Southern Extension of the Range of Human Babesiosis in the Eastern United States

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We sought evidence of babesiosis in three residents of New Jersey who were suspected of local acquisition of Babesia microti infection. We tested serial blood samples from these residents for B. microti antibodies and amplifiable DNA by using immunofluorescent antibody and PCR techniques. All three residents experienced symptoms suggestive of acute babesiosis. The sera of each of the patients reacted against babesial antigen at a titer fourfold or higher in sequentially collected blood samples. PCR-amplifiable DNA, characteristic of B. microti, was detected in their blood. These data suggest that human B. microti infections were acquired recently in New Jersey, extending the range of this piroplasmosis in the northeastern United States.

The distribution and frequency of human babesiosis due to Babesia microti in North America appear to be increasing. The health relevance of this zoonosis first became evident in 1969 in a resident of Nantucket Island, Mass. (17). Clusters of cases became evident there and on eastern Long Island, N.Y., during the mid-1970s (3). Human infections later appeared in the north central United States (in Wisconsin) and along the southern margins of Connecticut and Rhode Island (5, 6, 14). Evidence of infection in rodent reservoir hosts (white-footed mice [Peromyscus leucopus]) and in vector ticks (Ixodes dammini, which differs from the more southern Ixodes scapularis) confirms the discontinuous northern pattern of distribution of this zoonosis (11, 12). Interestingly, the coinfecting agent of Lyme disease is far more widely distributed. Sparse evidence of this zoonosis (11, 12). Interestingly, the coinfecting agent of Lyme disease confirms the discontinuous northern pattern of distribution of mini

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Although *B. microti* DNA was amplified from the sample, no piroplasms were identified in the Giemsa-stained thin blood smear, nor were IgM or IgG *B. microti* antibodies detected. Two months later, the patient continued to experience sweats and fatigue. Reciprocal *B. microti* immunofluorescence titers of 1:128 for IgM and 1:32 for IgG were detected. No piroplasms were discovered in thin blood smears, and no babesial DNA was amplifiable.

All of the patients whose experiences are documented here are residents of northern New Jersey. None had traveled outside the state during the previous year, received a blood transfusion, or had detectable antibodies against Lyme disease or human granulocytic ehrlichiosis.

All three of these New Jersey residents appear to have experienced infection by the agent of human babesiosis, *B. microti*, and all experienced symptoms suggestive of acute babesiosis. The sera of each of these patients reacted against babesial antigen at a titer fourfold or higher in sequentially collected blood samples. PCR-amplifiable DNA characteristic of *B. microti* was detected in their blood. The failure to observe piroplasms in Giemsa-stained blood films is considered conclusive, because these slides were prepared 2 weeks or more after the onset of illness. Our patients showed convincing evidence of babesial infection.

Although Lyme disease is a frequent health hazard near our patients’ homes in central New Jersey (2), no episode of human babesial infection has previously been reported from this region. The infection frequently is diagnosed on eastern Long Island and along the Connecticut and Rhode Island coasts some 200 km to the east of New Jersey. Intense infections have been noted even further to the east, on Nantucket Island in Massachusetts, and in north central Wisconsin nearly 2,000 km to the west. In Connecticut, about 11% of people suffering from Lyme disease also have experienced babesial infection (6).

Definitive proof of the perpetuation of this zoonotic pathogen in a region, of course, requires epizootiologic evidence. The vector tick must have established a stable cycle of transmission, and this appears to have occurred in northern New Jersey during the late 1970s (1). The first mainland infestation was reported in 1961, in coastal Rhode Island (4). The infecting agent of Lyme disease appeared in central Connecticut during the mid 1970s and in Westchester County, near New York City, several years later (13, 18). Ticks infected by *B. microti* have only recently been discovered in New Jersey (16). None of the study patients had traveled to a region where babesiosis is endemic during the year before their infection, and none had ever received a blood transfusion. The environmental conditions necessary for the transmission of *B. microti* apparently are present in Flemington, N.J., as well as the zoonotic circumstances necessary to produce a cluster of human infections. Physicians practicing in central New Jersey, therefore, should be aware that *B. microti* infection may threaten the health of their patients.

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