Endemic Lepromatous Leprosy

SIR—Although it was quite interesting to read the report of lepromatous leprosy in a renal transplant recipient [1], I was surprised that Mushatt et al. were permitted to speculate that the patient might represent a case of contact with armadillos, since he apparently grew up and lived in an area where leprosy is well known to be endemic—New Orleans and Baton Rouge in south Louisiana. In contrast, northern Louisiana has not been associated with the transmission of endemic leprosy until recently [2]; there have been virtually no other cases in these parishes where lifelong residents—with no contact with patients with leprosy—developed leprosy. In northern Louisiana, the argument is that armadillos are the only obvious source of *Mycobacterium leprae*. In contrast, it has been known for >200 years that living in south Louisiana poses some risk for the acquisition of leprosy without travel and without contact with patients with diagnosed cases. The risk predates armadillos in Louisiana [2, 3].

In addition, the case report by Mushatt et al. nicely included HLAs (human leukocyte antigens) and showed once again that the risk of developing leprosy is associated with class II antigens. The analysis that we conducted, which included the six cases previously reported from northern Louisiana and a meta-analysis of data in the literature at that time, showed an association between lepromatous leprosy and HLA-DR2 and HLA-DQw1 [4]. Concerns about the pathogenesis, epidemiology, endemic transmission, and diagnosis of leprosy (including the acquisition of endemic disease) should continue to receive educational and research efforts, particularly in countries where it is endemic, such as the United States [5].

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References

to soil may have been the source of infection. We did mention that armadillos had been observed in the area where the patient did gardening, since armadillos do seem to play an as yet poorly understood role in the ecology of *Mycobacterium leprae*. Blake and colleagues [2] themselves acknowledged that “in effect, humans and armadillos may pass the disease back and forth.” We agree with the contention of Blake et al. [2] that “it is, however, equally possible that human beings and armadillos share an environmental niche with a third component that is infected with or harbors *M. leprae*.”

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Subacute Pneumococcal Endocarditis

Sir—We read with interest the excellent review on pneumococcal endocarditis by Aronin et al. [1]. In that article, Aronin et al. commented on the predisposing risk factors, clinical presentation, diagnosis, and management of this infrequent type of endocarditis, all of which were based on 197 adult cases reported in the English-language literature since 1966.

On the basis of the 69 cases for which the duration of illness before presentation was determined, the median time was 7 days (range, 1–150 days). Later, Aronin et al. commented on the existence of a subset of patients with subacute presentation.

Pneumococcal endocarditis is usually acute at the time of presentation, typically aggressive, and associated with high rates of morbidity and mortality, mostly because of the rapid destruction of the endothelial tissue of the affected valve (which is usually left-sided and previously undamaged). However, in 1986 Powderly et al. [2] and in 1992 Gelfand and Threlkeld [3] reported two cases each of subacute pneumococcal endocarditis. In 1994, we [4] reported the fifth case, which occurred in a 42-year-old man with a previous mitral valve prolapse. However, our case was not reviewed by Aronin et al. because it was recently reviewed [5], in addition to the four cases previously reported in the Spanish-language literature [4]. On closer examination of the 197 adult cases of pneumococcal endocarditis that we recently reviewed [5], in addition to the four cases previously reported [2, 3], we identified eight other patients who had evidence of an indolent course [6–12]. The median duration of symptoms before diagnosis for these 12 patients (6.1%) was 35 days (range, 10–150 days), and for the three patients who died, the median duration of symptoms before death was 92 days (range, 67–120 days). For these 12 patients, the male-to-female ratio was 1:1, and the mean age was 54 years. Underlying medi-

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Reply

Sir.—Infective endocarditis was previously classified as either acute or subacute on the basis of the natural progression of untreated disease. Patients with untreated acute infective endocarditis were defined as having a fulminant course leading to death within 6 weeks. By contrast, patients with untreated subacute infective endocarditis were defined as having a more indolent course resulting in death after this time. As this distinction is an arbitrary one, the terminology has been refined such that classification of infective endocarditis is now based on the etiologic agent responsible for disease.

Patients with untreated pneumococcal endocarditis typically present with Osler’s triad. First, they usually have pneumococcal pneumonia following which, after a brief apparent recovery, they develop fever, sepsis, cardiac failure, meningitis, and death within 30 days of the onset of pneumonia. An early review of nearly 600 cases of pneumococcal endocarditis [1], however, made reference to a subset of patients with a more subacute course.

Since this early review, subacute endocarditis due to *Streptococcus pneumoniae* has been described in only four patients in the English-language literature [2, 3], and one case has been reported in the Spanish-language literature [4]. On closer examination of the 197 adult cases of pneumococcal endocarditis that we recently reviewed [5], in addition to the four cases previously reported [2, 3], we identified eight other patients who had evidence of an indolent course [6–12]. The median duration of symptoms before diagnosis for these 12 patients (6.1%) was 35 days (range, 10–150 days), and for the three patients who died, the median duration of symptoms before death was 92 days (range, 67–120 days). For these 12 patients, the male-to-female ratio was 1:1, and the mean age was 54 years. Underlying medi-