

ERYSIPELOTHRIX RHUSIOPATHIAE INFECTION IN A CAPTIVE BALD EAGLE (*HALIAEETUS LEUCOCEPHALUS*)

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Abstract: An adult bald eagle (*Haliaeetus leucocephalus*) kept in captivity for nearly 7 yr at the Patuxent Wildlife Research Center, Laurel, Maryland, died suddenly with gross and microscopic lesions characteristic of septicemia. *Erysipelothrix rhusiopathiae* was isolated from the liver. Fish comprised part of the bird's diet and may have been the source of the organism.

Key words: Bald eagle, *Haliaeetus leucocephalus*, *Erysipelothrix rhusiopathiae*, septicemia.

INTRODUCTION

Erysipelothrix rhusiopathiae can cause acute infection and variable flock mortality rates in domestic poultry, especially turkeys, and has been reported as a cause of disease outbreaks in a variety of captive wild birds.^{2-4,9,10} This organism has also caused epizootic mortality in at least two species of free-ranging wild birds, the little swift (*Apus affinis*)¹² and the eared grebe (*Podiceps nigricollis*).⁷ *Erysipelothrix rhusiopathiae* causes individual deaths of birds more frequently than large-scale mortality and has been isolated from many species.^{1-3,5,8,11} In this report, we describe the death of a captive bald eagle (*Haliaeetus leucocephalus*) resulting from *E. rhusiopathiae* infection.

CASE REPORT

In early April 1977, an adult bald eagle was found in the vicinity of Back Bay National Wildlife Refuge, Virginia, with a wing injury that rendered it flightless. The eagle

was transferred to the Patuxent Wildlife Research Center on 19 April 1977 and maintained in captivity for nearly 7 yr in pens either 3.0 or 6.1 m wide, 15.2 m long, and 1.8 m high and equipped with sheltered perches, stainless steel water pans, and wooden feeding blocks. General feeding and care were similar to that described previously.¹³ The diet included fish about three times weekly. After about 8 mo, the bird developed bumblefoot (pododermatitis) lesions that persisted throughout the remainder of captivity but otherwise remained visibly healthy. The eagle died suddenly on 15 January 1984, with no observed clinical signs.

Pathologic findings

At necropsy, the adult male eagle weighed 2,518 g and had little subcutaneous fat and a slight reduction of the pectoral musculature. The old wing injury consisted of a dislocation of the right humerus. The heart was diffusely pale with light streaks suggestive of necrosis in the epicardium and the myocardium. The atrioventricular valve leaflets were thickened with numerous vegetations (proliferative outgrowths) on their surfaces. Irregular cream-colored 1-2-mm foci were observed on the surface and within the parenchyma of the liver. The spleen was normal in size but uniformly pale. Urates were visible on the cut surfaces of the kidneys. A small amount of brown fluid was present in the stomach, and a large amount of cream-colored fluid was present in the cloaca.

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Histologic examination confirmed the vegetative valvular endocarditis and revealed features consistent with septicemia and acute myocardial infarction. The vegetative valvular lesion was composed almost entirely of fibrinous material and small, slender gram-positive rods. Scattered macrophages were filled with similar gram-positive rods. Necrotic, inflammatory debris, consisting of degenerating lymphocytes and heterophils, was present along the periphery of the vegetative valvular lesion. Acutely ischemic zones of myocardium had regionally extensive coagulation necrosis characterized by hypereosinophilic myofibers with granular and floccular degeneration. Peripheral to these regions were infiltrations of numerous macrophages with occasional erythrocytes, lymphocytes, and plasma cells. Other areas of myocardium had characteristics of more chronic alterations consisting of shrunken irregular myofibers, numerous hemosiderin-laden macrophages, and occasional lymphocytes. Numerous cardiac myocytes had perinuclear accumulations of golden-brown granular pigment consistent with lipofuscin. Microscopic evidence of septicemia included numerous centrilobular and periportal to random foci of hepatic necrosis with accumulations of heterophils and lymphocytes, mild diffuse heterophilic splenitis, and multifocal heterophilic interstitial and glomerular nephritis.

Microbiology

Liver was submitted for routine culture on blood agar. After incubation at 37°C for 18 hr, there was abundant growth of tiny, translucent colonies exhibiting a narrow zone of alpha hemolysis. These colonies consisted of slender, slightly curved gram-positive rods that decolorized readily. The organism was catalase negative, H₂S positive on triple sugar iron, and exhibited characteristic "test-tube brush" motility after 5 days. The isolate was sent to the National Veterinary Services Laboratories, Ames, Iowa, where the identification of *E. rhusiopathiae* was confirmed.

DISCUSSION

The source of *E. rhusiopathiae* in this case may have been the fish that were fed to the eagle. Although food items were not cultured in this case, the organism can be found as a contaminant on fish, and fish or fish meal have been implicated in avian infections.³ *Erysipelothrix rhusiopathiae* was isolated from 11 of 12 brown pelicans (*Pelecanus occidentalis*) during an epizootic that killed an estimated 400 pelicans in California in 1988 (K. A. Converse, pers. comm.). Many of the pelicans had been feeding on discarded fish, from which *E. rhusiopathiae* was also isolated. The suggested routes of entry for *E. rhusiopathiae* include oral, respiratory, and abraded skin.⁶ The eagle could have acquired the infection through any of these routes, quite possibly through abraded skin on the feet associated with the bumblefoot lesions. Bald eagles typically grip food items with their feet while tearing off small pieces of flesh with their beaks. No previous or later problems with *E. rhusiopathiae* have been noted at the Patuxent Wildlife Research Center, although many captive eagles have been kept under similar conditions and fed similar diets.

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