Lobomycosis in Inshore and Estuarine Dolphins

To the Editor: Lobomycosis is a chronic dermal infectious disease affecting humans and some species of dolphins but not, to date, freshwater dolphins. Because this disease is still considered rare despite the increasing number of reported cases in humans and cetaceans, clinical and epidemiologic information must be accurately reported to help clarify many of the unknown aspects of this disease.

We address this point because after carefully reading the excellent report by Elsayed et al. on the first human case of lobomycosis in Canada, we noticed that the authors describe the natural disease as occurring in humans and marine and freshwater dolphins only (*1*). However, this information is only partially correct because to date lobomycosis has not been described in freshwater dolphins. What is more worrisome is that this information is beginning to be referenced in other published articles (*2*). So far, lobomycosis has been confirmed in 2 species of inshore and estuarine Delphinidae: 1) the common bottlenose dolphin (*Tursiops truncatus*) from Brazil, the Atlantic coast of the United States, and Europe and 2) the Guiana dolphin (*Sotalia guianensis*) from the Surinam River estuary (*3*–*8*).

The fact that lobomycosis is endemic in humans in the Amazon basin could logically raise the suspicion that other animal species in this area may act as reservoirs or even be affected by the disease. However, the infection has never, to our knowledge, been reported in botos (*Inia geoffrensis*) or tucuxis (*Sotalia fluviatilis*) from the Amazon and Orinoco Rivers. Preliminary field studies, like the one carried out by da Silva et al., failed to demonstrate the disease in any of the 385 live-captured *I. geoffrensis* boto specimens from the Mamirauá Reserve in the central Amazon region of Brazil (*9*); similarly, our observational studies in the Venezuelan Orinoco River failed to detect the disease. On the other hand, despite the absence of indigenous cases of lobomycosis in humans reported in the United States, the disease is endemic in dolphins from the Indian River Lagoon in Florida (*7*), suggesting that no apparent epidemiologic link may exist between humans and cetaceans. Unfortunately, the etiologic agent of lobomycosis, *Lacazia loboi* (Figure), has not been cultured in vitro (*10*) despite exhaustive attempts, making its isolation from probable and suspected environmental sources impossible.

Dolphin-to-human transmission of lobomycosis has been reported only 1 time; the case-patient was an aquarium attendant who had had close physical contact with an affected dolphin (*5*). However, because the possibility of zoonotic transmission of this disease remains latent and because many pathologic and clinical aspects of the disease remain poorly understood, it is imperative to clarify these ecological concepts. Up-to-date molecular epidemiology studies to compare the strains affecting humans and dolphins and their possible phylogenetic relationship are needed.

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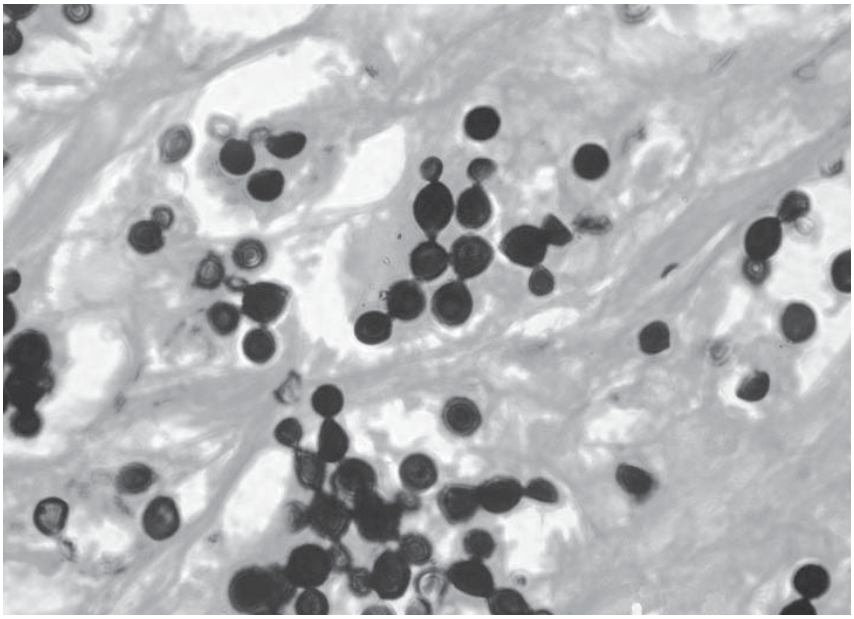

Figure. Grocott methamine silver–stained section from a skin biopsy specimen of a bottlenose dolphin (*Tursiops truncatus*) showing abundant *Lacazia loboi* yeast cells individually and in chains connected by thin tubular bridges. Magnification ×400.

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Variations in Leprosy Manifestations among HIV-Positive Patients, Manaus, Brazil

To the Editor: Contrary to early expectations, the co-occurrence of leprosy and HIV has not increased globally (*1*). However, most of the larger studies on the subject were conducted in the early to mid-1990s in African countries, and the research designs had limited power to describe the true effects of co-infection (*1*). Moreover, the introduction of highly active antiretroviral therapy (HAART), which has been used routinely in Brazil since 1996, altered the clinical evolution of HIV infection (*2*) and led to increasing reports of immune restoration inflammatory syndrome (IRIS) associated with leprosy (*3*,*4*). Although some researchers have argued that this association may not affect public health (*2*), its true importance remains to be clarified. Finally, leprosy has a wide range of clinical manifestations, which sometimes imposes a clinical challenge and may lead to misdiagnosis (*5*). Together, these factors may have helped mask the true scenario of leprosy and HIV co-infection, particularly in areas where these conditions are highly endemic. In this context, case reports from referral centers that reflect the broad clinical aspects of leprosy and HIV co-occurrence are important to increase clinicians' awareness of both diseases.

We report 3 HIV-positive/AIDS patients who showed different clinical manifestations of leprosy; their conditions were diagnosed before and after HAART initiation. All patients lived in Manaus, the capital of the state of Amazonas in Brazil, an area where both leprosy and HIV infection are endemic. The 3 patients represent a sample from our 11-year experience with 21 patients with leprosy and HIV co-infection.

Patient 1 was a 29-year-old woman whose HIV-1 infection was diagnosed in May 2002 at antenatal examination. Her CD4 cell count in 2002 was 513 cells/μL. In November 2007, she sought treatment at the Institute of Tropical Medicine of Amazonas with a 3-month history of a single erythematous plaque on her left arm, which was clinically diagnosed as borderline tuberculoid (BT) leprosy. The patient's sensitivity to pain was decreased. There was no nerve enlargement. Histopathologic examination confirmed the diagnosis, showing a granulomatous dermatitis with no acid-fast bacilli on Wade stain. At this time, her CD4 cell count was 342 cells/μL. HAART and multidrug therapy (MDT) for paucibacillary leprosy were initiated. The leprosy resolved, and the lesion disappeared within 2 months of therapy.

Patient 2 was a 22-year-old man who had neurocryptococcosis and HIV infection diagnosed in September 2007. At that time, he exhibited disseminated, infiltrated lesions on the trunk and upper and lower limbs. Borderline lepromatous (BL) leprosy was clinically diagnosed. Skin biopsy confirmed the diagnosis; the biopsy specimen showed a granulomatous dermatitis, foamy cells, and multiple acid-fast bacilli. His CD4 cell count was 6 cells/μL. HAART and MDT for multibacillary leprosy were prescribed. In February 2008, the patient was readmitted to the Institute of Tropical Medicine of Amazonas and died of nonspecified bacterial pneumonia and sepsis.

Patient 3 was a 23-year-old woman who had HIV-1 infection (CD4 cell count 435 cells/μL) diagnosed in November 2006 at antenatal examination. HAART was begun 3 months later. In August 2008, she sought treatment with a 3-month history of a single patch on the left leg with erythematous papules on its border (Figure). There was decreased pain sensitivity in the