Lobo's Disease in an Atlantic Bottle-Nosed Dolphin

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SUMMARY

Lobo's disease was diagnosed histologically in the skin of the tail stock and flukes of a feral Atlantic bottle-nosed dolphin (Tursiops truncatus) found in a bay on the west coast of Florida. The cutaneous lesions appeared as extensive white crusts. There were large, discrete, histiocytic granulomas in the dermis, resulting in severe acanthosis. The causative mycotic organism, Loboa loboi, was readily demonstrated in the granulomas with Gridley fungus stain or with periodic acid-Schiff techniques.

LOBO'S DISEASE, known also as keloidal blastomycosis, is a chronic granulomatous infection of the skin caused by the fungus Loboa loboi. The disease has been reported only in man and only in Brazil, Surinam, and Costa Rica.3,6,7 The lesions are localized in the skin, without visceral involvement. Though cutaneous lesions may occur anywhere, the principal sites are the feet, legs, hands, arms, buttock, and face. Inasmuch as lesions are seldom found on the back, spread is thought to occur by traumatic auto-infection. The characteristic gross lesions are slowly growing smooth nodules that may become verrucose, crusty, whitish plaques after many years.

This report deals with a case of Lobo's disease in a feral dolphin.

Case History

A female Atlantic bottle-nosed dolphin of unknown age and weight, measuring 2.35 meters from tip of rostrum to caudal notch, was seen by one of us (B.I.) in the Gulf Intracoastal Waterway near Sarasota, Fla. The dolphin was distinctive because of its white, crusty tail stock, which was readily visible when the animal sounded. Thirty minutes later she appeared to be in great distress, coming out of the water, spinning on her tail, and otherwise thrashing. At one time she swam rapidly with her head above water for approximately 30 m. She was restrained, but within minutes she died.

Gross Findings.—The dolphin appeared slightly underweight. Teeth were worn, and several were missing. Visceral organs were normal. Lesions were confined to the skin and consisted of extensive white crusts on the tail flukes and part of the stock extending forward from the tail (Fig. 1A). Most of the right side of the fluke and the area around the caudal peduncle extending

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10 cm. anteriorly on the tail stock were affected. The right half of the fluke appeared withered and was bent ventrally (Fig. 1B). Incision of the affected skin showed that the lesion extended about 1 cm. below the surface.

Microscopic Findings.—The epidermis was greatly thickened as a result of acanthosis (Fig. 2A and B). Large, discrete histiocytic granulomas were localized in the papillary layer of the dermis. The granulomas were cellular and consisted principally of histiocytes, giant cells, and fungal organisms. There were occasional foci of polymorphonuclear leukocytes. Stroma was minimal. The ratio of histiocytes to giant cells varied from granuloma to granuloma, from almost equal portions to a predominance of either. Giant cells were of the foreign body type. Nearly all the cells contained one or more large, round or lemon-shaped, faintly visible fungi having thick, refractile walls. One or more small, round faintly basophilic bodies were found within the centers of some of these fungi (Fig. 3). Small numbers of fungi contained a highly birefringent, slender rod, the nature of which was undetermined. These rods were more numerous or ap-

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Fig. 1—Top view (A) of the tail fluke of T. truncatus, showing white crusty lesions (arrow) on the skin. Lateral view (B), showing the bent tail fluke (arrow).

Fig. 2—Comparison of the normal skin (A) of T. truncatus with the affected skin (B) at the same magnification. Notice the acanthosis and irregular drowngrowths of the epidermis and the granuloma (1) in the papillary layers of the dermis in (B). H&E stain; x 13.
Fig. 3—A granuloma with numerous round, thick-walled, pale-staining fungi (Loboa loboi) in histiocytes and giant cells. Budding forms (1) and faintly staining centrally located basophilic bodies (2) are visible. H&E stain; x 600.

parent in the sections prepared by the periodic acid-Schiff (PAS) or Gridley technique. The cell wall and centrally located bodies of the fungus were strongly PAS-positive (Fig. 4) and stained well with Gridley's fungus stain (Fig. 5). The fungi varied from 5 to 10 μ in diameter, and the central bodies varied from 1 to 2 μ in diameter. They were arranged in branching chains of varying lengths, with the cells connected by short, thick tubes. The wall of larger, more centrally located fungi had a distinct rough and spiny surface (Fig. 6). The morphologic characteristics of this fungus were typical of L. loboi.

Large, irregular accumulations of macrophages, with abundant foamy cytoplasm, were located adjacent to the granulomas and deeper in the connective tissue of the dermis. The macrophages contained large amounts of PAS-positive granular material, which was probably debris from dead fungi (Fig. 7).

Fig. 4—Loboa loboi (1) are easily demonstrated in giant cells (2). The cell walls and centrally located bodies of L. loboi are PAS-positive. PAS stain; x 530.

Fig. 5—Loboa loboi arranged in long, branching chains. Gridley fungus stain; x 900.

Fig. 6—High magnification of Loboa loboi, showing the rough and spiny surface. Gridley fungus stain; x 2,180.
Fig. 7—Phagocytosed PAS-positive debris (arrows) of Lobo a lobo i found in the dermis near the granulomas. PAS stain; x 425.

and occasional recognizable fragments of fungal organisms.

Discussion

This is the only known report of Lobo’s disease in a mammal other than man. Successful inoculation of the fungus into the foot pads of 2 golden hamsters, followed by the development of characteristic granulomas 8 months after inoculation has been reported. Although there have been numerous attempts to cultivate this fungus, it has not yet been grown successfully. It was not attempted in this case.

Grossly, the lesions described here correspond to long-standing lesions in man, as exemplified by the development of verrucose, crusty plaques. Microscopically, the lesions in the dolphin had all the characteristics of Lobo’s disease in man.

In identifying the etiologic agent, *L. loboi* must be differentiated from *Blastomyces dermatitidis*, which has a bud with broad base attachment, and *Blastomyces brasiliensis*, which has multiple peripheral buds, and *Cryptococcus neoformans*, which has a thick mucinous capsule.

The fungus in these slides appeared typical for *L. loboi*, but because of the lack of reported cases of Lobo’s disease in animals, confirmation of its identity was sought elsewhere. It has been reported that the fungus is so distinctly different from anything seen in other fungal infections that it can be diagnosed without difficulty. Certain characteristics considered diagnostically important were observed in the sections of our case:

1) Solid inflammatory infiltrations of the dermis composed of histiocytes and giant cells, with resolution of collagenous tissue fibers; the foci of polymorphonuclear leukocytes are similar to those seen in ulcerating lesions of man.
2) Clusters of histiocytes, resembling the cells seen in granular cell myoblastoma, in peripheral parts of the inflammatory infiltration.
3) Higher than usual number of yeastlike fungal cells, most of them within giant cells and histiocytes.
4) Predominant arrangement of fungal cells in branching chains.
5) Lack of stainable contents in most fungal cells.
6) Birefringent inclusions in some fungal cells.
7) Spasmodic occurrence of fungal cells, with cytoplasmic mass and several small nuclei.
8) Single budding at the end of a chain and sometimes lateral budding, producing branched chains.
9) Low frequency of budding cells (about 1%).

It cannot be determined how a dolphin in Florida could contract a disease previously found only in human beings in the interior forests of certain areas of Central and South America. It is conceivable that the dolphin could have migrated to Florida with the disease. One investigator limits the southern

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distribution of T. truncatus to Mexico and Barbados, but another investigator has a skull of this species from Venezuelan waters. Evidence has not yet been presented, however, to indicate that T. truncatus does migrate over long distances as great as from South America to Florida. To the contrary, there is evidence for a home range in which distinctly marked dolphins return to the same area over extended periods. Further, one of us (B.I.) is conducting a dolphin-tagging study and has preliminary evidence to indicate that the same school of T. truncatus remained continuously in the study area for at least 8 months.

It would seem doubtful, therefore, that a single dolphin would carry the disease to Florida from Central or South America. Whether the disease could have spread slowly northward by contact between infected dolphins, or whether the disease might be enzootic and yet undetected or undiagnosed in dolphins, cannot yet be established.

The possibility must be considered that L. loboi is a marine fungus and that human cases of this mycosis could be incurred by exposure to tidal waters.

References