Idiopathic Myofasciitis in the Domestic Ferret

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Session #132

Summary Style Manuscript

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Introduction

Spontaneous inflammatory polymyopathies are well documented, especially in humans[1] and dogs.[2] Experimentally or iatrogenically-induced inflammatory myopathies also occur in animals.[3] The domestic ferret (*Mustela putorius*) is a popular house pet in the United States. During an 11-year span (1994–2005), Northwest ZooPath (NZP) has received 2040 domestic ferret biopsy or necropsy submissions, most of which were privately owned as pets. Since late 2003, a previously unrecognized inflammatory disease process involving muscle and fascia has been diagnosed in several ferrets at NZP.[4]

Signalment and Clinical Signs

Affected ferrets ranged in age from 5–24 months of age with an average age of 10 months. There is no gender bias. Clinical signs include high fever, lethargy, recumbency, ataxia, posterior paresis or pain when moving, inappetance or anorexia, and abnormal stools. Blood work reveals mild to marked leukocytosis with mature neutrophilia, and mild to moderate, usually nonregenerative, anemia. Common serum abnormalities include mild to moderate elevation of ALT, mild hyperglycemia, and hypoalbuminemia. Treatment, which has included various antibiotics, anti-inflammatory drugs, glucocorticoids, antipyretics, pain killers, interferon, and cyclophosphamide, has been ultimately unsuccessful in all cases and the patients have either died or were humanely euthanized.

Gross Lesions

Gross lesions include red and white mottling and dilatation of the esophagus, and white streaks in the heart, diaphragm, and intercostal muscles. Generalized muscle atrophy may be prominent even in animals with adequate fat stores. Fat may have red mottling. Lungs are sometimes congested. The spleen is usually markedly enlarged and pale.

Histologic Lesions

Histologic changes include moderate to severe suppurative to pyogranulomatous inflammation involving the skeletal muscle and blood vessels at multiple sites, particularly the esophagus, heart, and muscles of the hind limbs and lumbar region. Myleoid hyperplasia of spleen and/or bone marrow, hepatitis, pneumonia, and mediastinitis are also prominent features.

Ancillary Procedures

Bacterial and viral cultures have been negative for pathogens. Electron microscopy, PCR, and immunohistochemistry have been negative for various infectious agents.

Conclusions

The etiopathogenesis of polymyositis in ferrets is not known. It is a fatal disease of young adult ferrets characterized by rapid onset of clinical signs, high fever, neutrophilic leukocytosis, treatment failure, and death (or euthanasia). The distribution of histologic lesions, particularly in the esophagus, suggests that this is likely a single distinct entity. Cultures and extensive microscopic examination have failed to detect infectious agents. The only commonality in affected ferrets is administration of a particular brand of canine distemper vaccine, which is no longer available. Interestingly, the disease has been inadvertently reproduced during an experimental vaccine trial by one of the authors (N. J. S.).

Acknowledgments: The authors are grateful to the following for submission of cases: Brantley and Jordan Animal Hospital, Macon, GA; Ocean State Veterinary Specialists, East Greenwich, RI; Somers Animal Hospital, Somers, NY; Essex Animal Hospital, Bloomfield, NJ; Midwest Bird and Exotic Animal Hospital, Westchester, IL; Rutherford Animal Hospital, Rutherford, NJ; Affordable Pet Clinic, Nampa, ID; All Pet Complex, Boise, ID; South Wilton Veterinary Clinic, Wilton, CT; Bird and Exotic Pet Hospital, Midvale, UT; and Tennessee Valley Veterinary Clinic, Tuscumbia, AL.

References

1. Cherin P. Inflammatory myopathies. *Acta Clin Belg*. 2004;59:290–299.

2. Evans J, Levesque D, Shelton GD. Canine inflammatory myopathies: a clinicopathologic review of 200 cases. *J Vet Intern Med*. 2004;18:679–691.

3. Ytterberg SR. Animal models of myopathy. *Curr Opin Rheumatol*. 1991;3:934–940.

4. Garner MM, Ramsell K, Schoemaker NJ, et al. Myofasciitis in the domestic ferret. *Vet Pathol*. In press.