

IMPORTANT MUSCLE DISEASES IN THE VIZLA DOG: WHAT MY VETERINARIAN SHOULD KNOW TO REACH A CORRECT DIAGNOSIS

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Immune-mediated diseases affecting muscle are diagnosed with increasing frequency in dogs. Many of the muscle diseases are treatable if diagnosed early and treated appropriately. A syndrome of wasting of the muscles of the head, problems swallowing and drooling, and in some cases, generalized weakness, has recently been identified in Vizsla dogs. The important thing for veterinarians to know when evaluating dogs with this clinical presentation is that there are at least 3 different immune-mediated or presumed immune-mediated diseases that can cause similar clinical signs, and all of these diseases can affect the Vizsla. This article describes what testing can be performed to differentiate these diseases as treatments and long term prognosis may differ.

Masticatory muscle myositis (MMM, aka eosinophilic myositis or atrophic myositis) – MMM is an autoimmune disease resulting from inflammation (myositis) of head muscles (masticatory muscles) which are important in prehending (picking up balls and toys, food) and chewing. Most commonly, patients present with jaw pain, inability to open the jaw, and either swelling or wasting of these muscles, depending on the stage of disease. Remaining body musculature is generally spared. Diagnosis of MMM is made by a simple blood test (2M antibody test), which

detects autoantibodies against specific muscle proteins. In some cases, a muscle biopsy may be required. MMM is a treatable disease if diagnosed early and treated appropriately. However, MMM can be disfiguring and jaw function impaired if left untreated.

Myasthenia gravis (MG) – MG is an autoimmune disease in which muscle weakness is the predominant abnormality. Clinical presentation of canine MG may vary from localized signs including swallowing difficulties, gagging and regurgitation from an enlarged esophagus, to generalized muscle weakness and collapse. The voluntary muscles of the body are controlled by nerve impulses that arise in the brain. These nerve impulses travel down the nerves to the place where the nerves meet the muscle fibers, the neuromuscular junction (NMJ). When the nerve impulse reaches the nerve ending a chemical is released that binds to a receptor on the muscle signally the muscle to contract. In MG, the immune system of the patient makes antibodies against this receptor protein at the NMJ. These autoantibodies cause destruction of the specific receptors and ultimately the muscles are unable to contract. Diagnosis of autoimmune MG is also made by a simple blood test (Acetylcholine receptor antibody test) which detects autoantibodies against these muscle receptors. MG is a treatable disease in the dog if diagnosed early and treated appropriately.

Polymyositis (PM) – Muscle inflammation with clinical signs of atrophy of the masticatory muscles, difficulty swallowing with excessive drooling, and an enlarged esophagus causing regurgitation (Important! This is not vomiting), has recently been identified in several young adult Vizsla dogs from the UK (for information on the UK dogs go to <http://vizslamyositis.blogspot.com/>) and from the USA (<http://vetneuromuscular.ucsd.edu>,

September 2007 Case of the Month). Although atrophy of the masticatory muscles is a prominent clinical sign, there may also be loss of muscle mass over the entire body, and exercise intolerance. The serum creatine kinase concentration, an indicator of muscle damage, is usually elevated. Dogs with PM should not be confused with MMM, as clinical signs in PM involve many muscle groups, and the serum antibody titer against masticatory muscle type 2M fiber proteins are negative. The serum acetylcholine receptor antibody titers for MG are also negative. Electrodiagnostic studies performed on dogs with PM have indicated generalized abnormalities of muscle with normal nerve function. Muscle biopsies are critical for confirmation of a diagnosis of PM and for accurate classification of this disease. Many of the affected Vizsla dogs in the UK were related, suggesting a possible breed predisposition to this myopathy such as occurs in the Newfoundland breed. Information is not yet available on USA dogs. While the underlying cause of this inflammatory myopathy is not yet known, an immune-mediated disorder is suspected.

I am interested in identifying additional Vizsla dogs with MMM, MG and PM in the USA (contact musclelab@ucsd.edu). Animal owners should not contact the laboratory directly, as for legal reasons, we cannot discuss the diagnosis or clinical management of pets directly with their owners. Please ask your veterinarian to contact the laboratory and provide a complete history and results of blood evaluations on any suspected affected dogs. A muscle biopsy will be required to confirm the diagnosis of PM, and I would be happy to speak with your veterinarian regarding appropriate sample collection. Only with complete evaluations of these dogs can we learn more about these disabling, disfiguring and possibly life threatening conditions, identify optimal treatments, and investigate possible inheritance patterns which would be helpful to

breeders. In addition, please complete the Vizsla Club of America Welfare Foundation 2008 Health Survey (<http://ipgwcua.org/vizsla/>). This will greatly help us determine the incidence of MMM, MG and PM in the Vizsla population.