

Masticatory Myositis of a Dog – Case Study

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Abstract. Atrophic myositis is the result of prominent atrophy of masticatory muscles. The masseter, temporal, and pterygoid muscles are selectively affected. A dog was presented to the Emergency Clinic of our Veterinary Faculty with the following clinical signs: progressive dyspnea and weight loss, apathy, cough, adinamia. The 11 years old, female, cross-breed dog was reported to be unable to completely open his mouth (trismus), with the head having a fine fox-like contour, with unusual prominences of zygomatic arches. Following necropsy, the gross exam revealed advanced bilateral atrophy and fibrosis of temporal, pterygoid and masseter muscle, accompanied by a primary mammary carcinoma with pulmonary metastases. Histological lesions from the masticatory muscles were very patchy. In some areas there was an admixed inflammatory infiltrate predominately composed of lymphocytes and plasma cells. Muscle fiber atrophy was associated with focal areas of chronic proliferative fibrosis. In conclusion, masticatory muscle myositis needs to be included in the differential diagnosis of disorders in dogs with trismus and abnormal jaw function.

Keywords: dog, masticatory muscles, myositis, trismus

INTRODUCTION

Canine masticatory muscle myositis (CMMM) is an immunomediated canine myopathy, which selectively causes focal myositis and progressive destruction of type IIM myofibres. The masticatory muscles (masseter, temporal, and pterygoid) are selectively affected, leading to trismus in dogs (Brogdon, 1991; Nelson et al., 2000). This disease must be included in the differential diagnosis list of problems of the temporo-mandibular joint (e.g.: trigeminal neuritis or the early form of tetanus in dogs) (Blot, 1995; McGavin, 1995; Shelton, 1991). The condition was previously designated as two separate disorders: eosinophilic myositis and atrophic myositis which are now recognized to be two ends of the spectrum of a single disease known as masticatory myositis. Dogs have a thick fascia of the masticatory muscles, thus the swelling that can occur in masticatory myositis may be due, at least in part, to ischemic damage due to compartment syndrome initiated by increased pressure within inflamed muscle. Atrophic myositis is the result of prominent atrophy of temporal and masseter muscles (McGavin et al., 2007, Jubb et al., 2007). This report shows an 11 years old, female, cross-breed dog, reported to have chronic masticatory problems. Histological examination from muscle biopsy following necropsy enabled the diagnosis of this myopathy to be made.

MATERIALS AND METHODS

A dog was presented to the Emergency Clinic of our Veterinary Faculty with the following clinical signs: progressive dyspnea and weight loss, apathy, cough, adinamia. The 11 years old, female, cross-breed dog was reported to be unable to completely open his mouth (trismus), with the head having a fine fox-like contour, with unusual prominences of zygomatic arches. The owner decided for euthanasia. Necropsy exam followed by classical histopathological exam (Hematoxilin and Eosin stain) were performed inside the Pathology Department, from within the Faculty of Veterinary Medicine, Cluj-Napoca.

RESULTS AND DISCUSSION

Gross necropsy findings included advanced bilateral atrophy and fibrosis mainly of temporal, pterigoid and masseter muscle (fig. 1A and 1B). Muscle atrophy gradually became very obvious and the head appears to had a fine fox-like contour with unusual prominence of the zygomatic arches. These muscle lesions consisted with the late stages of the condition, and we noticed advanced atrophy and fibrosis. Beside the muscle lesions, the dog had a mammary carcinoma with lung metastasis, pulmonary edema and left ventricular hypertrophy, dilatation, and endocardial fibrosis.

Histological lesions from the masticatory muscles were very patchy. In some areas there was an admixed inflammatory infiltrate predominately composed of lymphocytes and plasma cells (fig. 1E). The primary inflammatory cells causing destruction of myofibers in masticatory muscle myositis of dogs are lymphocytes, presumably recruited following binding of antibody. Lymphocytes may be admixed with plasma cells, and plasma cells may predominate in some cases. Macrophages will also be prominent in cases with marked myofiber necrosis. Fiber atrophy is common, and atrophic fibers can be seen associated with inflammation (fig. 1E) or with dense fibrosis (fig. 1C) (Jubb et al., 2007). In some microscopical areas, beside the normal muscle, there were areas of muscle fiber atrophy, accompanied by muscle fiber compensatory hypertrophy (fig. 1D).

Histologic examination of muscle from suspect masticatory myositis dogs can often be frustrating. The inflammatory lesions are patchy in some cases, and biopsy may reveal only generalized fiber atrophy. Any evidence of fiber degeneration or regeneration, even in the absence of obvious inflammation, should be considered suspicious if the clinical history is consistent with myositis. Examination for evidence of endomysial and perimysial fibrosis is important, as severe fibrosis will negatively impact on the prognosis for return of full jaw mobility and muscle mass (Jubb et al., 2007).

The masticatory muscles of dogs, especially the temporal muscles, appear to be particularly prone to a variety of generalized myopathic and systemic disorders. Dogs with X-linked muscular dystrophy often have prominent atrophy of the temporal muscle and are unable to fully open their mouths, although this condition is not accompanied by pain. Labrador Retrievers with inherited myopathy also have prominent temporal muscle atrophy. Polymyositis may appear primarily as a problem with muscles of mastication, although careful evaluation will reveal abnormalities in other muscles. Dogs with any generalized, illness often develop rapid atrophy of the temporal muscles that resolves with treatment for the primary problem (Jubb et al., 2007). In our case, the primary mammary carcinoma with lung metastases obviously represented the main disease, and caused most of the clinical changes encountered in this case (progressive dyspnea and weight loss, apathy, cough a.o.). Persistent bilaterally symmetric atrophy of masticatory muscles not associated with pain,

immobility of the jaw, or generalized disease also occurs in dogs. These latter cases of masticatory muscle atrophy appear to be idiopathic and are not associated with inflammation (Jubb et al, 2007).

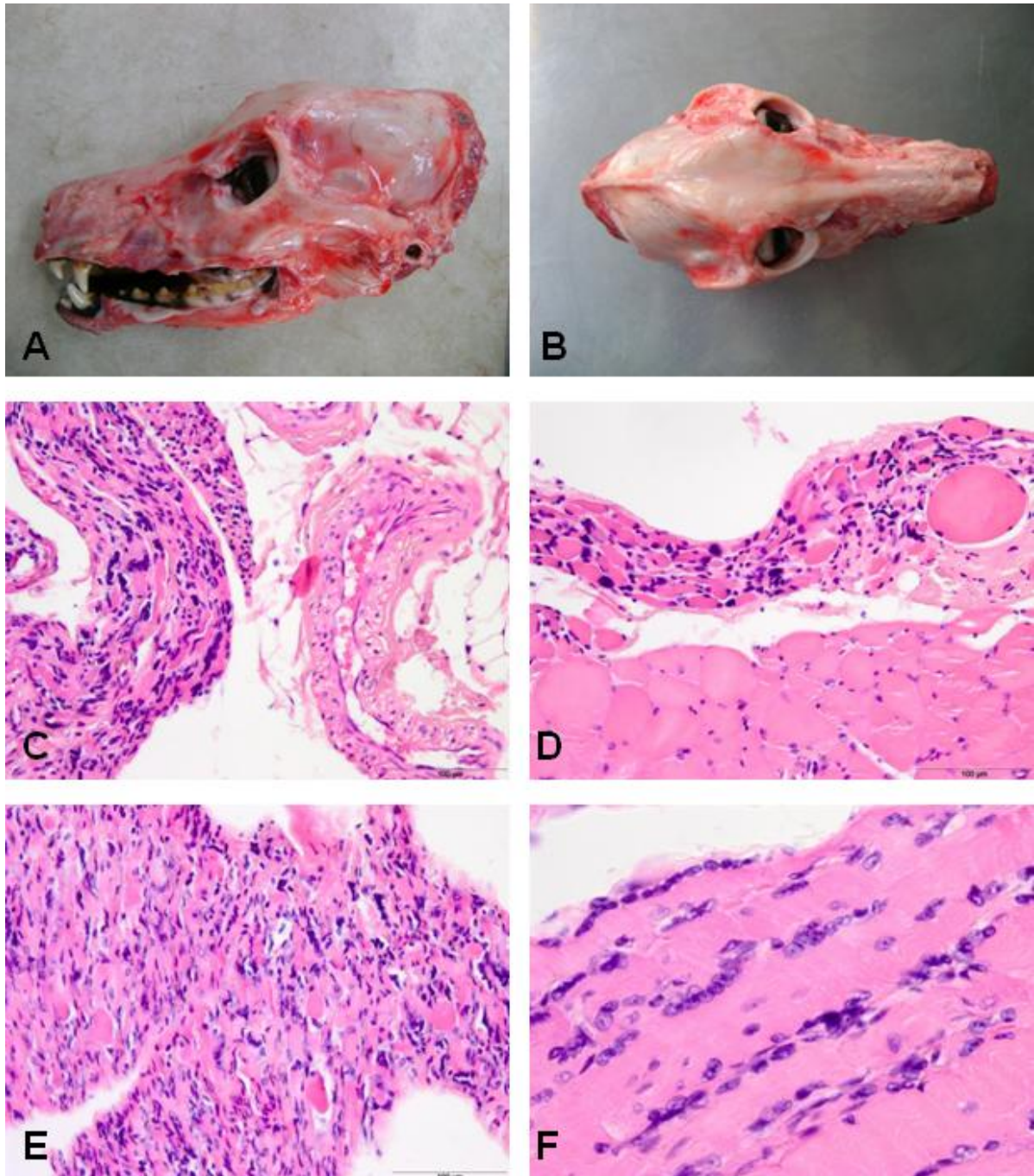


Fig. 1: A and B - Bilateral atrophy and fibrosis mainly of temporal, pterygoid and masseter muscle (gross lesions); C – Focal atrophy of masticatory muscles. D – Area of muscle atrophy, with mild inflammatory infiltrate and rare hypertrophic muscle fibers (rounded, enlarged, eosinophilic); E – Area of muscle atrophy, mild fibrosis and mild inflammation mainly with mononuclear cells and macrophages; F – Areas of muscle regeneration with proliferation of satellite cells.

CONCLUSIONS

To summarize, masticatory muscle myositis should be included in the differential diagnosis of disorders in dogs with trismus and abnormal jaw function. Muscle biopsy examination can be a very useful technique which would enable the definitive diagnosis and prognosis of canine atrophic masticatory muscle myositis to be made.

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