MASTICATORY MYOSITIS

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AKA: EOSINOPHILIC MYOSITIS

It may start suddenly one day or come on gradually. The dog seems to be painful when his mouth opens or he attempts to chew. Perhaps he will not open his mouth at all. In time, the muscles around the head (particularly the temple region) hollow out giving the dog a thin faced look. What is happening? There are plenty of possibilities but here is one that must be ruled out.

WHAT IS “TRISMUS?”

In short, trismus is the inability to open the mouth. Regardless of whether or not the act is painful to attempt, the mouth simply cannot be opened. Of course, a dog with a painful mouth may be unwilling to open his jaws and there is no way to ask the dog if simply hurts to open the mouth or if it is not possible to open the mouth. Here are the possible explanations:

- A foreign body may be stuck in the soft tissues of the mouth
- Tetanus infection
- An abscess behind the eye (which almost always stems from an injury inside the mouth)
- Dislocation of the jaw (the jaw can actually be fused closed if there is enough arthritis)
- Polymyositis (a general muscle inflammation)
- Muscular Dystrophy
- Craniomandibular osteopathy (a jaw bone growth abnormality)
- Masticatory Myositis

Sorting these out will require general anesthesia to get the mouth open and check for painful oral conditions (broken teeth, oral foreign body, growths inside the mouth), and radiographs to assess the temporo-mandibular (jaw) joints and jaw bones themselves. General blood tests are also drawn including a special test for Masticatory Myositis (the “2M antibody” test). In more advanced cases of Masticatory myositis the patient’s jaws will not open even under general anesthesia.

Polymyositis is difficult to distinguish from Masticatory myositis. Polymyositis is a more generalized muscle inflammation involving other muscles beyond those of mastication. Polymyositis patients will be negative on the 2M antibody test but so are up to 15% of patients
with Masticatory myositis. A muscle biopsy may be necessary to distinguish these conditions. Electromyography (which measures electrical activity in muscle) may also be helpful.

WHAT ARE “MASTICATORY” MUSCLES?

The masticatory muscles are the muscles used in mastication (chewing). They include the powerful jaw muscles and muscles of the temples (the temporalis muscles, the masseter muscles, the pterygoid muscles and the rostral digastricus muscles). The word “myositis” literally means “muscle inflammation.” No other muscles are affected in Masticatory Myositis.

The masticatory muscles are all innervated by the Mandibular branch of the Trigeminal nerve. Any disease that affects the Trigeminal nerve will lead to marked atrophy of the muscles of mastication. These patients, however, have dropped jaw that cannot stay closed rather than trismus.

WHAT MAKES THESE MUSCLES SO UNIQUE THAT A DISEASE PROCESS WOULD AFFECT ONLY THEM?

Embryologically, the chewing muscles have a special molecular structure because of the unique motor nerve branches that serve them. Chewing muscles contain what are called type 2M muscle fibers, which occur no where else in the body. Masticatory myositis arises when the immune system inappropriately attacks these 2M muscles fibers. What causes the immune system to do this is still unknown.

PROFILE OF THE MASTICATORY MYOSITIS PATIENT

The average patient age is 3 years. The most common breeds are German shepherds, Labrador retrievers, Doberman pinschers, Golden Retrievers, and Cavalier King Charles spaniels. Patients can be of either gender. In the acute phase of the disease, the masticatory muscles are swollen and the eyes appear to bulge due to the swollen pterygoid muscles behind them. There may be a fever and local lymph node swelling at this stage. Results are best if therapy is initiated at this point but unfortunately many owners do not notice the problem until the muscles begin to atrophy and the jaws are rigidly closed making eating difficult.

One would expect symmetrical atrophy and pain but this is often not the case. Lack of symmetry certainly is not evidence against Masticatory Myositis.
THE 2M ANTIBODY BLOOD TEST

Thanks to Dr. G. Diane Shelton at the University of California at San Diego, there is a blood test to make the diagnosis of Masticatory Myositis. Positive results are felt to be accurate in all cases (no false positives have been seen with this test) though up to 15% of patients will falsely test negative. In these patients further testing is needed to reach the correct diagnosis.

TREATMENT

In short, treatment is suppression of the immune system usually through high doses (rather than the more commonly used lower “anti-inflammatory” doses) of prednisone or dexamethasone. High doses should be maintained until the jaw seems to open normally. After that, the dose may be gradually tapered over 6 months. In many cases the drug cannot ever be completely stopped.

Patients on long term prednisone will drink and urinate excessively. Screening for latent bladder infection is important.

If prednisone therapy is problematic, azathioprine can be used to spare the amount of prednisone necessary to achieve remission. Azathioprine is an agent of chemotherapy as well as an immune suppressive agent and is not used lightly. Monitoring blood tests are recommended with long term use.

If therapy is discontinued prematurely, relapse is common. Prognosis is better the earlier treatment begins. If too much scarring has been caused by the inflammation, results are not as good. A muscle biopsy can helpful to assess the extent of the scarring.

Semi liquid diets may be needed to feed the patient with trismus. It is important not to try to force the jaws open but encouraging chewing of toys can be helpful physical therapy.

Results of treatment are best early in the course of the disease. If the disease has progressed to an advanced state before treatment is initiated, there may be no response.

In a study of 18 dogs with Masticatory Myositis: Short term follow up was available in 14/18 dogs. Complete response, i.e., full range of jaw motion regained, was seen in 8/14 with 8/8 treated with immunosuppressive doses of prednisone. Partial response i.e., improved but not full range of jaw movement, was seen in 5/14 dogs--immunosuppressive doses of prednisone were given in 4/5 and an antiinflammatory dose of prednisone given in 1/5. No response was seen in 1/14 who was treated with low dose dexamethasone. Recurrence following initial treatment was seen in 3/13 with partial or complete response initially.

Long term (5 mos to 7 years) follow up was available in 9/14. Eight had no recurrence and good jaw mobility and 5/8 were off all medication, 2/8 died of unrelated causes while still on prednisone, and 1 was still on prednisone 1 year postdiagnosis. The remaining dog was the one who had shown no response--no improvement was seen.