The morphologic changes in idiopathic epilepsy are largely considered to be the result of epileptic attacks, but the lesions themselves may cause seizures. Similar lesions are also induced by hypoxic conditions. In some cases, the seizure activity and the consequent ischemic events may play a role in the development of lesions occurring in status epilepticus; however, in another study on experimental epilepsy, the strict control of blood pressure and oxygen tension has not been able to prevent the formation of brain lesions. Once produced, the lesions may become an epileptogenic focus, resulting in extensive brain damage.

References

Chronic Eosinophilic Enteritis Attributed to Pythium sp. in a Horse


Key words: Eosinophilic enteritis; horses; Pythium sp., pythiosis.

Pythiosis in the horse is usually a local subcutaneous infection, often accompanied by cutaneous ulceration and fistulous tracts. It is characterized by the presence of exuberant granulation tissue containing granular yellow or yellow-gray cores called “leeches” or “kunkers.” Eosinophils are the most common inflammatory cell, and the cores consist of degenerating or necrotic eosinophilic debris. A marked granulomatous reaction usually is present around the cores. Recently, two cases of equine enteric pythiosis were described. In both cases, there was a stenotic jejunal mass that consisted of dense fibrous connective tissue, containing granulomas or necrotic cores that were surrounded by granulomatous inflammation. In one case, variably intense infiltrates of eosinophils were present. We describe a case of equine enteric pythiosis in which eosinophils were the predominant inflammatory cell.

A 7-year-old Arabian gelding was presented to the University of Illinois Veterinary Teaching Hospital with a 36-hour history of colic that failed to respond to medical treatment. Rectal palpation revealed colonic impaction. Large bowel tympany developed, and the horse was taken to surgery. A ventral midline laparotomy revealed three abnormalities: an impaction of the diaphragmatic flexure, gastric and large bowel tympany, and a mid-jejunal intramural soft tissue mass. After correcting the tympany and colonic obstruction, approximately 42 cm of the jejunum, including the mass, were removed and an anastomosis performed. The horse recovered uneventfully and was doing well, with no further episodes of colic, 1.5 years after surgery.

The jejunal mass was nodular, intramural, measured 3 × 4 × 5 cm, and occupied 320° of the intestinal circumference. The overlying mucosa was ulcerated with green-brown fibrillar material that adhered to the ulcerated surface. Histologically there was focally extensive mucosal ulceration overlying multiple irregularly shaped, 2- to 7-mm diameter intramuscular nodules. The nodules consisted of a central core of granular eosinophilic debris (predominantly necrotic eosinophils) surrounded by degenerating eosinophils and neutrophils with a few macrophages and an occasional multinucleated giant cell. Fragments of plant material were present within some of the eosinophilic cores. Colonies of coccobacilli were present at the peripheries of some nodules. Dense granulation tissue containing many eosinophils, lesser numbers of lymphocytes and plasma cells, and occasional


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small granulomas separated the nodules and extended between remnants of the circular and longitudinal muscle layers to the subserosa. There was moderate, multifocal subserosal hemorrhage. Moderate to marked villous atrophy and fusion were present in the intact jejunal mucosa adjacent to the ulcer. The lamina propria was congested and edematous with dense infiltrates of eosinophils and fewer numbers of lymphocytes and plasma cells. Some vessels in the lamina propria contained fibrin thrombi. The endothelium underlying the thrombi was absent, and there was focal subjacent proliferation of perivascular spindle cells with intercellular infiltrates of degenerating neutrophils.

Examination of the necrotic, eosinophilic coagula revealed occasional clear, nonstaining “shadows” of fungal hyphae in some nodules. In sections stained with hematoxylin and eosin, the hyphae appeared as irregularly branching empty spaces with nonparallel sides 4–7 µm wide (Fig. 1). Sections stained with Gomori’s methenamine silver showed the hyphae somewhat more clearly, although staining affinity was poor (Fig. 1, inset). These hyphae were irregularly branching, 4–7 µm in diameter, rarely septate, and had thick, nonparallel walls. Elastin fibers around the hyphae also stained black, partially obscuring the fungal organisms. The morphologic appearance of the fungal hyphae was compatible with that described for Pythium sp. The organisms were positively identified as Pythium by the use of immunohistochemistry.3

The morphologic diagnosis was chronic eosinophilic enteritis. The presence of plant material within these lesions suggested that the infection may have originated from a penetrating intestinal wound. Intestinal mucosal lesions due to common equine enteric pathogens might also contribute to the development of intestinal pythiosis, since damaged plant or animal tissues are chemotactic for Pythium zoospores.5

Chronic eosinophilic gastroenteritis of undetermined etiology has been reported in horses from Australia and Texas, geographic areas where pythiosis is endemic. The intestinal lesions reported for that syndrome were quite similar to those present in this case.29 The lesions described for chronic eosinophilic gastroenteritis were more diffuse, however, often also affecting the pancreas and liver. Some cases had cutaneous (coronary band) involvement. A clinical syndrome characterized by diarrhea and weight loss due to malabsorption was described. Parasitic migration and hypersensitivity reactions were considered as possible etiologies, although no parasitic fragments or ova could be identified in affected animals.

This case demonstrates that Pythium should be considered as a possible etiologic agent when examining chronic eosinophilic visceral lesions in horses. The organisms can be very difficult to identify. They may be present in small numbers, necessitating examination of multiple sections, and Pythium hyphae, like those of Conidiobolus and Basidiobolus, do not stain with hematoxylin and eosin.3,6,8 Stains and reactions typically used to identify fungal organisms, such as Gomori’s methenamine silver and periodic acid–Schiff, may also be unsatisfactory. Immunohistochemical examination appears to be the most promising method for both demonstrating the organisms within suspicious lesions and identifying them definitively as Pythium sp.3

Although equine zygomycosis is most commonly observed in semitropical climates, this horse was born and raised in Illinois. We have subsequently diagnosed cutaneous pythiosis in another Illinois horse. Any lesion containing eosinophilic nodules, therefore, whether cutaneous or visceral, should be closely examined for Pythium sp. or a zygomycete agent if no other etiology is apparent.

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References


Bacterial Histiocytic Colitis in a Lowland Gorilla (Gorilla gorilla gorilla)

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Key words: Colitis; gorillas.

In 1980, Molteni et al. described a case of “chronic idiopathic diarrhea” in a captive lowland gorilla (Gorilla gorilla gorilla) from the Lincoln Park Zoological Gardens (Chicago, IL).3 Those authors chose to interpret the illness as a small bowel disorder, but the histologic slides and published photographs of villi were unconvincing; furthermore, the light and electron microscopic evidence we had developed suggested that a pronounced colitis was responsible for the diarrhea, wasting, and ultimate death of the animal. Here, we briefly reiterate the clinical and laboratory data and show these additional findings.

The affected individual was a 10-year-old female gorilla that had been born in Cameroon, West Africa, and raised in captivity. Clinical signs first seen in 1975, when the animal was 7 years old, consisted of persistent, watery diarrhea, gradual weight loss, and hypochromic anemia. Various treatments tried at that time included a lactose and gluten-free diet, diphenoxylate hydrochloride, diiodohydroxyquin, chloramphenicol, and sulphasalazine. The diarrhea persisted despite these treatments and, in 1976, additional diagnostic tests were initiated. Proctoscopic examination to a depth of 25 cm revealed no abnormalities, and none were detected in radiographic studies following both oral and rectal barium administration. A peroral small bowel biopsy was attempted without success. Fecal examinations revealed numerous Balantidium coli and Troglodytella gorillae, of which the latter was soon controlled by medication. In October 1976, a fecal examination proved positive for neutral fats, but this was an isolated finding that could not be reproduced subsequently.

The gorilla’s condition continued to deteriorate and, in the spring of 1977, additional treatments were attempted. Oral prednisolone (20 mg twice daily) and a liquid potassium supplement induced a mild but temporary improvement. Brewer’s yeast and tylosin were included in the diet, but produced no clinical changes. At this time, the blood urea nitrogen (182 and 267 mg/dl) and serum creatinine (5.6 and 6.0 mg/dl) taken 3 days apart and the serum glutamic oxaloacetic transaminase (62 U/liter) were elevated. Throughout the course of the illness, serum concentrations of glucose, calcium, bilirubin, glutamic-pyruvic transaminase, alkaline phosphatase, and uric acid were normal.2 Serum concentrations of sodium, potassium, chloride, phosphorus, magnesium and albumin, and also blood hemoglobin, packed cell volume, and lymphocyte counts were all abnormally low; serum cholesterol, total protein, and serum gamma globulin concentrations were elevated.2 Blood leukocyte and neutrophil counts were also elevated. Thyroid function, while normal initially, was severely depressed late in the disease (T₄, 2.0 μg/dl, T₃, 34%, T₄ iodide 0.4 μg/dl).

By September 1977, the animal was in extremely poor condition and was euthanatized. At necropsy, the investigators2 found what they described as thinning of the jejunum and ileum, with partial loss of villi and diffuse petechiation. Small hemorrhagic areas occurred in the colon and cecum. Several mesenteric lymph nodes were enlarged. Microscopic examination of the jejunum and ileum were said to show a broadening and fusion of villi, with a diffuse lymphocytic and plasmacytic infiltration. Electron microscopic examination revealed spherical microorganisms (1–2 μm in diameter) in the jejunal epithelial cells. Based on these findings, the previous investigators reported that the gorilla had developed a malabsorption disorder similar to human sprue.2

Pursuant to a consultation while the gorilla was alive, we received 2 × 2 color transparencies of all gross lesions and formalin-fixed tissues of all organs. Tissues were embedded in paraffin, cut at 6 μm, and stained with hematoxylin and...