adult red-tailed hawk represents one of the few such cases reported in the United States. The potential for spread of the virus to other hawks may occur particularly during the nesting season when an infected adult could conceivably pass the virus to a mate and nestlings by direct contact or fomites.

The foot lesions in this bird were severe and probably partially attributable to secondary infection with *Staphylococcus aureus* and fungi. The lesions grossly resembled "scaly-leg" caused by mites of the genus *Knemidocoptes,* however, none was found in our examination of the red-tailed hawk. Foot lesions as severe as those we observed were noted also by Pearson et al. (1975, op. cit.) and undoubtedly can result in a diminished ability to capture prey. The apparent systemic bacterial infection was also a major reason for debility in this case.

Little is known of the natural history of avian pox infection in birds of prey. In other birds it is generally considered mild and self-limiting; however, eye lesions resulting in impaired vision may lead to starvation (Karstad, 1971, *In* Infectious and Parasitic Diseases of Wild Birds, Davis et al. (eds.), The Iowa State Press, Ames, Iowa, pp. 34–41).

We appreciate the technical assistance of Kathy Lauhala for the electron microscopic efforts. This work was supported by Department of Energy under contract number DE-AC06 76RLO 1830.

Journal of Wildlife Diseases, 21(3), 1985, pp. 301–305

Mycotic Pneumonia and Meningoencephalitis due to *Aspergillus terreus* in a Neonatal Snow Leopard (*Panthera uncia*)

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On 14 May 1983, two female snow leopard (*Panthera uncia*) cubs were born in the Kansas City Zoological Gardens to a primiparous female. The female showed little interest in the cubs, one of which had a body temperature of 30 C, so they were removed for hand-rearing. On 15 May, one cub was less active, and did not nurse as well as its littermate. Parenteral antibiotics were started (Ampicillin—11 mg/kg BID, Kanamycin—5.5 mg/kg BID). By 16 May, the cub had lost 20 g, and although antibiotics were continued, the cub continued to deteriorate. Blood was noted in the stool, and a bloody nasal discharge was seen on 17 May. The cub was more depressed on 18 May, and had increased difficulty in breathing, due to the bloody nasal discharge. A blood sample was obtained, and revealed a packed cell volume of 42, total protein of 5.4 g/dl, and a white blood count of 5,200. A differential of the white blood cells showed a prominent immature neutrophil component with both bands and progranulocytes present. Small radiodense lesions were visible in a radiograph of the lungs. On the afternoon of 18 May, the cub became cyanotic and was placed in an oxygen chamber. Samples of the stool, nasal discharge, and blood were taken for culture. Atropine (0.5 mg/kg)

Received for publication 2 November 1984.

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and furosemide (4 mg/kg) were given parenterally. Pooled feline serum was given intravenously (5 ml) and subcutaneously (15 ml), and 5% lactated Ringers solution (20 ml) was given subcutaneously. The cub's condition continued to deteriorate, and it was euthanized on the evening of 18 May.

At necropsy, the surface of both lungs was covered with cheesy, yellowish areas. Clotted blood filled the nasal cavity and turbinates, and extended from the nasal cavity to the base of the brain. The small intestine contained focal areas of hemorrhage.

Microscopic changes were found in the nasal cavity, lungs, brain, and thymus. The nasal cavity was filled with clotted blood and the ciliated pseudostratified columnar epithelium lining the cavity was ulcerated. The lamina propria, beneath ulcerated areas, contained extensive hemorrhage and numerous thrombosed veins and arteries. Adjacent mucosa with intact surface epithelium was edematous and diffusely infiltrated by small numbers of macrophages and neutrophils. Septate hyphae were present on ulcerated surfaces, in the lamina propria, and within vascular thrombi.

The lungs had multiple, localized areas of inflammation in the parenchyma and diffuse exudation on pleural surfaces. The majority of parenchymal lesions were centered around the bronchial tree, but others were randomly dispersed in pulmonary alveolar tissue (Fig. 1). Lumina of affected bronchioles and the surrounding alveoli were partially filled by fibrin, neutrophils and macrophages in approximately equal numbers, and erythrocytes. Many respiratory epithelial cells on the surface of the bronchiolar mucosa were necrotic. Pulmonary and bronchial arteries in peribronchiolar adventitia were occluded by cellular thrombi (Fig. 2). White blood cells in the thrombi were composed primarily of macrophages with occasional neutrophils. Multiple random lesions located in alveolar parenchyma were characterized by foci of granulomatous inflammation composed almost exclusively of macrophages. Pleural surfaces were covered by a thick layer of degenerating neutrophils and macrophages enmeshed in fibrin. Subpleural fascia was edematous and contained numerous blood-filled capillaries and dilated lymphatic vessels. Numerous branching hyphae were observed in the lumina of bronchioles, arterial thrombi, and parenchymal granulomas (Fig. 3). The branching pattern was dichotomous and hyphae were septate with an outer diameter of up to 5 μm.

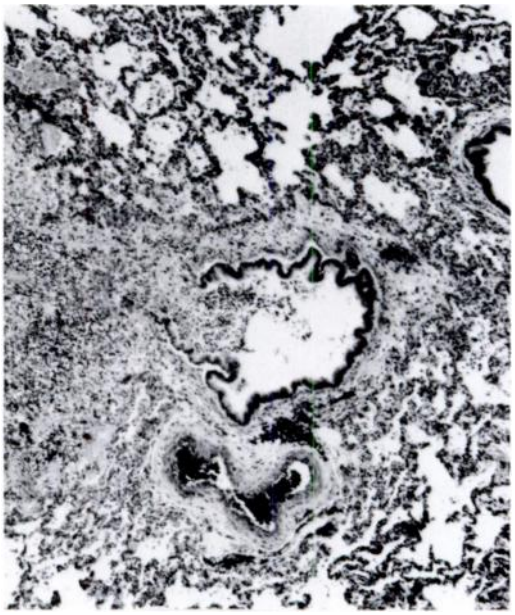

FIGURE 1. Section of lung with a parenchymal lesion involving a bronchiole and extending out into the parenchyma. The lumen of the bronchiole is filled with fibrin, neutrophils, macrophages, and erythrocytes. H&E, ×40.

Lesions were present in both the leptomeninges and the underlying cerebral cortex. Widespread hemorrhage permeated the subarachnoid cavity. Erythrocytes were intermingled with macrophages whose cytoplasm contained either phagocytosed red blood cells or hemosiderin granules. Arteries in the arachnoidal space were occluded by cellular thrombi and

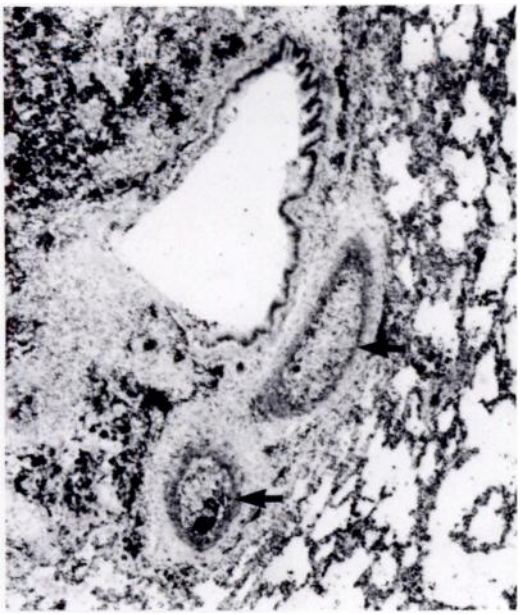

FIGURE 2. Section of lung with cellular thrombi occluding bronchial arteries (arrows). H&E, ×40.

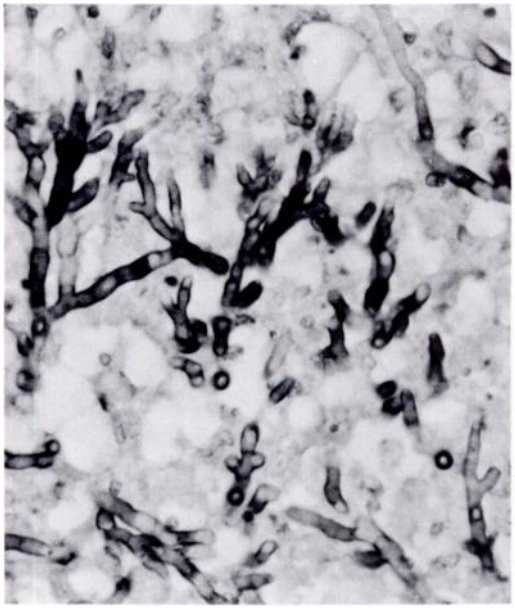

FIGURE 3. Bronchial lumen containing numerous septate, branching hyphae. Gomori's methenamine silver, ×400.

fungal hyphae similar to those described in the lung. The cerebral cortex had multiple areas of perivascular hemorrhage and perivascular pyogranulomatous inflammation. Numerous clear vacuolar spaces consistent with edema were observed around neurons, cortical vasculature, and within the neuropile. Branching septate hyphae pervaded the walls of inflamed blood vessels and the surrounding gray matter of the cerebrum.

The thymus showed mild to moderate thymocyte depletion characterized by a loose arrangement of lymphocytes embedded in the supporting meshwork of epithelial reticular cells.

Nasal septum scrapings and portions of lung were placed on a glass slide in P-51 stain (10% KOH and Parker 51 ink), heated briefly, and crushed with a coverslip. After 48 hr, numerous collections of blue-stained dichotomously branching hyphae, 4–5 μm wide, with parallel walls, were seen in the slides from both tissues.

Portions of infected tissue were smeared on plates of brain–heart infusion agar enriched with 5% defibrinated bovine blood and onto plates of potato dextrose agar. The plates were incubated at 27 C, and after 1 wk brownish sporulating fungal colonies were observed in the lung and the nasal cavity cultures. Spores were picked from a representative colony and used to prepare a single-cell isolate of the organism, which was subsequently transferred to Czapek's agar media for identification. The organism was identified as *Aspergillus terreus* (Raper and Fennell, 1965, The Genus *Aspergillus*, William and Wilkins Company, Baltimore, Maryland, 693 pp.) with cinnamon colored, smooth-walled spores approximately 2 μm in diameter, borne in chains in compact columns from phialides on metulae borne on a hemispherical vesicle at the end of a smooth-walled, colorless conidiophore. Lateral pedunculated conidia (Fig. 4), most of which were about 6 μm in size, were found in wet mount preparations. They were usually solitary, but occasionally occurred in

groups of two or three. This was further support for the identity of the organism, as *A. terreus* is a member of a group of Aspergilli that form lateral conidia.

Escherichia coli and *Klebsiella pneumoniae* were isolated from the feces. *Pseudomonas fluorescens* was isolated from the nasal discharge and from the lungs at necropsy.

Disseminated aspergillosis leading to central nervous system involvement is most common in immunocompromised hosts (Beal et al., 1982, Neurology 32: 473–479). Berkow et al. (1983, J. Pediatr. 103: 49–53) reviewed the literature on paranasal invasive aspergillosis in children with malignancy, and found an overwhelming percentage had a severe granulocytopenia, and had been treated with broad spectrum antibiotics for several weeks prior to the onset of invasive aspergillosis. Also, Tracy et al. (1983, Am. J. Clin. Pathol. 80: 728–733) demonstrated chemotherapy-induced leukopenia prior to *A. terreus* infection. A recent case of disseminated *A. terreus* infection in a dog was reported by Mullaney et al. (1983, J. Am. Vet. Med. Assoc. 182: 516–518). Corticosteroids had been administered to the animal 3 mo and 5 days before the onset of clinical signs and possibly contributed to the severity of the disease. Severely depressed lymphocyte responsiveness to phytomitogens as evaluated by in vitro lymphocyte blastogenesis tests was documented in three cases of canine aspergillosis (Barrett et al., 1977, J. Am. Anim. Hosp. Assoc. 13: 328–334).

Cell-mediated mechanisms are involved with clearing mycotic infections, and the status of these mechanisms in neonatal felines is not well defined. Impaired function of macrophages and neutrophils is seen in chronic granulomatous disease in humans and invasive aspergillosis is often a complication of that disease (Cohen et al., 1981, Am. J. Med. 71: 59–66). Schaffner et al. (1983, J. Clin. Invest. 69: 617–631) recently demonstrated that either functional neutrophils or macrophages were sufficient to prevent experimental disseminated aspergillosis in a mouse model. Impairment of both systems was necessary for the disease to be expressed. Macrophages appeared to prevent germination and eradicate spores, while neutrophils killed hyphae both in vitro and in vivo in mice. The paucity of lymphocytes in the thymus of the snow leopard cub discussed in this report may indicate a cellular immunodeficiency which predisposed to fungal infection.

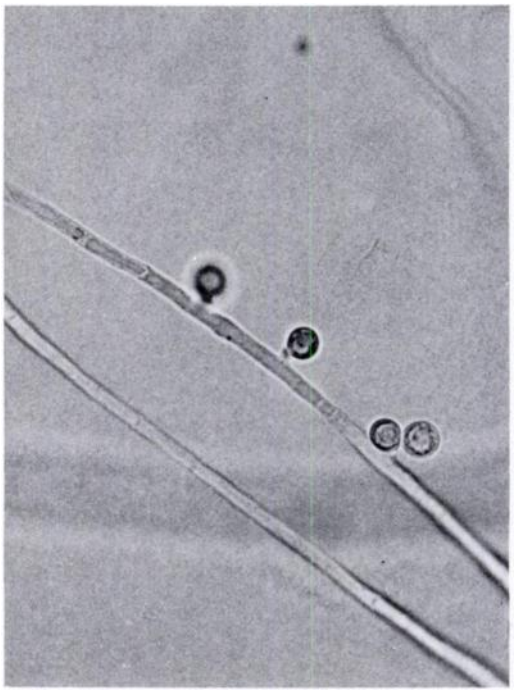

FIGURE 4. Lateral, pedunculated conidia of *A. terreus*. Pohlstoffe, ×400.

Antibody appears to have limited importance in protecting against fungal disease. Hypogammaglobulinemia has been documented in snow leopards (Worley, 1982, Int. Ped. Book of Snow Leopards, pp. 129–130), and low levels of gamma globulins have been detected in germ-free domestic kittens at birth (Hiraga et al., 1981, Lab. An. Sci. 31: 391–396), and conventional newborn domestic kittens also

have low levels of total protein and gammaglobulins (Okoshi et al., 1968, Jap. J. Vet. Sci. 29: 337–345). In the cat, most immunoglobulins are transferred via colostrum, rather than the placenta (Okoshi et al., 1968, op. cit.; Schultz et al., 1974, Infect. Immun. 9: 391–393). Probably this cub did not receive adequate colostrum at birth; but whether this inhibited its ability to combat infection is unknown.

Aspergillosis in cats is primarily a bronchopulmonary disease (Bright, 1981, *In* Pathophysiology in Small Animal Surgery, M. J. Bojrob (ed.), Lea and Febiger, Philadelphia, Pennsylvania, pp. 335–349) and seldom involves the brain.

The portal of entry in this case for the central nervous system was probably via the nasal cavity, with direct extension into the brain. The bloody nasal discharge occurred before other respiratory signs, and the finding of a blood clot extending to the base of the brain from the nasal cavity reinforces this hypothesis. Pulmonary infection was probably simultaneous with the nasal infection, and therefore hematogenous spread from the lungs to the central nervous system cannot be dismissed. Rapid hematogenic dissemination does occur, at least in turkey poults exposed to aerosols of *A. fumigatus* spores (Richard and Thurston, 1983, Avian Dis. 27: 1025–1033). In humans with direct spread from paranasal sinuses to the CNS, most had signs of sinusitis before meningeal involvement was noted (Bhalla et al., 1980, Acta Neurochir. 55: 135–139; Mohandas et al., 1978, J. Neurol. Sci. 38: 229–233). The severe meningitis seen in this case points to a "break" in the normal CNS barrier (Rippon et al., 1974, Sabouraudia 12: 157–161), rather than just seeding by septic emboli, although both processes may have been involved.

The source of the infection in this case was unknown. The cub's littermate was unaffected. Whether this unusual infection was due to an overwhelming insult to an immature immune system, or to an immune deficiency, is also unknown. The documentation of hypogammaglobulinemia in snow leopards suggests that some may have an impaired immune function. However, even a normal cub might be susceptible to opportunistic mycotic infection after being temperature stressed, colostrum deprived, and treated with broad spectrum antibiotics. Nevertheless, the possibility of multiple immune deficiencies in this endangered species should not be overlooked, and warrants investigation.

Journal of Wildlife Diseases, 21(3), 1985, pp. 305–309

Coccidioidomycosis (*Coccidioides immitis*) in the Collared Peccary (*Tayassu tajacu*: Tayassuidae) in Texas

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Coccidioidomycosis is primarily a respiratory disease caused by the fungus *Coccidioides immitis*. The fungus thrives in soil (especially rodent burrows) and produces arthrospores which usually are inhaled by mammals, causing a primary lung infection. The disease has been reported in a variety of free-ranging and captive wild mammals and domestic

Received for publication 30 November 1984.