Masticatory muscle myositis in dogs

Categories : Vets

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LUCA MOTTA looks at the acute and chronic forms of this condition in dogs, discussing diagnosis, current and future therapy, as well as prognosis

Summary

Masticatory muscle myositis (MMM) is a common idiopathic autoimmune inflammatory myopathy in dogs characterised by a focal inflammation of the masticatory muscles digastricus, temporalis, pterygoid and masseteric. Large dog breeds seem to be predisposed and the median age of onset is around two years. There are two forms of MMM: acute and chronic. A common clinical feature in both types of MMM is the limited ability to open the mouth. Dogs may not be able to eat properly and can show weight loss. Dogs with “lockjaw” present a challenge when endotracheal intubation and general anaesthesia needs to be carried out. Other diseases that cause a limited ability to open the oral cavity should be ruled out before diagnosing MMM. The diagnosis of MMM is based on various criteria including the clinical signs, imaging, electromyography, presence of antibodies against type 2M myofibres, and muscle biopsy. The measurement of the titre of the antibodies against type 2M myofibres is 100 per cent specific and 85 per cent to 90 per cent sensitive. Therapy for acute and chronic cases of MMM consists of immunosuppression with corticosteroids. Generally, the prognosis is good.

Key words

masticatory muscle myositis, dogs, lockjaw, 2M myofibres

MASTICATORY muscle myositis (MMM) is a common idiopathic inflammatory myopathy in the dog. It is characterised by a focal inflammation of the masticatory muscles digastricus, pterygoid, temporalis, and masseteric.

The masticatory muscles are composed of a specific type of myofibres, namely type 2M myofibres. It is suggested canine MMM is a CD8(+), T-cellmediated, immunopathological disease that initiates myofibre destruction and leads to production of autoantibodies. The major histocompatibility complex class one and class two expression may play a role in the initiation and maintenance of MMM. The autoantibodies specifically target type 2M myofibres only and are not present in dogs with other immunemediated inflammatory myopathies. Thus, the autoantibodies in MMM are disease specific and they are not secondary to non-specific myofibre damage.

Clinical findings

MMM has been reported in dogs of any age or gender. However, larger breeds seem to be predisposed and the median age of onset is around two years. There are two forms of MMM: acute and chronic. A common clinical feature in both types of MMM is trismus, also called “lockjaw”, in which dogs have a limited ability to open the mouth due to contraction, pain and mechanical resistance of the muscles concerned with the jaw movements.

In the acute form of MMM, bilateral masticatory muscle swelling, facial myalgia, monolateral or bilateral exophthalmus, monolateral or bilateral protrusion of the third eyelid, high body temperature, tonsils and mandibular lymph nodes swelling can be observed.

In the chronic type of MMM, there is bilateral symmetrical masticatory muscle atrophy and fibrosis consequent to the progression of the myofibre damage that has started in the acute phase of the disease and has progressively continued (Figure 1).

Basically, when myo.bres reach the end stage of degeneration, they can be replaced by brous tissue. This phenomenon results in a decreased range of motion of a muscle because of mechanical resistance and, in the specific case of MMM, an inability to open the jaw sometimes more than several centimetres wide. Dogs may not be able to prehend and chew the food and can show weight loss and generalised muscle atrophy secondary to malnourishment. It is worth underlying that forceful physical attempts to open the jaw in dogs under sedation or general anaesthesia must not be carried out because there is the risk of causing mandibular luxation or fractures.

The inability to fully open the mouth in a dog with MMM presents a challenge when endotracheal intubation needs to be carried out. If endotracheal intubation is not possible, a laryngeal mask airway can be inserted to maintain the patient on inhalational anaesthesia. During the anaesthesia, the patient needs to be carefully monitored because complications may occur. An interesting case report showed that during anaesthesia in a dog with MMM, tongue protrusion occurred. Since there was trismus, it was not possible to reposition the tongue within the oral cavity and this resulted in venous congestion and severe swelling of the tongue. The jaw mobility was not improved even after removal of the rostral digastricus and masseteric muscle attachments from the mandible. Only a mandibular symphysiotomy resolved the tongue swelling.

Differential diagnoses for limited ability to open the oral cavity in dogs include ankylosis, luxation, dysplasia and osteoarthritis of the temporomandibular joint, craniomandibular osteopathy, osteomyelitis, neoplasms affecting the ear and/or the jaw (especially those in proximity of the temporomandibular joint), focal tetanus and generalised idiopathic polymyositis. As mentioned before, dogs with the chronic form of MMM will show bilateral masticatory muscles' atrophy. Unilateral masticatory muscle atrophy is usually caused by ipsilateral trigeminal nerve dysfunction, such as peripheral nerve sheath tumour.

Diagnosis

The diagnosis of MMM is based on various criteria, including the clinical signs, imaging, electromyography, presence of antibodies against type 2M myofibres, and muscle biopsy, with the analysis of inflammatory cellular infiltration by histochemical and immunological techniques.

MRI and CT are helpful in ruling out other causes of "locked jaw", and may be useful adjuncts in the selection of sites for diagnostic muscle biopsy. On CT, the masticatory muscles affected by MMM may reveal changes in size and are often characterised by hypoattenuation with different distribution patterns.

On post-contrast CT images, heterogeneous enhancement is usually visualised. On MRI, common findings include bilateral asymmetric multifocal ill-defined T2-weighted, T1-weighted and short TI inversion recovery (STIR) hyperintense lesions in the masticatory muscles and sometimes within the extraocular muscles. On post-contrast MRI images, mild to moderate heterogeneous enhancement can be visualised (Figures 2, 3 and 4).

Electromyography is a useful diagnostic tool to confirm the selective involvement of masticatory muscles. It also helps to confirm whether the MMM is part of a generalised polymyopathy. Electromyogra phy may reveal pathological muscle activity, such as fibrillation potentials and/or positive sharp waves (Figures 5 and 6), and can be normal in dogs with end-stage disease.

The measurement of the serum titre of the antibodies against type 2M myo.bres is 100 per cent specific and 85 per cent to 90 per cent sensitive. It should be noted, however, that previous corticosteroid therapy or end-stage MMM may produce a false-negative antibody titre. In such cases, evaluation of a muscle biopsy is recommended, especially when bilateral masticatory muscle swelling/atrophy and trismus are seen, the imaging findings are suggestive of MMM and electromyography has revealed abnormal activity of the masticatory muscles.

Muscle biopsy analysis is also useful in chronic cases because the amount of myofibre.brosis can be evaluated and a prognosis for return of muscle mass and jaw function can be hypothesised. Before carrying out the muscle biopsy, the laboratory that processes and interprets the specimens should be contacted to acquire information regarding how the sample should be obtained, stored and sent to the laboratory to ensure maximum diagnostic information is obtained. The quality of the

information obtained from a biopsy specimen will depend on its quality and in what condition it will arrive at the laboratory.

Masticatory muscle biopsies of dogs with the acute form of MMM are characterised by intense multifocal lymphocytic and plasmacytic perivascular infiltration, occasional eosinophils, and necrosis and phagocytosis of type 2M myofibres (Figure 7 and 8). In the chronic form, there is less inflammation and the.brosis, which follows the necrosis of the myofibres, is visualised (Figure 9).

Therapy

Therapy for acute and chronic cases of MMM consists of immunosuppression with corticosteroids. In the acute cases, oral prednisone at 1mg/kg to 2mg/kg twice daily is given until the jaw function returns to normal. Physical therapy should be started as soon as possible in an attempt to normalise the masticatory muscles' tone and to increase the jaw opening. Encouraging the dog to play with tennis balls or chew rawhide is recommended. Most dogs cannot eat properly and food should be blended to a consistency for licking. In most cases, a rapid clinical improvement is seen. Once the jaw function returns to normal, the prednisone dose is gradually tapered down and is maintained for four to six months.

Therapy for chronic cases of MMM consists of corticosteroids at 0.25mg/kg 0.5mg/ kg twice daily for about one month. The dose can be then slowly tapered down. The most common causes of treatment failure in MMM, as in other autoimmune diseases, are inappropriate therapy and too early discontinuation of the drug(s).

Other immunosuppressive agents, such as azathioprine, ciclosporin A or mycophenolate mofetil, may be indicated in dogs that fail to respond to corticosteroids, that relapse when the dose is tapered or that show severe side effects from the corticosteroids. Adjunctive immunosuppressive therapy in veterinary medicine is largely based on clinical experience.

Future therapies

It is suggested serotonin is involved in the development of muscles fibrosis via transforming growth factor B-1 signalling pathway (Pavone et al). This interesting finding would open the way to the development of new strategies that target serotonergic system for the therapy of idiopathic autoimmune inflammatory myopathies in both dogs and humans.

Prognosis

Generally, the prognosis is good, especially if the diagnosis is made early and therapy is started during the acute phase of the MMM. If the diagnosis is made too late, dogs may never regain the ability to adequately open the mouth.

The muscle atrophy may persist after treatment and can also be exacerbated by the corticosteroid therapy. Thus, the atrophy does not necessarily indicate a progression of the disease.

• Not all of the drugs mentioned in this article are licensed for use in dogs.

References and further reading

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Figure 1. Labrador retriever with the chronic form of MMM. Note the marked atrophy of the temporalis and masseteric muscles.

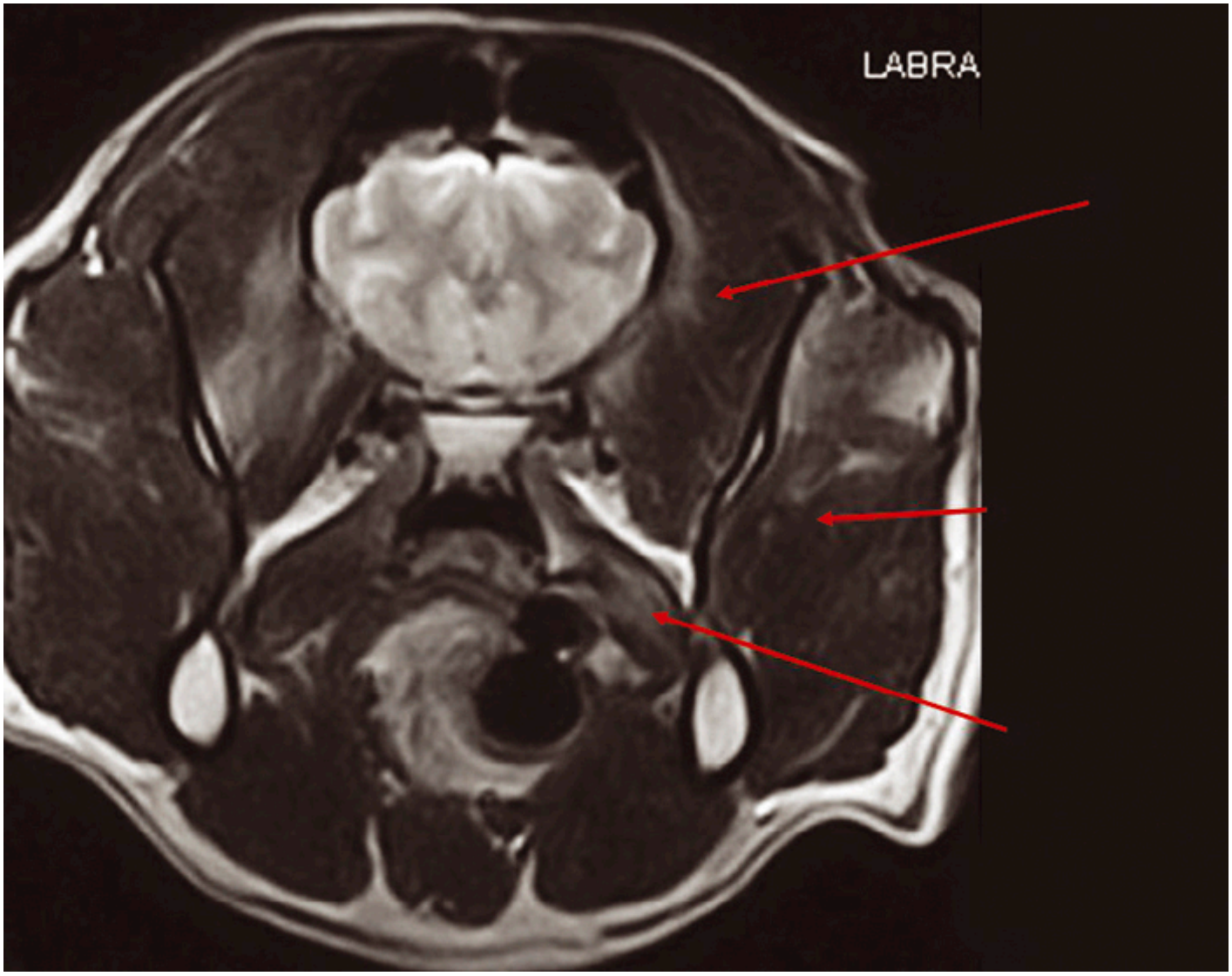

Figure 2. Labrador retriever with MMM. Transverse T2-weighted MR image showing bilateral multifocal asymmetric ill-defined heterogeneously hyperintense lesions within the temporal, masseteric and pterygoid muscles.

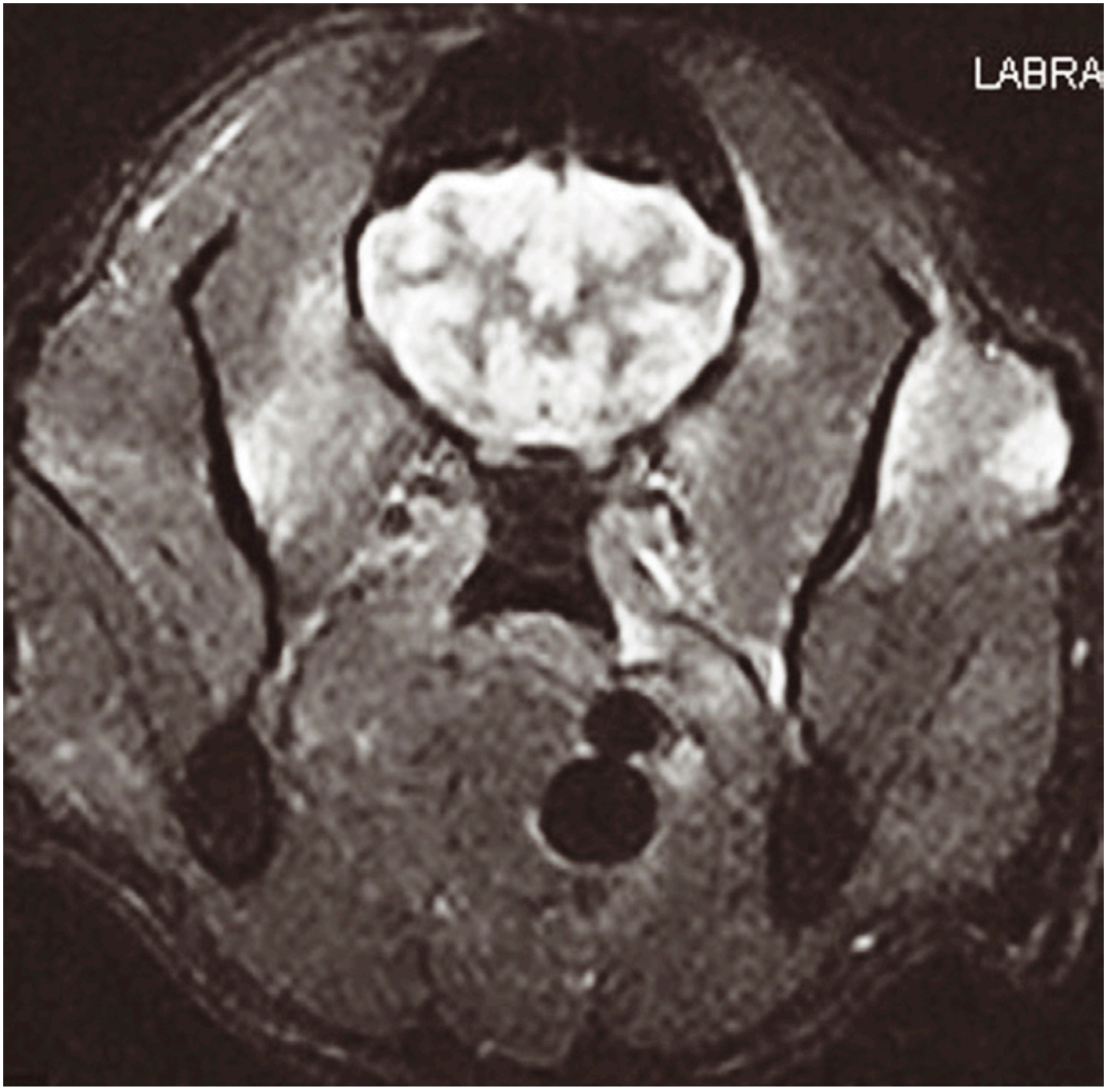

Figure 3. Labrador retriever with MMM. Transverse short TI inversion recovery (STIR) MR image at the same level of Figure 2. The muscle lesions are easily detected and are

heterogeneously hyperintense.

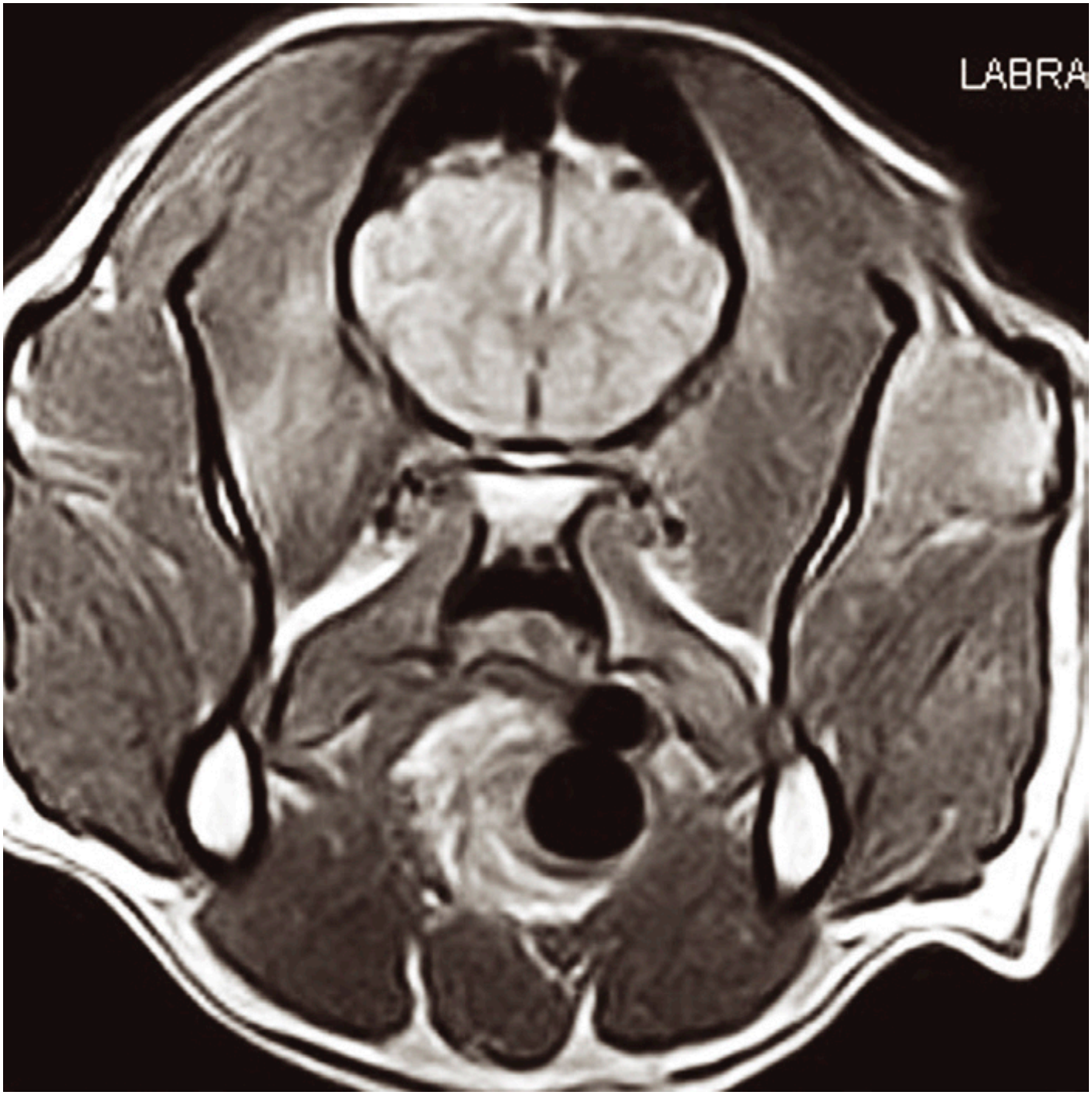

Figure 4. Labrador retriever with MMM. Transverse postcontrast T1-weighted MR image at the same level of Figure 2. The muscle lesions show moderate heterogeneous enhancement.

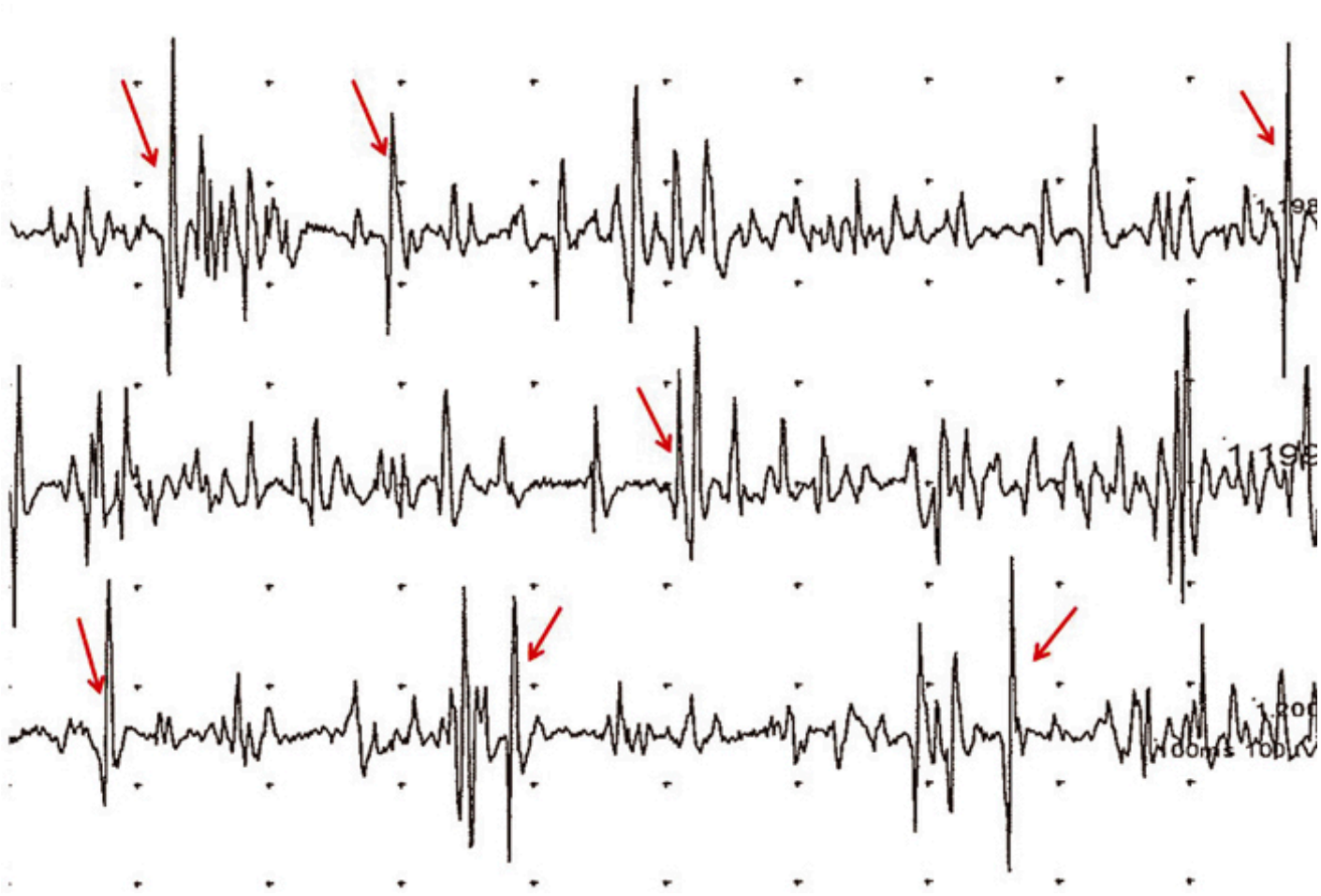

Figure 5. Electromyography (temporalis muscle) from an eight-year-old Labrador retriever with MMM. Note the multiple fibrillation potentials (some of them have been highlighted with red arrows).

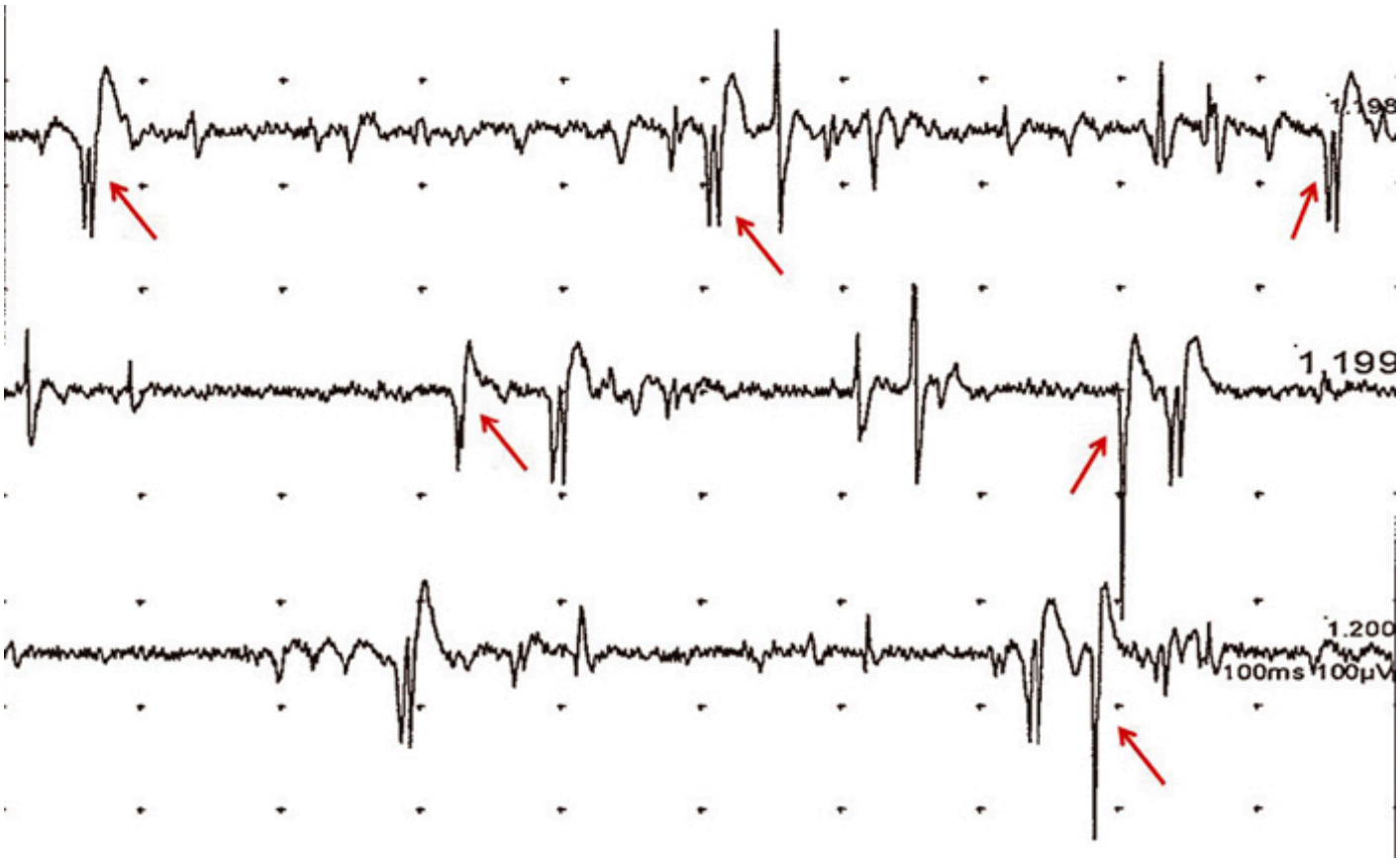

Figure 6. Electromyography (masseteric muscle) from the same dog as in Figure 5. Note the multiple positive sharp waves (some of them have been highlighted with red arrows).

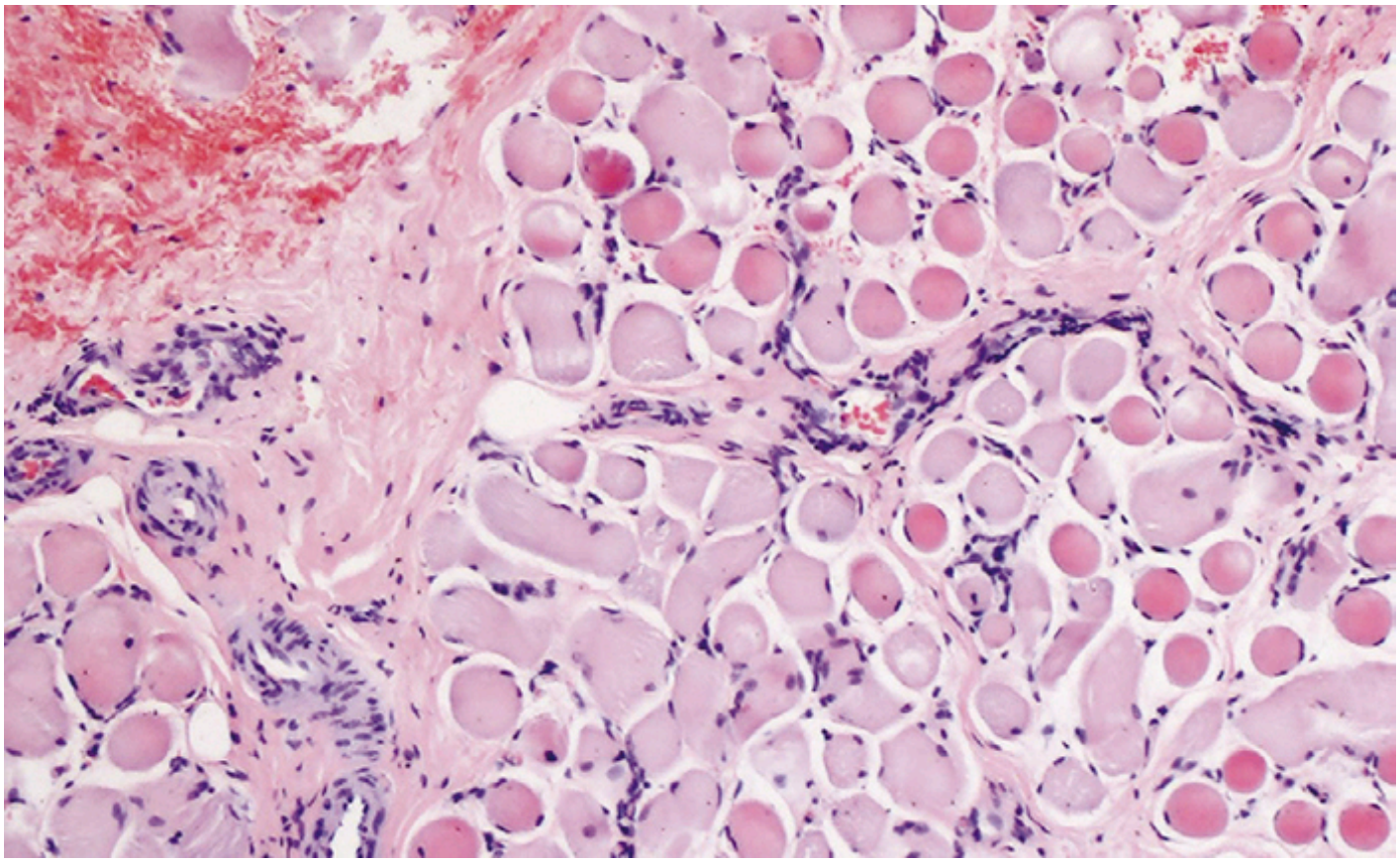

Figure 7. Dog with the acute form of MMM. Note the perivascular infiltrates involving perimisial vessels. (H and E, × 10).

Images: MARIA TERESA MANDARA, FACULTY OF VETERINARY MEDICINE, PERUGIA, ITALY.

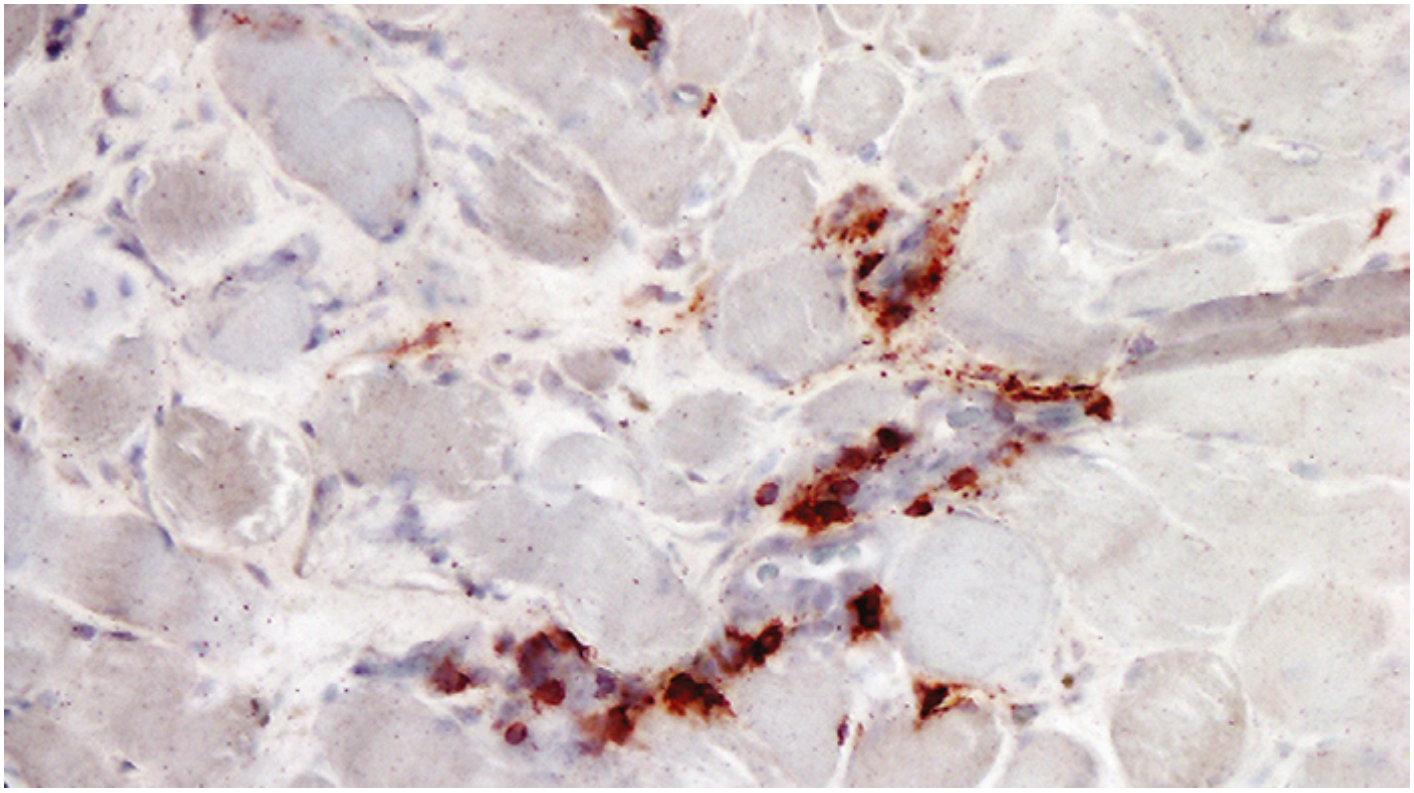

Figure 8. Dog with the acute form of MMM. Lymphocytes B are the mainly represented cells in the inflammatory infiltrates (IHC for CD20, ABC method, Carazzi's haematoxylin counterstain, × 20).

Images: MARIA TERESA MANDARA, FACULTY OF VETERINARY MEDICINE, PERUGIA, ITALY.

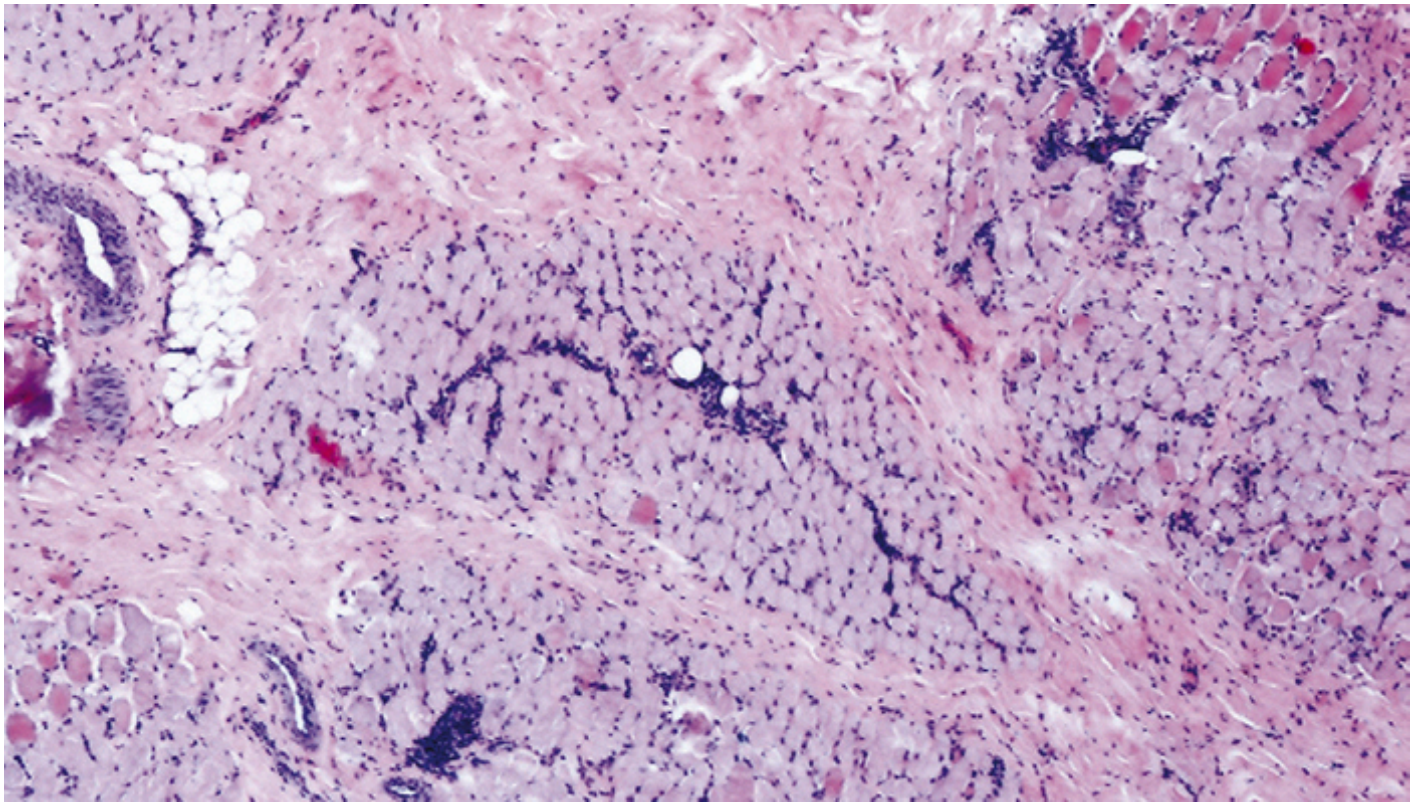

Figure 9. Dog with the chronic form of MMM. Scattered lymphocytic cell infiltrates associated with a severe diffuse perimisial fibrosis (H and E, × 4).

Images: MARIA TERESA MANDARA, FACULTY OF VETERINARY MEDICINE, PERUGIA, ITALY.