PULMONARY ASPERGILLOSIS IN PRAIRIE FALCON NEST MATES

Authors: WARD, F. PRESCOTT, FAIRCHILD, DAVID G., and VUICICH, JEANNE V.

Source: Journal of Wildlife Diseases, 6(1) : 80-83

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-6.1.80

PULMONARY ASPERGILLOSIS IN PRAIRIE FALCON NEST MATES

Case Histories

Two prairie falcons (*Falco mexicanus*), a male and a female approximately two weeks old, were taken from a Wyoming eyrie containing five nestlings. The birds, to be used for falconry, were raised in separate homes.

Three weeks after capture, their wing and tail primary feathers had matured and training began. A week later the male developed a yellow, necrotic lesion that encircled the middle of his tongue. The owner tentatively diagnosed a *Trichomonas gallinae* infection of the mouth (Stabler and Herman, Trans. 16th N. Am. Wildlife Conf.: 145-163, 1951), and treated the bird with a single 125 mg oral dose of 1,2-dimethyl-5-nitroimidazole.*

Simultaneously, the female became ill; during the five days before presentation, she lost approximately 20% of her body weight, became listless, and the owner reported vague respiratory difficulty. She stopped eating and, when force-fed, regurgitated most of the meat. When presented, the falcon was weak but alert. She was very thin, her eyes were sunken, and respirations were rapid and deep.

The next morning, her feathers were ruffled, her eyes were sunken and closed, respiration was labored, and she was extremely weak.

A fecal specimen was mixed with a saturated solution of sodium nitrate, and many embryonated ova, averaging 30 microns x 45 microns, were recovered.

A presumptive diagnosis of pulmonary aspergillosis was made and the falcon was treated with 0.75 mg of amphotericin-B** intravenously and 8 ml of fluid*** intraperitoneally. Because of the hawk's respiratory difficulty, oxygen therapy was initiated. She died the following afternoon.

The day before the female died, the male was presented for examination. He had stopped eating and was losing weight rapidly. The lesion was still present on the tongue, and the owner believed the bird was reluctant to eat because of this lesion. On physical examination the male was alert, thin, weak, dehydrated, and respirations were deeper than normal. The owner force-fed him nearly a full crop of pigeon breast muscle. The next day about half of the meat had been regurgitated, and respirations were labored.

A fecal examination was negative for ova or parasites. Amphotericin-B (0.5 mg) was administered intravenously, and oxygen therapy was initiated. The following day he was in severe respiratory distress. Amphotericin-B (0.5 mg) was again administered intravenously. The bird died the following day.

Necropsy Findings

When necropsied, the female was emaciated with no subcutaneous or visceral fat. Several small thread-like nematodes, later identified as *Hartertia spp.*, were removed from the proventriculus. Multiple yellow to yellow-gray nodules were seen in the lung parenchyma, in the air sacs, and adhered to the visceral and parietal pleura of the thorax. These varied in size from less than 1 mm to about

*Emtryl; Salsbury Laboratories, Charles City, Iowa.
**Fungizone Intravenous; E. R. Squibb & Sons, Incorporated, New York, New York.
***Aminoplex Injection; A. J. Buck & Son, Incorporated, Baltimore, Maryland.

4 mm in diameter. The interior of several of the larger nodules contained a dark gray, finely matted material. Other organs were normal.

No acid-fast organisms were seen in a direct smear from the cut surface of a lung lesion. Inoculations were made on Sabouraud's agar; one nodule was cut and placed directly on the agar, a swab from another lesion was streaked, and a third nodule was ground with saline and sand and streaked on the agar. All plates had fungal colonies growing within 48 hours.

The fungus grew rapidly with a velvety, flat surface. Its center surface was gray-blue and there was no reverse pigment. When a portion of the growth was stained with lactophenol cotton blue and examined microscopically, branching septate hyphae were seen. Conidiophores were expanded terminally into vesicles that were covered with small sterigmata. These characteristics identified the fungus as *Aspergillus spp.*, likely *A. fumigatus* (*Laboratory Procedures in Clinical Mycology*, Department of the Army Manual 8-227-8, Washington, D.C. 1964).

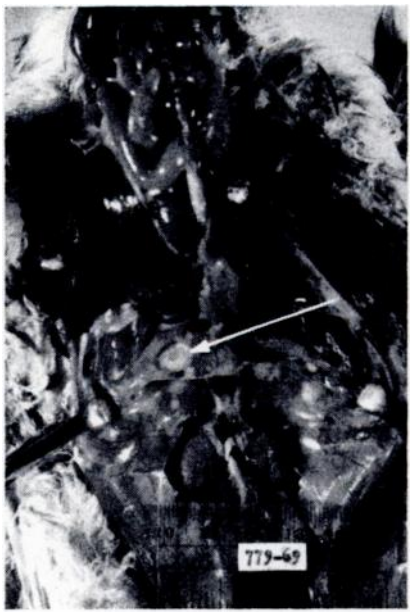

FIGURE 2. — Aspergillus spp. *lesions in the male's thorax (arrow).*

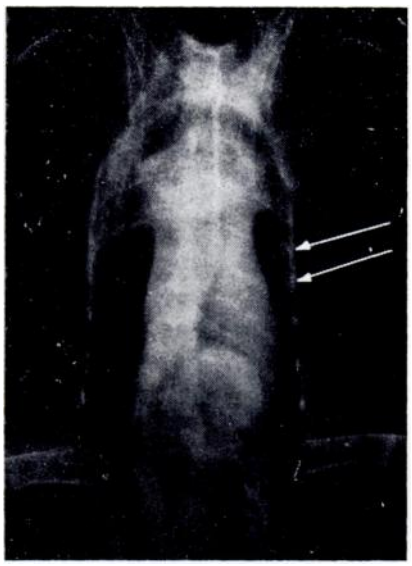

FIGURE 1 — *Post-mortem radiograph of the thorax and abdomen of the male. The arrows indicate two lesions.*

Microscopic examination of lung sections stained with hematoxylin and eosin corroborated the diagnosis of aspergillosis. Mycotic granulomas were scattered throughout the lungs and air sacs and the surfaces of various organs in contact with the air sacs. Masses of branching, septate fungal elements were readily seen when a silver staining solution was applied to the tissue sections.

In the male, before necropsy, we took a post-mortem radiograph of the thorax (Figure 1). Many round radiodensities were seen in the area of the lungs. When necropsied, the male had fewer but larger (1 mm to 7 mm in diameter) lesions in the lungs (Figure 2), and his air sacs were not affected. In addition, several pleural adhesions, a caseous mass at the bifurcation of the trachea, and fungal

bony involvement of several ribs and vertebral bodies were seen. A firm nodule, about 2 mm in diameter, was found on the surface of the proventriculus. The lesion encircling the tongue appeared to be healing. *Aspergillus spp.* was cultured from nodules in the lungs and on the proventriculus, and the lesions were histopathologically similar to the female's. A small feather was found embedded in one of the granulomas, deep in the lung parenchyma.

Discussion

Aspergillosis is probably the most common (Cooper, Vet Record 84: 454-457, 1969) and the most lethal (Beebe and Webster, *North American Falconry and Hunting Hawks,* World Press, Inc., Denver, Colorado, 1964) disease of captive birds of prey. Non-migratory or weakly-migratory species from northern latitudes (gyrfalcon and goshawk) seem especially susceptible. McIntyre (J. North Am. Falconers' Assoc., 7: 37, 1968) stated that prairie falcons usually are resistant to the infection, but he did report one severe case.

The disease in raptors is usually fatal, and symptoms (primarily a catastrophic weight loss, marked dehydration, and excessive thirst) are generally evident for no more than a week before death. Attempts at preventing aspergillosis have been made by falconers and at zoological gardens by fogging susceptible birds intermittently with a dilute solution of amphotericin-B (McIntyre, J. North Am. Falconers' Assoc., 7: 37, 1968).

We have been successful in treating suspected cases of aspergillosis in a juvenile female gyrfalcon and a juvenile male goshawk with intravenous amphotericin-B, 0.75 mg/kg, once daily (no symptoms of toxicity were noticed); a definitive diagnosis, however, was never established in these birds.

Ainsworth and Rewell (J. Comp. Path. 59: 213-224, 1949) presented a comprehensive review of aspergillosis in captive wild birds. They stated that it is primarily a disease of newly-caught birds, and doubted that mycosis often occurs in the wild. Two theories on the pathogenesis were presented; either the condition is "endemic" and latent in the wild and is "unmasked" by the unnatural conditions of captivity, or the disease is a hazard of captivity to which wild birds have a low resistance. They continued, "Radiography of the lungs of birds in an infected flock is no help in diagnosis, as so many other structures may intervene and obscure even the cervical air sacs."

Diagnosis of aspergillosis is difficult, especially considering the rapid course of the disease. Contrary to Ainsworth and Rewell, we believe radiography is a satisfactory diagnostic tool. In any X-ray, there are intervening structures; with practice, one learns to define normal radiographic anatomy, and can then interpret the abnormal. The lesions in Figure 1 are obvious.

Blood culture as a diagnostic aid would exclude bacteremia from the differential diagnosis. Fecal examination may help to eliminate gastrointestinal parasitism or verminous pneumonia as the primary disease.

Friend and Trainer (Bull. Wildlife Disease Assoc. 5(3): 271-275, 1969) isolated *Aspergillus fumigatus* from tracheal swabs taken from captive herring gulls in an infected colony; isolations were successful in four of five sick birds and one of five "normal" pen mates.

In a fulminating infection, thoracentesis, observing strict asepsis, may also be helpful. Material aspirated from the thorax could be cultured for bacteria, cultured on Sabouraud's agar for fungi, and a smear could be stained to demonstrate mycobacteria.

The feather found deep in the lung parenchyma of the male was well-encapsulated, and was not considered to be the primary or initiating lesion in this bird. The parasites in the female were also incidental.

The coincident course of the disease in these two falcons might indicate a common source of infection. Since they were raised separately, they were possibly exposed simultaneously to *Aspergillus* spores as nestlings. This hypothesis supports the opinion that the organism is a facultative pathogen that fulminates because of the stress of captivity.

In susceptible species of raptors, intermittent inhalation of aerosolized amphotericin-B may be indicated for prevention. A quick diagnosis is essential in active cases of aspergillosis, and can be based on history, physical examination, radiography, blood culture, fecal examination, tracheal culture, and perhaps thoracentesis. If diagnosed soon enough, intravenous amphotericin-B and supplemental fluid therapy may be effective.

Acknowledgements

We gratefully acknowledge the assistance of Mrs. MayBelle Chitwood and Major Robert A. Whitney, Jr. in the preparation of this manuscript.

F. PRESCOTT WARD

DAVID G. FAIRCHILD

JEANNE V. VUICICH

Veterinary Pathology Branch
Veterinary Medicine Department
Medical Research Laboratory
Edgewood Arsenal, Maryland 21010

October 1, 1969