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# Babesiosis in California

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BABESIOSIS IS A TICK-BORNE ZOONOSIS caused by an intraerythrocytic parasite. The cycle of transmission of the protozoan *Babesia* species between the tick vector and vertebrate host is occasionally interrupted by humans, resulting in human infection. Babesiosis is most common in otherwise healthy persons, many of whom remain asymptomatic. Although patients who have had a splenectomy are not more susceptible to infection with a *Babesia* species, they are more likely to become symptomatic, probably due to suppressed immune function. Symptomatic babesiosis is characterized by persistent fever and chills, hemolytic anemia, myalgias, fatigue, and hepatosplenomegaly. *Babesia divergens* causes severe and often fatal disease and is found throughout Europe. *Babesia microti,* found most often in the northeastern United States, typically causes a mild flulike illness, but a broad range of severity exists, and *B microti* babesiosis can be severe and occasionally fatal. Sporadic cases in other parts of America, often caused by non-*B microti* species, have been reported over the past 25 years, including two cases in California.[1,2] We report the third case of babesiosis contracted in California.

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## Report of a Case

The patient, a 22-year-old male soldier, presented to Hays Army Community Hospital at Ford Ord, California, with fever and chills, headaches, nasal congestion, and vomiting of five days' duration. He had been on field exercises at Fort Hunter Liggett in central California when his symptoms first began. There he was prescribed penicillin V (previously called penicillin phenoxymethyl) at a battalion aid station when he reported a history of splenectomy in 1986 for idiopathic thrombocytopenic purpura. Symptoms persisted after several days of penicillin treatment, and when he began to complain of neck pain radiating down his back, he was admitted to the hospital. On admission he also noted generalized weakness, myalgias, and anorexia. He did not have sore throat, diarrhea, or productive cough.

He had spent most of the preceding year on field exercises in central California. He had not had recent mosquito or rodent bites but did recall a tick bite at Fort Ord, in the coastal region of central California, about five months previously without subsequent illness. He had no travel to the American Northeast, Wisconsin, or Minnesota within the previous year and had never traveled to Eastern or Western Europe, Africa, Asia, or South America. He had no pets and had not had close contact with any domestic animals including dogs, cattle, or horses. There was no history of blood transfusion or intravenous drug use. He had received a pneumococcal vaccine shortly before his splenectomy.

On physical examination the patient appeared lethargic but oriented. His temperature was 39.4°C (103°F), his blood pressure was 136/70 mm of mercury, and pulse 98 beats per minute. The skin was without rashes or bite marks. There was neck pain that radiated down his back on neck flexion. The physical examination was otherwise unrevealing.

The initial laboratory blood findings are summarized in Table 1. A urinalysis was remarkable for 2+ protein and 10 to 15 red blood cells per high-power field. A lumbar puncture yielded clear, colorless fluid; the Gram's stain showed no organisms or cells; latex agglutination tests for *Streptococcus pneumoniae, Neisseria meningitidis,* and *Haemophilus influenzae* were negative; and

TABLE 1.—*Laboratory Blood Findings in a 22-Year-Old Man With Febrile Illness*

| Variable | Laboratory Value | |
|---|---|---|
| | *SI Units* | *Traditional Units* |
| Hemoglobin | 132 grams/liter | 13.2 grams/dl |
| Hematocrit | 0.40 | 40% |
| Leukocyte count | 5.6 × $10^9$/liter | 5,600/μl |
| Platelet count | 46.0 × $10^9$/liter | 46,000/μl |
| Westergren sedimentation rate | 2 mm/hr | same |
| Electrolytes | | |
| Sodium | 128 mmol/liter | same |
| Potassium | 3.9 mmol/liter | same |
| Chloride | 89 mmol/liter | same |
| Bicarbonate | 31 mmol/liter | same |
| Glucose | 6.1 mmol/liter | 111 mg/dl |
| Creatinine | 97.2 μmol/liter | 1.1 mg/dl |
| Blood urea nitrogen | 4.3 mmol/liter | 12 mg/dl |
| Alkaline phosphatase | 1.6 μkat/liter | 99 IU/liter |
| Aminotransferase* | | |
| Aspartate | 2.7 μkat/liter | 163 IU/liter |
| Alanine | 1.3 μkat/liter | 79 IU/liter |
| Lactate dehydrogenase | 25.5 μkat/liter | 1530 IU/liter |
| Total bilirubin | 34.2 μmol/liter | 2.0 mg/dl |

SI = Système International

*Formerly glutamic-oxaloacetic transaminase (aspartate) and glutamic-pyruvic transaminase (alanine).

the protein concentration was 280 grams per liter (28 mg per dl), and glucose 4.07 mmol per liter (74 mg per dl). The cerebrospinal fluid cell count was 7.0 × $10^6$ total cells per liter (7 per μl) with 5.0 × $10^6$ erythrocytes per liter and 2.0 × $10^6$ leukocytes per liter (2 per μl)—0.60 (60%) lymphocytes and 0.40 (40%) monocytes. A chest radiograph and sinus series were normal. Cultures of blood, urine, and cerebrospinal fluid were started, and the patient was treated empirically with intravenous benzylpenicillin pending results.

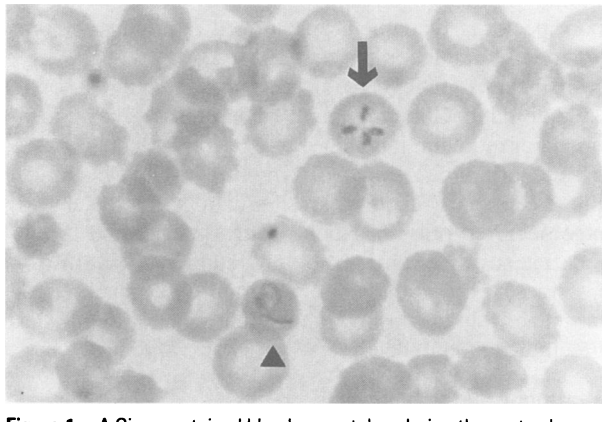

**Figure 1.**—A Giemsa-stained blood smear taken during the acute phase of the patient's illness shows a *Babesia* tetrad **(arrow)** and ring **(arrowhead)** (original magnification x 7,000).

On microscopic inspection of the blood smear (Figure 1), 1.2% of erythrocytes were found to be parasitized with small nonpigmented ring forms. Rare parasite tetrads were also observed, but no schizonts or gametocytes were seen. This established a preliminary diagnosis of babesiosis. Smears of serum and blood drawn within a week of presentation (acute phase) were sent to the Centers for Disease Control and Prevention (Atlanta, Georgia) for immunofluorescence testing and review. A titer of less than 1:8 for *B microti* returned, and parasite morphology was also consistent with non-*B microti* infection. Subsequent testing of the same serum specimen at the University of California at Davis veterinary laboratories revealed a positive titer of more than 1:64,000 to *Babesia gibsoni.* A Lyme assay was negative.

Treatment with quinine sulfate, 600 mg orally three times a day, and clindamycin phosphate, 1.2 grams intravenously twice a day, both for ten days, was instituted. The patient's symptoms began to subside within 24 hours. Parasitemia resolved after four days of treatment, as did hyponatremia, thrombocytopenia, hyperbilirubinemia, aminotransferase elevations, and proteinuria. Blood, urine, and cerebrospinal fluid cultures remained negative. He was discharged 11 days after admission without symptoms other than weakness, and five months later he reported no residual effects.

## Discussion

Babesiosis is a tick-borne zoonosis of wild and domestic animals that causes substantial livestock loss worldwide.[3,4] It predates malaria as the first disease of vertebrates for which an arthropod vector was demonstrated.[5] Reservoirs include cattle, sheep, horses, cats, dogs, deer, and rodents. Babesia is transmitted by nymphal ticks of the *Ixodes* genus.[5] These ticks can also harbor *Borrelia burgdorferi,* and co-transmission of Lyme disease has been reported.[6]

Humans acquire infection accidentally and play no role in its transmission except in rare transfusion-associated cases[7-11] and one possible case of transplacental transmission to an infant.[12] The earliest human case was reported in Yugoslavia in 1957.[1] More than 200 human cases have since been reported worldwide in various age groups, including infants and children.[12-15] In the United States most infections occur between May and July on Nantucket and Martha's Vineyard islands (Massachusetts), Long and Shelter islands (New York), and the neighboring coastal areas; *Babesia microti* is the predominant infecting species, and the white-footed mouse (*Peromyscus leucopus*) is the primary reservoir.[16] Recently Connecticut has also emerged as an endemic area.[10,17,18] Sporadic cases have been reported in Washington State,[19] New Hampshire,[11] Wisconsin,[20,21] and Georgia.[20]

Only two cases of babesiosis have previously been contracted in California, one north of San Francisco (1966)[1] and one in central California (1979).[2] In both instances, immunofluorescence testing for *B microti* was negative. The previous rarity of infection in California can probably be attributed to the lack of an endemic *Babesia* species. The current report adds to a growing body of evidence that this situation may be changing. The case reported herein is the first in California for which a probable causative species, *B gibsoni,* is suggested.[1,2] To our knowledge, no other human case of *B gibsoni*

babesiosis has been detected in any region of the United States. *B gibsoni* is harbored in dogs and is endemic in Ceylon, Malaysia, Korea, and Egypt.[22] No canine infection with *B gibsoni* was reported in the United States until 1968, when it was detected in a dog imported from an endemic area in Asia.[23] Subsequently, only isolated reports from several states, each involving one dog, had been published in the veterinary literature until 1991, when Conrad and co-workers reported a cluster of 11 cases of *B gibsoni* canine babesiosis in Los Angeles, Kern, and San Bernardino counties.[22] None of these dogs had a history of out-of-state travel, suggesting that *B gibsoni* has become established in dogs in certain areas of southern California.

The geographic location, animal source, and vector of infection in this case all remain unclear. Although the patient had no pets and had had no exposure to domestic animals including dogs, it is certainly likely that there were many dogs living nearby and that any one could have traveled to or been imported from an area of California in which *B gibsoni* is found. Alternatively, *B gibsoni* may now be established in wild or domestic animals (or both) native to central California, with which the patient could have come in contact while at home or on field exercises. Finally, although *Ixodes pacificus* is the suspected vector for babesiosis in California,[19] confirmation of transmission by this tick is lacking. Seroprevalence studies of humans, wild and domestic animals, and ticks in California, similar to those previously done in the eastern United States, will be required to help answer these questions.

## Pathophysiology

*Babesia* species multiply within erythrocytes, resulting in mechanical and probably autoimmune hemolysis.[24] Cellular immunity represents the primary line of defense. T lymphocytes activate macrophages, principally in the spleen, and induce the release of factors toxic to the parasites.[25] Thus, splenectomy worsens the course of infection. Infected patients have decreased numbers of T-suppressor cells and an increased T-cytotoxic cell fraction, allowing increased T-helper cell induction of antibody-producing B lymphocytes.[26] Antibodies play a disease-modifying role; passive transfer decreases parasitemia but does not prevent infection.[27] Advanced age and immunodeficiency also predispose to severe illness.[28,29]

## Clinical Presentation

The typical incubation period of babesiosis is one to four weeks, although many patients never recall a tick bite. There is a gradual onset of fever, chills, myalgias, and fatigue. The "picket fence" temperature curve seen with malaria is conspicuously absent. A physical examination is typically unrevealing except for fever and in some cases mild hepatosplenomegaly. Laboratory investigations usually show a hemolytic anemia of mild to moderate degree; parasitemia tends to be less than 10%.[3] Leukocyte counts are often depressed, and thrombocytopenia is frequently observed.[10] Alkaline phosphatase, liver aminotransferase, and bilirubin levels are typically slightly increased, and electrolyte levels are usually normal. Although these findings characterize most cases of babesiosis, a broad range of severity exists, and infections complicated by the adult respiratory distress syndrome,[5,30] high-output cardiac failure,[20] severe neutropenia,[9] disseminated intravascular coagulation,[31] acute renal failure,[32] and retinal infarcts[33,34] have been reported.

The cause of the hyponatremia and proteinuria in this patient is not known. This is the first reported case of *B gibsoni* babesiosis in a human; thus, the clinical characteristics of infection with this species have not been previously delineated, and it is not known whether they differ notably from those described for *B microti* infections. These abnormalities, which resolved rapidly with treatment of the disease, may have been unique to the patient in question and not hallmarks of *B gibsoni* infection, but further human cases will be required to clarify this issue.

The diagnosis of babesiosis is made by examination of a Giemsa-stained thick blood smear (Figure 1).[35,36] *Babesia* parasites are pleomorphic, nonpigmented and ring-shaped, with an average diameter of 1.0 to 2.0 micrometers. Tetrads, representing dividing parasites, are rare but pathognomonic for *Babesia.* Schizonts and gametocytes are not seen. Diagnosis is often delayed due to the rarity of the disease and its superficial resemblance to malaria. Consultation with an experienced parasitologist may help differentiate among these pathogens and may aid in elucidating the infecting species of *Babesia* based on subtle but well-described morphologic differences apparent on blood smear (K. Ehnert, DVM, MPVM, Monterey County Epidemiologist, oral communication, November 1991).

Indirect immunofluorescence testing for antibodies to *B microti* is available from federal and state parasitology laboratories; a titer of 1:64 or higher constitutes a positive test at the California State Infectious Disease Laboratory; however, this criterion may differ at other laboratories. Immunofluorescence testing for antibodies to less common species is available at specialized veterinary and university research laboratories.

In healthy patients with a spleen, infection is often asymptomatic. Clinical illness, when present, may be fulminant or indolent; it may last weeks and include a convalescent period of as long as 18 months.[29] Whereas clinical illness eventually abates, parasitemia may not.[11] Relapses have occurred only in patients with the acquired immunodeficiency syndrome, who may require long-term suppressive therapy.[28] Only one reported case of American babesiosis—interestingly, in a patient with a spleen—has directly resulted in death.[30]

## Treatment and Prevention

Patients with intact spleens may require only symptomatic treatment. Those without their spleen should be treated with antiparasitic drugs to decrease the severity of symptoms and ensure recovery. The regimen of choice is quinine, 650 mg orally three times a day, and clindamycin, 600 mg orally three times a day, or 1.2 grams given intravenously twice a day, for five to ten days.[3,8,20] Treatment failures have been reported,[11] and in these cases

exchange transfusion was curative.[9,11,37,38] Most of these patients were debilitated at the start or had a much higher degree of parasitemia than was seen in the case reported here.

Prevention is based on avoiding ticks.[4] When exposure is unavoidable, frequent body inspections should be done by individual and "buddy" systems. Nymphs should be detached immediately because the tick must feed for at least 12 hours before infection can be transmitted.[4,38] The use of diethyltoluamide-containing insect repellents is also recommended.[38] Unfortunately, transfusion-associated cases will probably continue to occur because widespread screening of donated blood products is not justified based on the low incidence of human infection. Babesiosis is a reportable disease in California (California Code of Regulations, sections 2502 and 2503).

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# Rocky Mountain Spotted Fever Following Cardiac Transplantation


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ROCKY MOUNTAIN SPOTTED FEVER was first recognized in the early 1900s in the Snake River Valley of Idaho and the Bitterroot Valley of western Montana.[1] In 1909, Howard Taylor Ricketts established the ixodid tick as the vector for the disease.[2] Following the tick bite, the incubation period averages seven days (4 to 10 days), and the disease varies in severity and course. Typically a patient's temperature remains elevated to 39°C to 40°C (102°F to 104°F). A characteristic rash, a relatively late manifestation, may appear on the wrists and ankles and extend throughout the body, including the palms and soles. Initially there are erythematous macules that blanch with pressure, but after several days the rash becomes maculopapular and petechial. The rash begins to clear as the

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