BREED SPECIFIC POLYMYOSITIS IN THE HUNGARIAN VIZSLA DOG. AC Haley[1], SR Platt, M Kent, SJ Schatzberg, A Durham, S Cochrane, GD Shelton. 1. University of Georgia, College of Veterinary Medicine, Athens GA. 2. University of Pennsylvania, School of Veterinary Medicine, Philadelphia, PA. 3. Veterinary Emergency Clinic and Referral Centre, Toronto, Ontario Canada, 4. University of California, San Diego, Department of Pathology, School of Medicine, La Jolla, CA.

Inflammatory myopathies are relatively common in dogs and may have a focal (masticatory muscle myositis) or generalized (polymyositis) distribution. Recently, a breed specific myositis presenting as pharyngeal dysphagia and masticatory muscle atrophy has been described in 14 Hungarian Vizsla dogs in the United Kingdom (Foale et al, BSAVA 2008). Here we report a similar syndrome in 5 Vizsla dogs from the North America. This retrospective study provides a preliminary clinicopathologic description and outcome in these 5 dogs.

Five Viszla dogs presented to 5 different veterinary hospitals (1 general practice, 2 referral practices, 2 university practices) with clinical signs of dysphagia (3/5), regurgitation (3/5), excessive salvation (3/5), masticatory muscle atrophy (4/5) and pain on opening the jaw (2/5). All dogs were male (4/5 castrated, 1/5 intact) and ranged in age from 1 to 9 years (mean 5.2 years). Creatine kinase activity was measured in 3 cases and was elevated (range 1061-9758 U/L; mean 5443). Antibodies against type 2M fibers and acetylcholine receptors were negative in those dogs tested (3/3 and 2/2 respectively). Three dogs had radiographically evident megaesophagus (ME). Two dogs had ME at the time of initial presentation while one dog developed ME 19 months after onset of dysphagia. Serum antibody titers against *Toxoplasma gondii* and *Neospora canis*, *Borrelia burgdorferi*, *Ehrlichia canis* and *Ehrlichia equi* were negative when tested. Electromyography was performed in 2 dogs with no abnormalities. Histopathologic examination of temporalis muscle biopsies was performed in 3 cases with multifocal areas of lymphocytic infiltration in 2 cases. Although cellular infiltrates were not evident in one case, bilateral multifocal hyperintensities in the temporalis muscle were observed on T2-weighted magnetic resonance images (MRI). A complete necropsy was performed on the fifth case and chronic lymphohistiocytic and plasmacytic myositis and fibrosis was evident in the esophagus, myocardium and skeletal muscle. Immunosuppression with prednisone and azathioprine has not resulted in clinical improvement at 2 and 3 months follow up in 2 dogs. Two dogs were lost to follow up. In conclusion this study, in combination with the previous report from the UK, should alert clinicians to the occurrence of a new breed associated polymyositis in the Vizsla dog which warrants further investigation to elucidate pathogenesis, genetic associations, response to treatment and prognosis.