Trismus & Ptyalism

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Eleanor, a 4-year-old, 25-kg (55-lb) spayed golden retriever, was presented after a 2-week history of dysphagia.

History
Starting ≈2 weeks prior, Eleanor had been pushing her food around the bowl when eating. She maintained a good appetite and was interested in eating but had difficulty getting kibbles into her mouth. She was intermittently drooling, and the clients thought her face looked “puffy.”

Eleanor was up to date on distemper, adenovirus, parvovirus, and parainfluenza vaccinations, as well as flea and heartworm preventive. She had been receiving levothyroxine (0.5 mg/kg PO q12h) for hypothyroidism and an otic suspension for otitis externa (date, dose, and duration unknown).

Examination Findings
The patient was bright, alert, and responsive. Heart rate, pulse, respiratory rate and effort, and temperature were within normal limits. BCS was normal at 5/9, and the patient was hydrated. Mucous membranes were pink, and there was moderate ptyalism. A good oropharyngeal examination could not be performed because of trismus, and Eleanor seemed uncomfortable with attempts to open her mouth or palpate her facial muscles or temporomandibular joints. There was mild swelling of the muscles over the top of her head and along the sides of her face (primarily the temporalis and masseter muscles), as well as mild bilateral exophthalmos and reduced movement on retropulsion. Other findings from the physical examination, including musculoskeletal and neurologic evaluations, were normal.

Diagnostic Results
Serum chemistry profile, CBC, and urinalysis were unremarkable except for a mild hypoalbuminemia, hyperglobulinemia, and increases in aspartate aminotransferase and creatine kinase (Table). Results of a 2M antibody titer test were positive at 1:4000 (reference range, <1:100).
Diagnosis
Masticatory muscle myositis (MMM)

This is a focal, immune-mediated, inflammatory myopathy in which the muscles of mastication are specifically targeted by antibodies. These antibodies may directly target 2M muscle fibers with a failure of the immune system to downregulate this self-recognition (ie, a true autoimmune disease), or they may target some foreign antigen, with the capability to also target 2M fibers (sometimes called molecular mimicry or secondary immune-mediated disease).1,2

Treatment
The patient was treated with an immunosuppressive dose of prednisone at 2 mg/kg/day (25 mg PO q12h). In addition, it was recommended that the client try feeding a gruel-consistency food in the short term.

Outcome
Within 3 days of starting prednisone, Eleanor appeared more comfortable and the facial muscle swelling had resolved. At a recheck 2 weeks later, she was doing well clinically but had evidence of mild atrophy of the masticatory muscles. She was still not able to open her jaw fully (although enough to prehend and eat kibble food again). After an additional 2 weeks, there was no further improvement, and the prednisone dose was reduced by 20% to 1.6 mg/kg q24h for another month, then reduced further at monthly intervals until being discontinued after 6 months.

As with many cases of acute MMM, this dog responded well to immunosuppressive doses of steroids but had some residual clinical signs, most likely caused by fibrosis and atrophy of the masticatory muscles. In cases in which the initial or acute phase of the disease is not recognized, patients usually have severe atrophy by the time they are presented—and, although there may still be some improvement with medications, some dogs are never able to open their jaws significantly.

Take-Home Points
1. Several conditions can be associated with jaw pain and trismus in dogs (see Differentials for Trismus, next page). Some are caused by local disease processes, whereas others may reflect a more widespread disease in which trismus is only one manifestation.
2. The signalment and a thorough physical examination can be helpful in narrowing the list of possible causes. MMM is most commonly reported in young to middle-aged, large-breed dogs, without evidence of a sex predilection,1,2 though it should be kept on the differential list for almost any age or breed. Physical examination typically confirms bilateral changes restricted to the muscles of mastication, although unilateral changes are sometimes reported. Regional lymphadenopathy or fever has occasionally been reported. Evidence of muscle pain or neurologic deficits in other parts of the body would be more consistent with other differentials for trismus.

| TABLE | LABORATORY RESULTS |
|-----------------|-------------------|-----------------|
| **Diagnostic Test** | **Result** | **Reference Range** |
| Serum chemistry profile | | |
| Albumin (g/dL) | 2.7 | 2.9-3.8 |
| Globulin (g/dL) | 5.9 | 2.2-4.2 |
| AST (IU/L) | 84 | 15-52 |
| CK (IU/L) | 713 | 49-244 |
| 2M antibody titer | 1:4000 | <1:100 |

AST = aspartate aminotransferase
CK = creatine kinase
MMM = masticatory muscle myositis
An oropharyngeal examination conducted under anesthesia may identify drainage from a retrobulbar abscess or help differentiate between a physical impairment to opening the jaw (ie, true trismus) and resistance to opening the jaw secondary to pain. However, caution must be exercised when using sedatives or anesthetics in patients with trismus, as they may be difficult to intubate and may be at increased risk for regurgitation. Radiography of the head may help identify temporomandibular joint abnormalities; CT or MRI may be useful in identifying abnormalities in the bones or muscles of the head. Finally, electromyography can help differentiate MMM from polymyositis and other neuromuscular disorders.

The positive 2M antibody test confirms the diagnosis of MMM. This test is 100% specific, as type 2M muscle fibers appear to be found only in muscles of mastication. Some dogs with MMM may have a false-negative test result (sensitivity, 64%-90%) and require further diagnostic testing. False negatives are more likely in patients that have received immunosuppressive medications and in those with chronic disease in which there may be dramatic fibrosis and loss of myofibers in affected muscles.

For cases in which MMM is suspected but 2M antibody titers are negative, temporalis muscle biopsy can confirm diagnosis. Biopsies can also provide information about disease severity and prognosis. It is important to avoid taking the biopsy sample from the frontalis muscle, which is not affected by the immune response in MMM.

Immunosuppressive therapy remains the cornerstone of MMM therapy. Corticosteroids are most commonly used. For cases in which steroids are contraindicated or side effects are excessive, other immunosuppressive medications (eg, azathioprine, cyclosporine, mycophenolate, leflunomide) may be used. Immunosuppression is typically recommended at least until there is no further improvement in the degree to which the jaw can be opened and serum creatine kinase levels have returned to normal. Doses of the immunosuppressive medication may then be slowly tapered but are typically not completely discontinued for at least 4 to 6 months. Forcibly opening the jaw with the patient anesthetized is not recommended.

Differentials for Trismus

- Masticatory muscle myositis
- Temporomandibular joint disease (eg, arthritis), luxation, or fracture
- Craniomandibular osteopathy
- Retrobulbar abscess
- Foreign body reaction
- Neoplasia
- Tetanus
- Muscular dystrophy
- Conditions that cause severe pain and reluctance to open the jaw but not actual trismus:
  - Polymyositis
  - Extraocular myositis
  - Dermatomyositis

References