Pulmonary Cryptococcosis in a Striped Dolphin (Stenella coeruleoalba)

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the Centers for Disease Control (CDC) was unable to confirm the morphological identification of *H. capsulatum* var. *capsulatum*. Additional immunofluorescence tests on replicate sections indicated that the fungus in this case was not *Cryptococcus neoformans*, *Blastomyces dermatitidis*, *Sporothrix schenckii*, nor a *Candida* sp. of medical importance (including *Torulopsis glabrata*).

Yeast forms morphologically characteristic of *H. capsulatum* var. *capsulatum* which would not stain with the specific FA conjugate for this fungus have been encountered rarely at CDC. It is probable that these yeast forms represent a unique serotype. Tissues were not available for culture or further characterization. Classification must be presumed based on the morphology observed using the GMS staining procedure.

Histoplasma capsulatum was isolated from five of six spotted skunks (*Spilogale putorius* L.) in Georgia (Emmons et al., 1949, Public Health Rep. 64: 1423–1430); the authors speculated that there was a high prevalence of histoplasmosis in skunks. A review (Sanger, 1981, op. cit.) noted other reports of histoplasma in skunks (including *M. mephitis*), but clinical cases were not reported. The present case represents a new host record of clinical histoplasmosis.

The gross and microscopic appearances of lung lesions in our case were similar to those described in progressive canine histoplasmosis (Farrell and Cole, 1968, Am. J. Pathol. 53: 425–434) except there was no fibroplasia. Fibroplasia was not a feature in lung changes reported in experimentally infected gnotobiotic dogs at 14 and 18 days post inoculation (DelFavero and Farrell, 1966, Am. J. Vet. Res. 27: 60–66).

Histoplasmosis is endemic in the region where this skunk was trapped. It may have been infected at the time of capture, and clinical disease appeared with the stress of captivity. The gross appearance of lesions were tumor-like and microscopic features superficially resembled a histiocytic lymphoma. Special stains were required to confirm a mycotic agent. This lends support to the contention by Smith et al. (1972, op. cit.) that many cases of histoplasmosis may be unrecognized.

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Pulmonary Cryptococcosis in a Striped Dolphin (*Stenella coeruleoalba*)

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The striped dolphin is widely distributed across the tropical and temperate waters of the Atlantic, Pacific and Indian Oceans. It is a gregarious species and large schools of up to 3,000 have been reported. The species is not often seen in coastal waters and appears to prefer a deeper offshore habitat (Watson, 1981, Sea Guide to Whales of the World, Hutchinson, London, England, pp. 264–265). This report concerns a 1.9-m immature striped dol-

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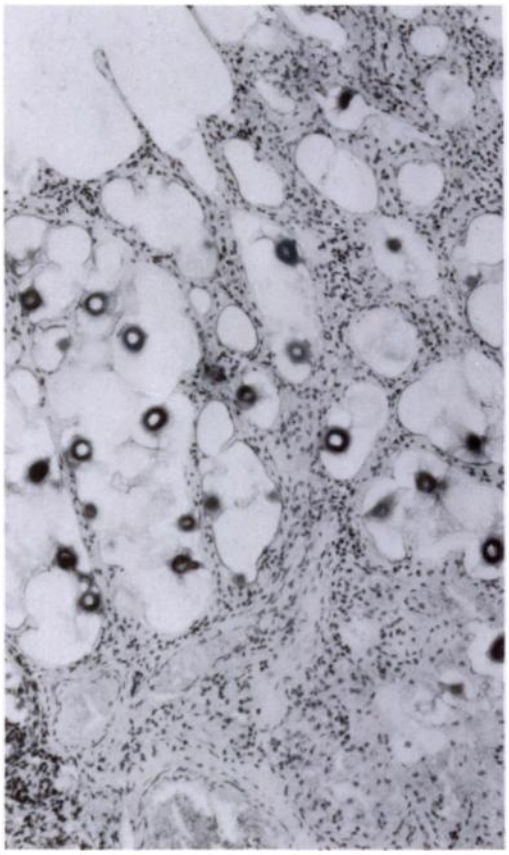

FIGURE 1. Granulomatous reaction in the interstitial tissue of the lung of a striped dolphin with cryptococcal bodies in the adjacent alveoli. H&E, ×211.

phin weighing 77 kg which was found dead in shallow water 5 m offshore at Geraldton, Western Australia (latitude 28°45'S). The carcass was fresh. There was no evidence of any injury apart from some mild abrasions on the ventral abdomen. From discussion with local fishermen, it appeared likely that the animal had died less than 12 hr previously.

At postmortem examination the blubber layer was of normal color and consistency, though somewhat reduced in thickness. The contents of the abdominal cavity appeared normal grossly but the gastrointestinal tract was relatively empty suggesting that the animal had not fed recently. The first chamber of the stomach contained 18 cephalopod beaks as well as a moderate number of nematodes (*Anisakis simplex*). There was no obvious evidence of parasites in other areas of the gut. In the thorax a firm whitish sub-pleural mass, 10 × 5 × 5 cm, was present on the dorso-medial aspect of the body of the right lung. The lesion involved approximately 50% of the body with the remainder appearing firm and congested. The left lung also appeared firm and congested and contained a few discrete firm white lesions of approximately 1 mm^3 scattered throughout the lung tissue. The mediastinal lymph node was enlarged. All other thoracic tissues were normal grossly. The brain was not examined. Tissues selected for microscopic examination were fixed in 10% buffered formalin. No bacteriological examination was undertaken.

Histopathological assessment of tissues confirmed the presence of cryptococcal organisms in the lung, mediastinal lymph gland and gastric mucosa. Examination of the lung revealed generalized interstitial congestion with proteinaceous fluid in many alveoli. Areas of granulomatous thickening were scattered throughout the interstitium (Fig. 1). These were characterized by loose fibrous tissue containing epitheloid cells, lymphocytes and plasma cells. Typical cryptococcal bodies, which stained well by the periodic acid–Schiff technique, were present in the middle of the reactive tissue and were usually numerous in the adjacent alveolar spaces. Giant cells were uncommon and there was no evidence of caseation as reported by Migaki (1978, Lab. Anim. Sci. 28: 603–606).

In the mediastinal lymph nodes (Fig. 2) large numbers of cryptococcal organisms were present in the lymph sinuses giving much of the tissue an edematous lacy appearance. Focal granulomatous reactions similar to that in the lung were observed

and giant cells were numerous, particularly in tissue adjacent to the trabeculae.

In stomach sections a small focus of cryptococci was present at the junction of the basal and middle third of the gastric mucosa. The epithelium in this area had disappeared leaving the cryptococci enmeshed in a reticular framework. There was no cellular reaction of any significance. The lesion appeared to be inert and was not associated with any other pathogenic process.

Infection with *Cryptococcus neoformans* usually results from the inhalation of the yeast. Primary foci in the respiratory organs are often the source of systemic dissemination which may involve central nervous system, bony structures, skin or other organs (Merchant and Packer, 1967, *In* Veterinary Bacteriology and Virology, 7th Ed., Iowa State Univ. Press, Ames, Iowa, pp. 549–566). In this case infection in the mediastinal lymph nodes and gastric mucosa as well as the lungs indicated that the infection was well established.

The effect of pulmonary infection on the clinical state of the dolphin prior to death is unknown however the extent of the lung involvement revealed by necropsy would suggest that there was substantial functional impairment. Cetaceans have had to evolve a highly efficient respiratory system in order to cope with a totally aquatic environment. Compromised respiratory function obviously has greater significance for a marine mammal and in the case of this dolphin it was probably the principal cause of death.

The possible source of infection in this dolphin is open to speculation. Unlike other fungi, *C. neoformans* exists as a yeast both in the environment and in tissue. While its presence has been detected in the terrestrial environment, it is not to our knowledge an inhabitant of the marine environment. The possibility of a bird being a carrier of infection would seem to provide a suitable explanation. *Cryptococcus neoformans* has been reported to be associated with pigeons and their habitat (Emmons, 1955, Am. J. Hyg. 62: 227–232), and, although unreported, could possibly be associated with seabird colonies. Dolphins may well contract the infection from such seabirds when both are simultaneously feeding on schools of pelagic fish.

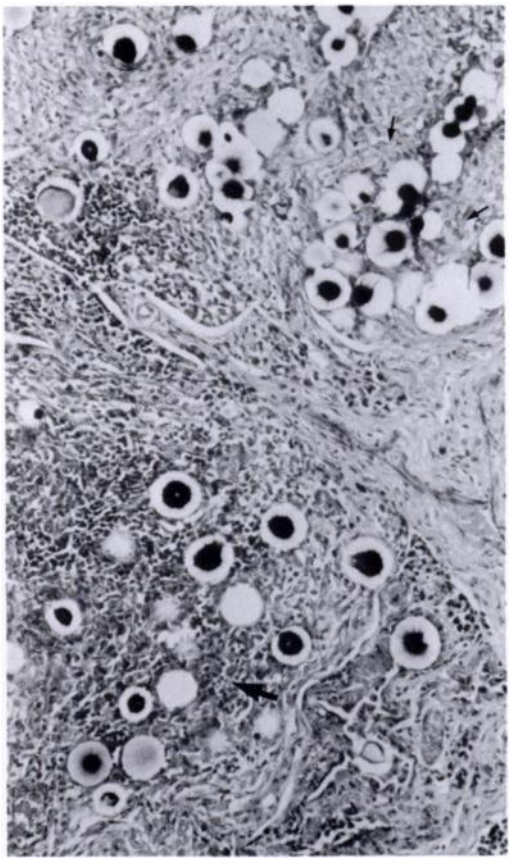

FIGURE 2. Cryptococci in a granulomatous formation (large arrow) and in lymph sinuses (small arrow) of a lymph node of a striped dolphin. PAS, ×211.

Cryptococcosis, the disease caused by *Cryptococcus neoformans*, has been reported widely in many domestic and wild terrestrial mammals including man. Reports of this infection in aquatic mam-

mals, however, are very rare. Migaki (1978, op. cit.) reported the incidental finding of *Cryptococcus* infection in a captive Atlantic bottlenose dolphin (*Tursiops truncatus*) that died of septicemia resulting from a perforated gastric ulcer.

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Fatal Systemic Toxoplasmosis in a Wild Turkey

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Toxoplasma gondii is a protozoan parasite belonging to the phylum Apicomplexa, class Sporozoea, subclass Coccidia, order Eucoccidiida and suborder Eimeriina (Levine et al., 1980, J. Protozool. 27: 37–58). Cats and other Felidae are the only definitive hosts for this coccidian parasite, but a wide range of vertebrates including birds and mammals can act as intermediate hosts. The life cycle of this parasite has been reviewed by several authors (Dreesen and Lubroth, 1983, Compend. Cont. Ed. 5: 456–460; Greene and Prestwood, 1984, *In* Clinical Microbiology and Infectious Diseases of the Dog and Cat, W. B. Saunders, Philadelphia, Pennsylvania, pp. 826–840).

Infection by *T. gondii* most often is inapparent clinically with the formation of bradyzoite-containing tissue cysts which may persist for the life of the host. Occasionally, clinical toxoplasmosis develops during the primary infection by *T. gondii* or following relapse subsequent to immunosuppression or for unknown reasons (Dreesen and Lubroth, 1983, op. cit.). We believe this is the first report of fatal toxoplasmosis in a free-ranging wild turkey (*Meleagris gallopavo*).

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In December 1983, a free-ranging female wild turkey, weighing 2.72 kg, was found dead on Blue Ridge Wildlife Management Area, Union County, Georgia. The carcass was frozen and submitted subsequently for necropsy.

At necropsy, massive splenomegaly was the most striking feature. Severe pneumonia, characterized by focal to confluent gray areas of consolidation, was present. Multiple 1- to 2-mm-diameter erosions covered by pseudomembranous exudate studded the cecal and colonic mucosa.

Representative samples of lung, spleen, liver, kidney, adrenal, esophagus, gizzard, proventriculus, brain, colon, and ovary were fixed in 10% buffered formalin, embedded in paraffin, sectioned at 7 μm, and stained with hematoxylin and eosin, Giemsa, Gram's and periodic acid–Schiff. For electron microscopy, pieces of formalin-fixed lung were embedded in Spurr resin, thin sectioned, and stained with uranyl acetate and lead citrate.

Histologic examination of the spleen revealed diffuse necrosis with massive fibrinous exudation and scattered accumulations of macrophages. In the lung, there was nonsuppurative interstitial pneumonia involving many respiratory lobules. Walls of air capillaries were thickened by lymphocyte and macrophage infiltration