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Case Report

Masticatory Muscle Myopathy in a Dog

Murat Sarıerler[1] , Zeynep Bozkan Tatlı[2], Bahar Bayraktar[3]

[1]Department of Surgery, [3]Department of Pathology, Faculty of Veterinary Medicine, University of Adnan Menderes, Aydın, Turkey, [2]Alba Veterinary Clinic, Kuşadası, Aydın, Turkey

A B S T R A C T

A 2,5 years old mixed breed male dog presenting complaints of inability to open the mouth, drooling, with no food and water consumption 3 days prior to referral was enrolled in the present article. Dehydration, bilateral severe swelling of the temporal and masseter muscles and constant pytyalism were detected at physical examination. Body temperature was within the reference ranges and neither orthopedic nor neurologic abnormalities were detected. Complate blood cell count did not show abnormalities other than eosinophilia of 2170/ml. Clinic and histopathologic findings indicated masticatory muscle myopathy. Initial treatment included prednisone (1 mg/kg/12h, i.m.) and procain peniciline (20000 IU/kg/day, i.m.) administrations. Related clinical signs were regressed following 3 days of therapy. The case regained normal jaw motions albeit severe atrophy was evident in masseter and temporal muscles. Almost 2 months later, the case was readmitted with similar symptoms, because of withdrawal of recommended drugs by the owner himself. Supportive therapy included dexamethasone (0.15 mg/kg, p.o. for 15 days). Jaw functions recovered within 4 days and were still normal after six months. As a result, it was concluded that immune-mediated inflammation of muscles of mastication in dogs may be treated with use of steroids for a long period, albeit in case of early withdrawal of therapy, recurrence may observed, following initial recovery swelling and severe atrophy could be developed on masticatory musles whereas this conditon may not limit jaw mobility.

Keywords: Dog, Masticatory muscle myositis, Otoimmune-mediated myositis

Bir Köpekte Çiğneme Kası Miyopatisi

Ö Z E T

Bu makalede, 3 gündür ağız açmada, gıda ve su alımında güçlük ve ağız akıntısı şikâyetiyle kliniğimize getirilen bir köpek (30 aylık, 22 kg ağırlığında, erkek, melez) ele alındı. Klinik muayenede dehidrasyon, temporal ve masseter kaslarında bilateral şiddetli şişkinlik ve sürekli salya akıntısı belirlendi. Vücut ısısı referans değerler içerisindeydi. Ortopedik ve nörolojik herhangi bir bozukluk saptanmadı. Hastada periferal eozinofili (2170/ml) bulunması dışında tam kan sayımında herhangi bir anormalite saptanmadı. Klinik ve laboratuar bulguları ile birlikte masseter kasından alınan biyopsi örneklerinin histopatolojik muayene sonuçları rehberliğinde çiğneme kası miyozitisi teşhis edildi. Prokain penisilin (20000 IU/kg, gün, i.m.) ve prednizolon (1 mg/kg, 12s, i.m.) uygulaması ile tedaviye başlandı. Üç gün sonra klinik bulgularda gerileme görüldü. Hasta, normal çene fonksiyonlarını tekrar kazandı, bununla birlikte masseter ve temporal kaslarda şiddetli atrofi gözlendi. Yaklaşık iki ay sonra, sahibi ilaç uygulamasını önerilen zamandan daha erken kestiği için, hasta başlangıçtakine benzer bulgularla tekrar başvurdu. Tedaviye dekzametazon (0.15 mg/kg, p.o., 15 gün) ile tekrar başlandı. Altı ay sonra hasta sahibi ile yapılan telefon görüşmelerinde köpeğin normal çene fonksiyonlarına sahip olduğu öğrenildi. Sonuç olarak, köpeklerde immun aracılı çiğneme kası miyozitisin uzun süreli steroid kullanımı ile tedavi edilebileceği, fakat tedavi erken kesilirse nüks olayların görülebileceği, iyileşmeden sonra çiğneme kaslarında şişlik ve şiddetli atrofiler şekillenebileceği, ancak bunların çene hareketlerini kısıtlamayabileceği kararına varıldı.

Anahtar Kelimeler: Çiğneme kaslarının yangısı, Köpek, Otoimmun miyozitis

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Correspondence to: Murat Sarıerler, Adnan Menderes Universitesi, Veteriner Fakültesi, Cerrahi Anabilim Dalı, 09016 ,Işıklı, Aydın, Türkiye. E-mail: msarierler@adu.edu.tr

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Introduction

Generalized inflammatory myopathies are attributed infectious disease (including the Leptospirosis, Toxoplasmosis, Neosporosis, Leishmaniasis, Hepatozoonosis, *Rickettsia spp.*, *Dirofilaria immitis* and *Clostridium spp* infections), immune mediated diseases (Systemic lupus erythematosus, Other connective tissue diseases, Drugs/toxins comprising cimetidine, trimethoprim–sulfadiazine, penicillamines etc., Paraneoplastic/metastatic neoplasia (Thymoma, Lymphoma, Idiopathic disease) . Focal inflammatory myopathies comprise Masticatory muscle myositis, Extraocular muscle myositis (Shelton, 1989; Lewis, 1994; Taylor, 2000). An autoimmune disease Masticatory muscle myositis (MMM), associated with autoantibodies against masticatory muscle type 2M fibers is focal inflammatory myopathy with clinical signs restricted to the muscles of mastication (3–5). MMM has historically been called eosinophilic myositis or atrophic myositis. However, these names representing the acute and chronic phases of masticatory muscle myositis suggest a different pathogenesis (Shelton, 1989).

MMM is usually bilateral and involve the temporalis, masseter, pterygoid, and rostral digastricus, all of which are innervated by the mandibular branch of the trigeminal nerve (Orvis, 1981; Shelton, 1987). MMM mainly affects large breed dogs (Lecoueur, 1989).

Acute phase of MMM include clinically swelling, jaw pain, inability to open the jaw (trismus). In chronic phase, there are marked muscle atrophy associated with myofiber loss and muscle fibrosis (Shelton et al., 1987; Lewis, 1994; Taylor, 2000).

This report describes inflammatory muscle myopathy restricted to the muscles of mastication.

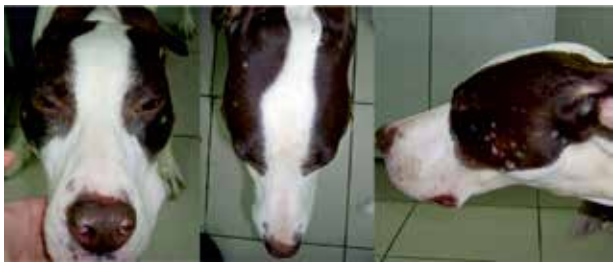

Figure 1. Bilateral severe swelling of the temporal and masseter muscles.

Şekil 1. Temporal ve masseter kaslarında bilateral siddetli şişlik.

Clinical and histological findings indicated that myositis described in the present case may be MMM. Initial treatment included administration of prednisone (prednisolon®-Fako, 1 mg/kg, 12h, i.m.) and procain peniciline (Iecilline®-İ. E. Ulagay, 20000 IU/kg, day, i.m.). After three days, partial recovery was observed. The dog regained normal jaw motions albeit severe atrophy was observed in masseter and temporal muscles. Treatment was continued 7 days with same doses. Treatment was maintained with prednisolone (1 mg/kg, day, i.m.), and lowest tapering doses were prescribed for following weeks to the owner as 0.5 mg/kg/day i.m., between 15th-30th days and 0.25 mg/kg/day, i.m., between 30th-60th days.

Almost 2 months later, the patient presented for reevaluation, with the complaint of swelling and stiffness at masseter and temporal muscles and restriction of jaw movement, again. As being informed, owner was stopped drug administration

Case History

Thirty months old, 22 kg, male mix breed dog was referred to department of surgery, faculty of veterinary medicine, Adnan Menderes University, clinically representing complaints of inability to open the mouth, drooling, inefficient drinking and eating behavior, progressing over the previous 3 days. Physical examination revealed dehydration, bilateral severe swelling of the temporal and masseter muscles and constant pytyalism (Figure 1). Body temperature was within normal range (38 °C), and neither orthopedic abnormality was detected. During the examination of mouth, pain and trismus were observed. Manual opening of the mouth was not available. No abnormalities were detected during neurologic examination. The palpebral, pupillar and spinal reflexes and also deep pain sensitization were presenting. Complete blood cell count was normal other than a peripheral eosinophilia of 2170/ml (reference range <1250/ml) (Shelton et al 2000) and mild increased CK (395 U/L; reference range 20 to 200 U/L) (Turgut, 2000) and globulin levels (6.1 g/dL: reference range 2.3 to 5.2 g/dL) (Turgut, 2000) were observed.

In an attempt to evaluate differential diagnosis dysphagia and ptyalism, the oral cavity and probable pharynx radiographs were obtained for foreign bodies, masses, abnormal anatomic structures, and dental or temporomandibular joint disease. No abnormalities were noted.

At the first referring date, biopsy application was not available as the owner rejected. Nevertheless, muscle biopsy was performed from right masseter muscle after seven days. During the biopsy procedure, severe atrophy restricting the exact margins of masseter muscle was evident. On histopathological examination, there were extensive loss of myofibres, regenerated muscle fibres with multinucletid fibres (asterisk), fibrosis, inflammatory reactions with macrophages, lymphocytes and plasma cells (Figure 2).

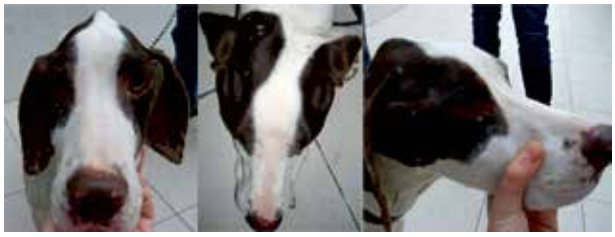

Figure 3. One week later, severe atrophy at masticatory muscles.

Şekil 3. Bir hafta sonra, temporal ve masseter kaslarında şiddetli atrofi.

before recommended date. Treatment was restarted by using dexamethasone (Dekort®-Deva) 0.15 mg/kg, day, p.o., and dose was decreased as follows; 15th-30th days 0.075 mg/kg, day, i.m., 30th-45th days 0.03 mg/kg, day, i.m., at 45th-60th days 0.015 mg/kg. Dog was regaining his normal jaw functions within six months. Trismus and swelling of masticatory muscles recovered but severe atrophy was occurred (Figure 3). However, no recurrence was observed during treatment period.

Discussion

MMM is a progressive myopathy of the dog and commonly affects large breed dogs (McKeown and Archibald, 1979; Lecoueur, 1989; Pedroia, 1989). The average age was reported as 3 years or younger. The present case was young and a middle sizes breed. Taking into account, the most commonly presenting problems associated with MMM have been reported as trismus, swelling or atrophy, and pain restricted

to the muscles of mastication (McKeown and Archibald, 1979; Glauberg and Beaumont, 1979; Smith, 1989; Gilmour, 1992). Clinical symptoms are usually bilateral. Ocular signs have been reported rarely, but swelling of the temporalis and pterygoid muscles can lead to exophthalmos, and atrophy of these muscles may cause enopthalmos (McKeown and Archibald, 1979; Shelton et al., 1987; Smith, 1989). Exophthalmos may lead to compression or stretching of the optic nerve which result to blindness (Pedroia, 1989; Smith, 1989). In the present case, severe atrophy of masticatory muscles was observed. Ocular, orthopedic, neurologic and radiologic abnormalities were not detected, other than masticatory muscles related signs. Peripheral eosinophilia is a common clinical laboratory finding (McKeown and Archibald, 1979; Gilmour, 1992) however sometimes consistent CBC count abnormalities may not be determined (Gilmour, 1992). According to serum chemistry profiles, increased creatinine kinase (CK) and globulin levels have been reported (Gilmour, 1992). In the present case; obvious peripheral eosinophilia and mild increased CK and globulin levels were observed.

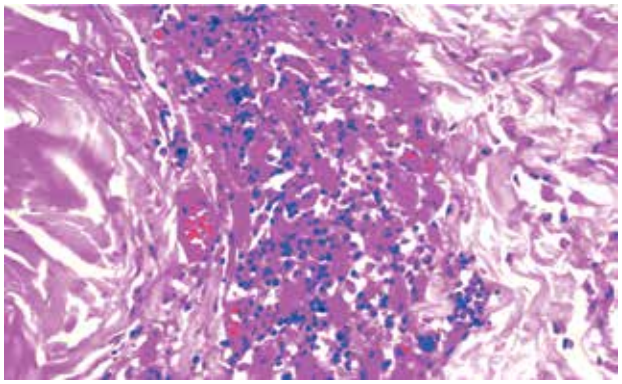

Figure 2. Fibrosis (F) and lymphocyte and macrophage infiltration areas in masseter muscle, H&E. X80

Şekil 2. Masseter kasında fibrosis (F) ile lenfosit ve monosit infiltrasyonu, H&E.X80

Clinical symptoms are important for suspicion of the MMM. Certain diagnosis may be confirmed with a muscle biopsy, and applying immunohistochemistry with spesific type 2M antibodies on frozen sections (Shelton, 1989). On the other hand, presence of spesific type 2M antibodies by ELISA is useful in sera samples. Although immunohistochemistry and ELISA had no oppurtinity, histological findings of mastication muscles were consistent with those of MMM (Vilafranca, 1995). On the other hand, the present case was rapidly answered steroid therapy for immune-mediated inflammatory reaction.

In conclusion, masticatory myositis associated with immune-mediated may be treated use of steroids for a long period, however if treatment discontinues earlier, relapse may occur. After recovering of masticatory muscles, swelling, severe atrophy may develop, but the jaw movement is not restricted by these.

In conclusion, clinicians and pathologists should bear in mind that immune-mediated inflammation, especially MMM, should be taken into consideration with mastication muscles showing clinical signs.

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