Masticatory Muscle Myopathy in a Pug – A Case Report

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A case of masticatory muscle myositis (MMM) in a five year old pug bitch was diagnosed by classical clinical signs and response to treatment with corticosteroid.

**Key words:** Masticatory Muscle Myopathy, Pug.

Masticatory muscle myositis (MMM) is an autoimmune, focal inflammatory myopathy with clinical signs restricted to the muscles of mastication which are innervated by the mandibular branch of the trigeminal nerve (Reiter and Schwarz, 2007). This disease is most common in German Shepherds, Labrador retrievers, Doberman Pinschers, Sharpei, Beagles and other large breeds. This condition is limited to the masticatory muscles because they have a characteristic molecular structure called II M (2M) muscle fibers and auto-antibodies against these fibers result in masticatory muscle myositis. The classical clinical presentation for masticatory muscle myositis is trismus, jaw pain, swelling or atrophy of the muscles of mastication and enophtalmos (Ito *et al.*, 2009). The average age of onset for masticatory muscle myositis is three years and the disease is most commonly reported in large-breed dogs. Complete physical and neurologic examinations are important to confirm that clinical signs are restricted to the muscles of mastication and confirmation is done by ELISA test for circulating antibodies against masticatory muscle type 2M fibers. The present communication describes successful therapeutic management of masticatory muscle myositis in a Pug.

A five year old Pug bitch was brought to the Teaching Small Animal Clinic of the University with the history of inability to open the mouth and unable to eat and drink for last 15 days. There was no history of dog bite and anti-rabies vaccination status was up to date. Clinical examination revealed wasting of masticatory muscles, locked jaw (trismus) and enophtalmos (Fig.1). Vital signs were normal. Neurological and orthopaedic examinations revealed no other abnormalities. Hematology revealed neutrophilia, cleucycytosis with moderate left shift. Muscle biopsy was not done as owner did not give consent for it. Based on classical clinical signs the case was tentatively diagnosed as masticatory muscle myopathy.

![Fig.1. Trismus in the Pug bitch](image)

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On first day the animal was treated with inj. dextrose normal saline @ 20 ml/kg b.wt. I/V, inj. Vitamin B complex 1ml I/M and prednisolone @ 1mg/kg b.wt. I/M. Treatment was followed by prednisolone 1 mg/kg b.wt. PO BID for 15 days. Prednisolone therapy was tapered slowly for subsequent two weeks. The animal showed uneventful clinical recovery after a month.

Serum assay could not be performed as this kit was unavailable. Similar clinical signs were reported by many workers (Tresamol et al., 2012 and Kavitha et al., 2016). Evaluating a muscle biopsy can also provide diagnostic confirmation of the disease as well as additional information regarding prognosis, particularly when muscle atrophy is present and significant fibrosis is suspected. Muscle biopsy documents the severity of fiber loss and degree of fibrosis, which are important in determining the long-term prognosis and probable success of therapy. Immunosuppressive doses of corticosteroids are the drug of choice for this condition regardless of muscle atrophy or the amount of fibrosis, similarily Pitcher and Hahn (2007) has also reported.

**References**


