Multisystemic eosinophilic epitheliotropic disease in a horse

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A multisystemic, eosinophilic, epitheliotropic disease of horses with one or more of: eosinophilic gastroenterocolitis, eosinophilic pancreatitis or eosinophilic dermatitis as the major clinicopathological manifestation has been reported from Australia (1,2), Canada (3), the USA (4), and the UK (5) in recent years. Another horse with this distinctive syndrome is reported here.

A five-year-old, Standardbred stallion was euthanized and presented for necropsy after six weeks of chronic, worsening diarrhea and patchy alopecia. Initial therapy by the attending veterinarian consisted of the combination of 5 mg dexamethasone, 2,000,000 IU procaine penicillin G, 2,500 mg dihydrostreptomycin sulfate and 50 mg chlorpheniramine maleate (Azymycin, Schering, Pointe Claire, Quebec) intramuscularly (IM) daily for two days, followed by 2,000 mg trimethoprim and 10,000 mg sulfadoxine (Borgal, Hoechst, Montreal, Quebec) IM, and 200 units bacitracin methylene disalicylate, 20 mg streptomycin sulfate and 850 mg roasted powder carob pulp (Entromycin, M.T.C. Pharmaceuticals, Mississauga, Ontario) orally one week later. No improvement was noted. The horse lost condition rapidly. The alopecia progressed and the skin became cracked with caked ulcers over most of the body. At necropsy, there was marked hyperkeratosis of the esophagus and nonglandular stomach, marked fibrotic thickening of the duodenal and ileal walls, the common and intrahepatic bile ducts, and extensive, firm, nodularity and enlargement of both arms of the pancreas. The mesenteric and cecocolic lymph nodes were conspicuously enlarged. The thickened wall of the terminal ileum caused a constriction of the lumen. For about one meter cranial to this functional blockage, the ileum, walls hard and thickened, was dilated and filled with ingesta. Bacteriological cultures of abdominal organs were unremarkable. Fecal flotation revealed small numbers of strongyle eggs.

Tissues were fixed in 10% neutral buffered formalin, sectioned at 6 μm, and stained with hematoxylin and eosin (H & E). Histologically, the lesions were similar qualitatively, but varied in severity among and within organs. Typically, a chronic, fibrosing, inflammatory reaction characterized by diffuse and focally intense eosinophilic infiltrates mixed with lymphocytes, plasma cells, macrophages and mast cells enveloped the esophagus, nonglandular and glandular stomach, duodenum, ileum, cecum and colon, mesenteric lymph nodes, pancreas, intra- and extrahepatic bile ducts, and skin. Infiltrates and fibrosis were transmural in the esophageal and gastroenteric sections, and perportal in the liver. Portal fibrosis and biliary proliferation were marked. Eosinophilic abscesses and microgranulomas surrounded by giant cells were randomly scattered in lesions in all organs (Figure 1). Orthokeratotic hyperkeratosis and epidermal hyperplasia with focal ulceration were pronounced in the skin, esophagus and nonglandular stomach. Lesions in the pancreas occurred in two distinct patterns consisting of: a modest diffuse infiltrate of a mixture of eosinophil-rich inflammatory cells with only modest disruption of the glandular architecture; and a more dense infiltrate of similar inflammatory cells with numerous randomly scattered eosinophilic abscesses and microgranulomas, and marked disruption of architecture (Figure 1). There was also an arteritis of small and medium-sized arterioles characterized by hypertrophied tunica media with an unrolled "onion-skin" appearance, and infiltrates of small numbers of mononuclear cells in the inflamed tissues. Parasites or parasitic remnants were not seen in tissues.

The cause of these widespread lesions is unknown. The lesions resemble an on-going, immediate hypersensitivity reaction (1-3). Ingested or inhaled allergens and parasites have been suggested as initiating causes of this multisystemic eosinophilic epitheliotropic disease in horses (1-5). Encouraging response by two horses with similar lesions to long-term and high-dosage glucocorticosteroid therapy (2) further supports an allergic etiology. The authors have suggested the following corticosteroid therapy for this condition (2):

"Initial stabilizing dosage" — 0.2 mg/kg bodyweight injectable dexamethasone sodium phosphate once daily for five days.

"Maintenance dosage" — 0.55 mg/kg bodyweight oral prednisolone twice daily for seven days, 1.1 mg/kg oral prednisolone once daily for seven days, 1.1 mg/kg oral prednisolone on alternate days for seven days. The alternate day dosage should be...
reduced to the lowest level which will control the clinical signs. This horse did not respond to therapy, but steroids were used at a very low dose and only for two days. As this condition becomes more clinically recognized, it will be interesting to see if steroid therapy continues to be efficacious.

References

Abstract
Sensitivity and specificity of canine serum total amylase and isoamylase activity determinations.
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Serum isoamylases were determined prospectively in dogs with pancreatic and extrapancreatic diseases. Mean serum isoamylase determinations were significantly different (p < 0.05) between normal dogs and dogs with pancreatitis and exocrine pancreatic insufficiency. The sensitivity of serum isoamylase determination exceeded that of total amylase activity for the diagnosis of pancreatitis. Serum isoamylase determinations were less influenced by extrapancreatic diseases compared to total amylase activity when used in the diagnosis of pancreatic disease. Neither serum isoamylase determination nor total amylase activity had adequate sensitivity to support their use in the diagnosis of exocrine pancreatic insufficiency. There were significant (p < 0.05) linear correlations between isoamylase determinations, total amylase activity, and trypsin-like immunoreactivity concentration.