



International Conference on Avian Herpetological and Exotic Mammal Medicine

0100 - Exocrine pancreatic insufficiency secondary to pancreatic acinar atrophy in a domestic ferret (*Mustela putorius furo*).

Jacobo Giner¹, Carles Juan-Sallés²

¹Centro Veterinario Menescalía, Valencia, Spain ²Noah's Path, Elche, Spain

A 2-year-old intact female ferret (*Mustela putorius furo*) was clinically evaluated because of a progressive weight loss, bulky stools with undigested food, and incremented appetite over three months' period. On physical examination, this ferret appeared to have a poor body condition and hair coat, as well as abdominal distension. Diagnostic tests performed included CBC, serum biochemistry, abdominal radiographs, abdominal ultrasonography, fecal flotation, total serum thyroxine (T4), and serum cobalamin and folate concentrations. Abnormal clinicopathologic findings included slightly hyperglobulinemia and marked increased serum folate concentrations, with normal serum cobalamin values. A presumptive diagnosis of exocrine pancreatic insufficiency (EPI) was made based on physical exam findings, laboratory analysis and lack of response to antibiotic therapy and dietary modifications during eight weeks. The ferret was treated with pancreatic enzyme replacement therapy and vitamin supplements. At reevaluation three months later, the ferret had an improved body condition, partial decrease in stool volume, and an almost normal hair coat. Six months later, this ferret was presented with significant weight loss, generalized weakness, lethargy, poor hair coat and severe seborrhea oleosa. Due to the ferret's poor condition, it was euthanized humanely. A complete necropsy and histopathology revealed marked pancreatic acinar atrophy, which confirmed the clinical diagnosis. Mild lymphoplasmacytic gastroenteritis was detected as an incidental finding. To the best of the authors' knowledge, this is the first description of EPI secondary to pancreatic acinar atrophy in a domestic ferret. The age of onset rules out pancreatic acinar hypoplasia and suggests a juvenile-onset of progressive pancreatic acinar atrophy.