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Masticatory Muscle Myositis in a Maltese Dog

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Abstract : A 8-year-old, castrated male Maltese with difficulty in opening his mouth was presented. Masticatory muscle myositis (MMM) was diagnosed by 2M antibody test. After treatment of MMM with corticosteroids, range of jaw motion improved and was almost normal at 5 months. In dogs with MMM, early detection and aggressive immunosuppressive therapy are required to improve the prognosis.

Key words **:** dog, masticatory muscle myositis.

Introduction

Masticatory muscle myositis (MMM) is an inflammatory myopathy occurred relatively frequently in dogs (6). This disorder is immune-mediated disease associated with clinical signs of difficulty in opening the mouth (called "trismus") due to severe atrophy and fibrosis of the muscles of mastication, including the temporalis, masseter, pterygoid, and rostral digastricus muscles (3). Autoantibodies against masticatory muscle type 2M fibers specifically target the masticatory muscles, thus detection of these antibodies in serum is useful in the diagnosis (8). Dogs with MMM generally have no other neurologic or physical abnormalities, which is helpful for differentiating MMM from other causes of trimus including traumatic myopathies, endocrine myopathies, trigeminal neuropathies, and age-related muscle atrophy (4).

The main clinical sign of MMM is difficulty in opening the mouth. In the acute stage, there are also swelling of the jaw and temporal muscles, and pain in the jaw, or may be a fever and a swollen local lymph nose. In the chronic stage, there is noticeable atrophy of the masticatory muscles and inability to open the mouth due to the fibrosis. Unfortunately, many owners do not recognize that their dogs are having a problem until they reach the chronic phase. Treatment is non-surgical and involves immunosuppressive doses of corticosteroids with optional supportive therapy. We here describe clinical findings, diagnosis, treatment, and case outcome in a Maltese dog with MMM.

Case

A 8-year-old, castrated male Maltese was presented to the Veterinary Medical Teaching Hospital of Seoul National University, with the complaint of lameness, depression and fever following rabies vaccination. Physical examination showed fever and enlargement of submandibular and popliteal lymph nodes. No abnormalities were identified on hematologic and serum biochemical examination, except increased CK (1005 U/L; reference interval, 8-216 U/L), ALP (672 U/L; reference interval, 47-254 U/L) and ALT (95 U/L; reference interval, 17-78 U/L) activities. Orthopedic examination revealed weight bearing lameness of left hindlimb, mild muscle atrophy of left thigh muscle, and moderate pain and severely reduced range of motion on manipulation of left hip joint. On survey radiographs, severe degenerative changes of left coxofemoral joint were observed. Femoral head and neck osteotomy was performed to relieve pain and improve lameness. After anesthetic induction to perform tracheal intubation, the mouth of the dog was unable to be fully opened. The temporomandibular joints are normal and no abnormalities were evident on skull radiographs. Other causes of trimus were ruled out through neurologic and physical examinations, then immune-mediated masticatory muscle myositis was suspected. However, further diagnostic examinations and treatments were not pursued due to the owner's request.

The clinical signs were progressed so that the dog was represented with an inability to open the jaw, difficulty in eating and drinking, jaw pain, continuous fever, and severe atrophy of masseter and temporalis muscles (Fig 1). The dog was unable to achieve more than a 2 cm opening between the maxillary and the mandibular incisor teeth (Fig 2). Biopsies from the masseter and temporalis muscles for histopathologic examination could not be performed because of severe atrophy of these muscle. Instead, the serum 2M antibody test was performed. Circulating antibodies against type 2M muscle fibers were detected in the serum, and based on these findings, a diagnosis of MMM was made.

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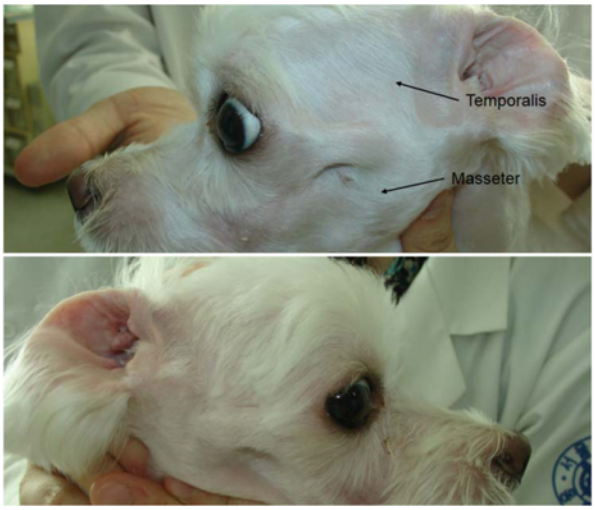

Fig 1. Muscle atrophy in a Maltese dog with masticatory muscle myositis.

Immunosuppressive therapy with prednisolone acetate (2 mg/kg orally twice daily for 7 days, then tapered over 5 months) was initiated. Physical therapy such as acupuncture 1-2 times/week was also performed as a supportive therapy.

A liquid diet and water were administered orally. Range of jaw motion gradually increased and the liquid diet was replaced with hard food. By 10 days after initial treatment, the jaw opened until 5 cm, by 1 month, until 7 cm, and by 2 months, until 8 cm (Fig 2). At 5 months, there was no further improvement in jaw mobility, and the volume of masticatory muscles was remarkably increased.

Discussion

MMM is an autoimmune disease and is limited to the masticatory muscles because they have a molecular structure, called 2M muscle fibers, which are only found in masticatory muscle (4). This unique type 2M muscle fiber isoform may be related to the different motor nerve branches that develop during embryologic development (9). Therefore, MMM is occurred when the immune system's antibodies specifically target these 2M muscle fibers. Previous studies reported autoantibodies against type 2M fibers in dogs with MMM (7,8). These antibodies are not reactive with any other muscle groups or found in any other muscle disease, such as polymyositis. It remains unknown what is the trigger for formation of autoantibodies or why are directed specifically against type 2M fibers. Some previous reports suggest that molecular mimicry may play a role, with antibodies or T cells generated in response to an infectious agent that subsequently cross-reacts with self-antigens (2,3). In the present case, the dog developed MMM within ten days of vaccination, and thus rabies virus antigen for vaccination might have played a role as a trigger for the aberrant immune response. Antibodies directed against these virus antigens could have potentially cross-reacted with these muscle fibers. A previous study in the human has been documented that autoantibodies directed against bacterial antigens attack cardiac and skeletal muscle (2).

Although MMM can occur in any canine breed, age, or sex, young large-breed dogs, including German shepherds, Labrador retrievers, Doberman pinschers, and Golden retrievers, are most commonly affected (10). Occurrence of MMM in small-breed dogs such as Cavalier King Charles spaniels and Pug has also been reported (4,5). In this case, MMM was diagnosed in the Maltese commonly distributed in Korea by the identification of the symptoms and the serum 2M antibody test. Acute and chronic forms of MMM have been described. Trimus and swelling of masticatory muscles are generally observed in the animals of the acute phase. Nonspecific clinical signs such as pyrexia and lymphadenopathy have also been reported during the initial 1-3 weeks of MMM (10). The dog in the present case showed unknown fever and swelling of submandibular lymph node in the initial phase. However, owner and veterinarian did not recognize a problem until the chronic phase, which is characterized by marked muscle atrophy with persistent trimus. Recognizing typically clinical signs of MMM and complete physical examination are important because treatment is most successful when initiated in acute phase.

For the diagnosis of MMM, various diagnostic tests, including complete physical and neurological examinations, general and specialized blood tests, muscle biopsy, radiogra-

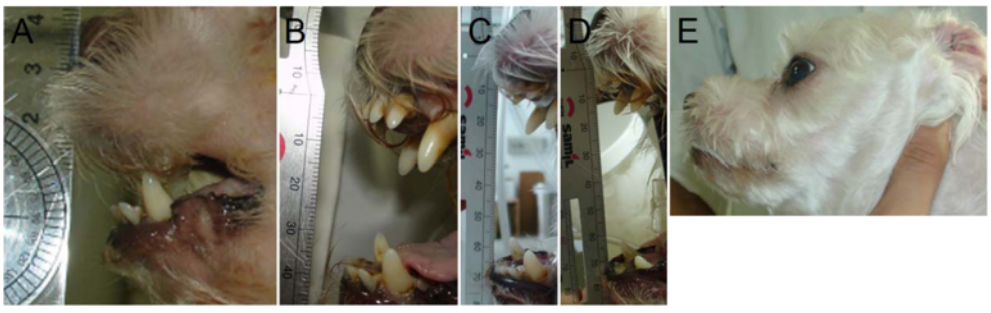

Fig 2. (A-D) Changes of distance between the maxillary and the mandibular incisor teeth in a Maltese dog with masticatory muscle myositis following treatment; (A) Before treatment, (B) 10 days, (C) 1 month, and (D) 2 months after initial treatment. (E) Increased volume of masticatory muscles was noticeable after 3 months after treatment.

phy, urinalysis, and electromyography, can be performed. The purpose of some of these tests is to rule out other possible causes of clinical signs. Initial diagnostic testing should include a complete blood count and serum chemistry profile, including a creatine kinase (CK). These evaluation in affected dogs is typically nonspecific. However, CK activity is frequently increased during the acute phase. In this case, CK level was elevated at the initial blood test. In general, clinical signs associated with MMM and positive results from a 2M antibody test confirm the diagnosis. Biopsy of the affected muscles can be also used to confirm MMM, and helpful in determining the stage or severity of the disease. However, this procedure may be invasive. In contrast, the serum 2M antibody test used in this case is relatively easy and simple. This test has been also proven highly specific (100%) and sensitive (85-90%) (1,3). Furthermore, this test can differentiate MMM from polymyositis because patients with polymyositis test negative for antibodies against type 2M fibers. Therefore, the serum 2M antibody test is a useful diagnostic test for MMM.

For a favorable outcome in MMM, early accurate diagnosis and appropriate therapy are necessary. Treatment is centered on aggressive immunosuppression (3). However, the dosage can be tapered gradually over 4-6 months in accordance with improvement in clinical signs. This dose should be maintained until maximum jaw function has been regained and CK levels have returned to normal. In this case, CK levels were decreased to 356 U/L after 10 days of the initial treatment. The medication was maintained during 5 months, and the dog responded well initially to corticosteroid therapy. Clinical signs of the dog were gradually improved following treatment. A relapse did not occur after completely stopping of the drug. The dog required a semi-liquid diet during initial recovery. Range of jaw motion gradually increased and the liquid diet was replaced with hard food. Generally, forcible manual retraction of the jaw is contraindicated. Previous report described that anesthesia was used to facilitate forced correction of the trimus in a Pug, which resulted in inadvertent tongue protrusion, venous occlusion, and marked tongue swelling that became life threatening (4). The prognosis for MMM is determined by the degree of fibrosis present and the clinical response to immunosuppressive treatment (3). Based on our experience in this dog, we recommend early detection of MMM by recognizing typical clinical signs and complete physical examination, and aggressive immunosuppressive therapy in dogs with MMM for good prognosis.

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말티즈견에서 저작근염 발생례

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요 약 : 8년령 중성화 수컷 말티즈가 입을 열기 어려운 증상으로 내원하였다. 2M 항체 검사를 통해 저작근염을 진단하였다. 코르티코스테로이드로 처치 후 턱관절의 가동범위가 증가하였고, 처치 5개월 후에는 거의 정상으로 돌아왔다. 저작근염이 발생한 개에서 초기 발견 및 적극적인 면역억제 치료는 예후 향상을 위해 필요한 것으로 사료된다.

주요어 : 개, 저작근염