

AN UNUSUAL CASE OF CHRONIC MASTICATORY MUSCLE MYOSITIS (MMM) IN A MONGREL

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Abstract: A three-year-old, unspayed mongrel was presented to Small Animal Medicine unit, TVCC, River with a history of inability to open the mouth, not taking feed or water, drooling of saliva, and dullness for the past 5 days. Upon clinical observation and laboratory examination the condition was diagnosed as chronic Masticatory Muscle Myositis (MMM) and was treated with immunosuppressive doses of corticosteroids and supportive fluid therapy was given. Thus, an unusual case of Chronic Masticatory Muscle Myositis is discussed here.

Keywords: Myositis, Trismus, Prednisolone.

Introduction

Masticatory muscle myositis is an autoimmune, focal inflammatory myopathy with clinical signs restricted to the muscles of mastication. The muscle fibers of the masticator muscles have a distinct embryological origin, which explains the presence of fibers called type 2M. These fibers enclose a specific myosin, which is specifically involved in this immune-mediated reaction. This myopathy affects all dogs regardless of age, gender or breeds (Ettinger, 2010).

Case History and Observation

A female, unspayed mongrel aged about three years, 10 Kg body weight was presented to Small Animal Medicine Unit, TVCC, RIVER, with the history of ptyalism, locked jaw and unable to take food or water for the past 5 days. Clinical examination revealed weak and emaciated body condition, dehydrated, sunken eye balls, pink conjunctival mucosa, normal palpable peripheral lymph nodes, rectal temperature of 103.1°F, stringy salivation, severe bilateral atrophy of masticatory muscles and trismus. Haemogram revealed Hb - 14.0 g%, PCV - 46%, TLC- 8,850 cells/mm³, DLC showed Neutrophils - 88% and Lymphocytes - 12% and negative for blood parasites. The Serum Creatinine Kinase level was found to be 94

U/L. By the characteristic clinical signs and laboratory findings the condition was diagnosed as Chronic Masticatory Muscle Myositis.

Treatment and Discussion

The dog was treated with Inj. Prednisolone @2.0 mg/Kg B.Wt, I/M, twice a day along with supportive fluid therapy and B-complex vitamins. Animal showed slight improvement and opened its mouth partially after three days of treatment. The dog started taking little fluids but the condition gradually worsened after a week and was succumbed to death due to inanition and debilitation. This disease is an autoimmune process in which circulating antibodies specifically target masticatory muscles. Preferential susceptibility of these muscles to agents that produce myositis is because of the unique myofiber composition (Orvis *et al.*, 1981). Autoantibodies produced by the immune system against type 2M fibers of masticatory muscles are associated with masticatory muscle myositis (Melmed *et al.*, 2004). The histopathology and immunochemical features of MMM suggested the disorder as immune mediated disorder by Anderson *et al.*, 1993.

Patients can be presented either in the acute or, more commonly, chronic phase of the disease. In the acute form hypertrophy of temporal and masseter muscles is observed with myalgia whereas in chronic form, severe, progressive muscular atrophy accompanied by fibrosis, resulting in reduced ability to open the mouth and trismus or lockjaw are noticed (Ettinger, 2010). Most commonly the cases are presented with jaw pain or with trismus. MMM was identified to be the second most potential cause for locked jaw syndrome after Temporomandibular joint ankyloses (Gatineau *et al.*, 2008).

Diagnosis can be made tentatively based on the clinical signs and confirmation can be done by the laboratory tests. Clinical signs include either hypertrophy of masticatory muscles with protruded eye balls or muscular atrophy with sunken eye balls, which are suggestive of acute and chronic stage of the disease. Laboratory tests are performed to evaluate hematology, serum Creatinine Kinase, and detection of type 2M antitype antibodies (Ettinger, 2010). Leucocytosis and Increased serum CK values are noticed often.

Electromyography (EMG) is performed to reveal abnormal spontaneous activity of the masticatory muscles. Diagnosis can be supported by characteristic histopathological changes like necrosis and phagocytosis in 2M muscle fibers. Immunoblot assays done by Shelton *et al.*, 1987 revealed that the antibodies were most often directed against a 185 K protein, myosin heavy chain, and a band that appeared to be LC2-M (myosin light chain 2-masticatory).

Treatment of MMM includes administration of immune suppressive doses of corticosteroids which usually resolves the clinical signs in most of the affected animals (Ettinger, 2010; Pitcher GD *et al.*, 2007; Gatineau *et al.*, 2008).

Early detection of the disease and aggressive immunosuppressive therapy is required for favourable prognosis.

In the present case, the animal showed slight improvement and opened its mouth partially after three days of treatment. The dog started taking little fluids but the condition gradually worsened after a week and the dog died due to chronicity of the disease, inanition and debilitation.

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Fig. No. 1 showing mongrel with atrophied masticatory muscles and sunken eye balls



Fig. No. 2 showing Trismus/ Locked jaw