JOURNAL OF CLINICAL MICROBIOLOGY, June 1999, p. 2051–2052 Vol. 37, No. 6
0095-1137/99/$04.00+0

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

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Received 19 January 1999/Returned for modification 2 February 1999/Accepted 1 March 1999

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 babesial infection, however, has been detected in ticks recently swept from vegetation in northwestern New Jersey, where Lyme disease has been zoonotic for at least a decade (16). We now present evidence of three human *B. microti* infections that were acquired recently in this region.

Piroplasms were identified microscopically in Giemsa-stained films prepared from EDTA-anticoagulated blood (15). Babesial infection was diagnosed serologically by an indirect immunofluorescence assay for immunoglobulin M (IgM) and IgG antibodies as previously described (7, 8). One of the coinvestigators (J.A.), who was unaware of the clinical status of the study subjects, conducted the *B. microti* PCR amplifications as described previously (9, 10).

The first patient was a 37-year-old female who had suffered from severe headaches since early July 1998 and sought medical care on 24 August. She had experienced drenching night sweats and arthralgia throughout the previous week, and her oral temperature had ranged from 38.2 to 39°C. Her primary care physician had prescribed doxycycline (100 mg twice a day) because Lyme disease was suspected. The patient subsequently became afebrile, but other symptoms persisted including generalized fatigue, anorexia, myalgia, vertigo, inability to concentrate, difficulty with short-term memory, insomnia, and unilateral numbness of the extremities of the right side. She recalled no contact with ticks and no rash. She previously had been healthy and medication free. The results of her physical examination were normal. Serological tests performed at a private laboratory revealed reciprocal *B. microti* immunofluorescence titers of 1:80 for IgM and IgG. In order to confirm the diagnosis of babesiosis, blood samples were submitted to the University of Connecticut Health Center in Farmington, Conn., on 31 August. Reciprocal *B. microti* immunofluorescence titers were 1:512 for IgM and 1:256 for IgG. Babesial DNA was amplified by PCR. No piroplasms were identified on Giemsa-stained thin blood smears. Following the administration of clindamycin and quinine, the patient experienced immediate relief of her symptoms. A blood sample was obtained on 12 October. No babesial DNA was evident, and reciprocal immunofluorescence titers were 1:512 (IgM) and 1:1,024 (IgG).

The second patient was an 11-year-old male who sought medical care on 10 July for low-grade fever, severe frontal headache, arthralgia, and fatigue, 9 days after an engorged tick was removed from his scalp. The results of his physical examination were normal. His physician suspected Lyme disease and prescribed doxycycline (100 mg daily). Three weeks later the patient continued to complain of headache, arthralgia, and fatigue. A blood sample sent to a private medical laboratory revealed a reciprocal *B. microti* immunofluorescence titer of 1:80 for IgG. Doxycycline was discontinued, and clindamycin was prescribed (150 mg three times a day). The patient's symptoms were resolved several days later, and the clindamycin was continued for 2 weeks. A consultation was sought at the University of Connecticut Health Center 2 weeks after the doxycycline had been discontinued. *B. microti* DNA was amplified from blood, but no IgM and IgG antibodies were detectable. No piroplasms were identified on Giemsa-stained thin blood smears. A second blood sample was obtained 1 month later. *B. microti* DNA was no longer amplifiable, but a reciprocal immunofluorescence titer of 1:512 for IgG was detected.

The third patient was a 39-year-old female who sought medical attention in August 1998 for a 2-month history of intermittent night sweats and fatigue. She had experienced several deer tick bites during the previous several months. The results of her physical examination were normal. *B. microti* DNA was amplified in blood sent to a private laboratory, but no specific antibabesial therapy was administered. Additional blood samples were sent to the University of Connecticut Health Center.

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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 inconclusive, 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 epizootiological 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 coinfecting 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.

This work was supported by grants from the National Institutes of Health: AI 42402 (P.J.K. and A.S.) and AI 19693 (A.S.).

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