy including the muscles of mastication, and esophageal and pharyngeal weakness [18]. The diagnosis confirms three or more of the following criteria: clinical signs, elevation of CK, abnormal EMG with a normal motor nerve conduction study, negative autoimmune and infectious disease antibody titers, and inflammatory muscle biopsy [16].

In our case, the clinical presentation, diagnostic criteria and histopathological findings were typical of canine polymyositis. The lack of response to corticosteroid therapy could be explained because the response is variable in patients and is usually dependent on the time between the onset of clinical and the diagnosis of the disease [18]. This time in this case was older than one year, and the extensive fiber loss and fibrosis make the clinical outcome not satisfactory.

Canine inflammatory myopathies are good parallel disorders to human inflammatory myopathies, providing insights into the human conditions [19]. The clinical signs, histopathological changes and populations of infiltrating cells are similar between canine and human polymyositis [20].

In humans, the frequency of heart involvement in patients with polymyositis varies between 9% and 72% [21], and the contrary happens in veterinary medicine, with only one case report published [22] despite the similarity between human and dog polymyositis.

Clinically manifest heart problems are infrequent in humans, and the most frequently reported clinically manifest of cardiac involvement is congestive heart failure. In addition, heart disease may occur at any phase, even when polymyositis is in remission [21]. In addition, the histopathological findings of the myocarditis are similar to the inflammation in the skeletal muscle with myocardial inflammatory, degenerative changes and necrosis, which indicate that the heart is a target organ of polymyositis in humans [21]. In this case, congestive heart failure was the main sign of the cardiac disease, and there are similar histopathological findings between heart and muscle samples.

According to the above, and given the similarities between dogs and people, it is possible that the incidence of myocarditis in dogs with polymyositis is greater than that described in veterinary literature.

In conclusion, nowadays the evidence of concurrent polymyositis and myocarditis in dogs could be underdiagnosed and could be interesting to include cardiac evaluation in dogs with polymyositis in order to study the real prevalence of this condition.

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