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CASE REPORT

Babesiosis: An emerging infectious disease that can affect those who travel to the northeastern United States

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Summary A case is presented of a healthy, 57 year-old male living in Ohio who traveled to Connecticut and later developed a severe case of babesiosis. The patient presented to his primary care physician with a history of intermittent fever and myalgias and was admitted to the hospital for investigation. On admission, he was found to have fever, left flank pain, and thrombocytopenia. The patient had an intact spleen, had no significant medical history, and had not received any blood products previously. During hospitalization, the patient developed pancytopenia and jaundice and became progressively more ill. A serendipitous conversation led to the investigation into babesiosis and empiric treatment. Infection with *Babesia microti* was confirmed by blood smear and PCR. In conclusion, obtaining a domestic, as well as international, travel history is important for identifying diseases, such as babesiosis, endemic to other areas.

Introduction

Babesiosis is a malaria-like illness that is transmitted through ticks. While babesiosis has long been known to infect animals and livestock, it is now emerging as an infectious disease in humans. It is transmitted by *Ixodes scapularis*, *Ixodes ricinus*, and *Ixodes ovatus*. The majority of human infection is caused by the species *Babesia microti*, but cases of babesiosis caused by *Babesia divergens*, *Babesia duncani*, and *Babesia venatorum* have been reported.[1] Additionally, patients have acquired babesiosis through transfusion of blood products collected from asymptomatic infected donors.

Infection with *Babesia* spp. ranges from subclinical to severe and can be fatal, even with appropriate treatment. Those at greatest risk are elderly, asplenic, and immunocompromised patients.[2] Treatments include antimicrobial therapy, transfusion, and, in severe cases, exchange transfusion.[3]

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In the United States, babesiosis is endemic to the New England region with sporadic cases of babesiosis infection reported in Wisconsin and Minnesota. In non-endemic areas, reported cases of babesiosis are rising due to infection through transfusion, as well as travel to endemic areas. The case of babesiosis in a 57 year-old Caucasian male residing in Ohio is presented.

Case

A 57 year-old Caucasian male presented to his primary care physician in Ohio in July, 2010 with a four-day history of intermittent fevers, general malaise, decreased appetite, and left flank pain. Previously, the patient was in good health with a medical history significant only for hypertension and high cholesterol. He had traveled to Connecticut four weeks prior where he stayed at a country estate, but he denied camping or tick exposure. He and his wife walked the grounds of the property near wooded areas but never entered any wooded area. The owner of the estate had two pet dogs, but the patient did not have any other exposure to animals. The only sick contact the patient had was his wife, who had been ill with a sore throat, earache, and fever that resolved without antibiotics a few weeks after returning from Connecticut. An electrocardiogram (ECG) performed in the office showed a short run of supraventricular tachycardia and a dropped beat suggestive of heart block, prompting the primary care physician to admit the patient to the hospital for monitoring and cardiology consult.

On admission, the patient was alert but pale with a temperature of 37.9 °C, blood pressure of 134/73 mmHg, pulse of 80 beats/min, and respiration rate of 18 breaths/min. Physical exam findings were unremarkable except for mild tenderness at the left flank. His cardiac exam did not reveal jugular venous distension, murmurs, or gallops. Auscultation of the chest revealed clear breath sounds bilaterally. The patient did not have any rash or neurologic findings. The patient's admission laboratory results, shown in Table 1, were notable for thrombocytopenia. The urine's appearance was yellow and clear, and urinalysis was unremarkable except for trace hemoglobin. Due to the patient's thrombocytopenia, a hematologist was consulted. The patient's continued intermittent fevers and malaise prompted a consultation with an infectious disease specialist.

The infectious disease specialist initially suspected a tick-borne disease including Lyme disease and babesiosis. However, he discussed the case with a colleague who dismissed the diagnosis of babesiosis, because the patient was not immunocompromised and had an intact spleen. Specialized testing for *Ehrlichia* spp. PCR, *Leptospira* spp., Q fever, Rocky Mountain Spotted Fever, *Rickettsia rickettsii*, and other infectious diseases were sent to Quest Diagnostic Nichols Institute (Chantilly, VA). These results did not return for several days, and no empiric treatment was started while waiting for test results. Results from the laboratory investigation are shown in Table 2.

A CT scan of the abdomen and pelvis performed on hospital day 2 showed borderline mild splenomegaly. The patient was on cardiac monitoring, and during his hospitalization, premature atrial complexes, premature supraventricular complexes, and atrial fibrillation with rapid ventricular response were noted on ECG on hospital day 3, 5, 6, and 7. An echocardiogram performed on hospital day 2 showed mild left ventricular hypertrophy with an ejection fraction of 70% and Stage 2 diastolic dysfunction. The right atrium, left atrium, right ventricle, and all valves were normal in size and function.

During the patient's hospital course, the patient appeared progressively more ill, and the patient continued to have thrombocytopenia. His hemoglobin, hematocrit, and white blood cell count steadily decreased until the patient became pancytopenic. On hospital day 3, the patient started to appear jaundiced. The hematologist specialist was concerned about the possible diagnosis of

Table 1 Laboratory study results.

	On admission 7/12	On day 3 14	On day 7 18	On discharge (day 11) 22	Reference range
Sodium Serum (mEq/L)	136	138	138		136–145
Potassium Serum	5.0	4.1	4.0		3.5–5.1
Chloride Serum (mEq/L)	103	105	106		98–107
CO_2 Serum (mEq/L)	28	28	27		21–32
Glucose Serum (mg/L)	114	119	103		74–106
BUN S Serum (mg/L)	13	9	10		7–18
Creatinine Serum (mg/L)	1.0	1.0	1.2		0.8–1.3
Calcium Serum (mg/L)	8.7	8.2	7.9		8.5–10.1
Anion Gap	11	9			8–16
WBC (thou/cmm)	6.3	4.6	3.7	4.9	4.4–9.7
Hgb (g/dL)	12.7	11.7	8.4	8.2	13.2–17.4
Hct (37.7)	37.7	35.2	24.8	24.4	39.6–50.7
MCV (fl)	92.0	91.7	89.5	89.1	81.8–95.6
RDW %	14.8	15.0	14.7	14.9	11.8–14.5
Platelet	44	Clumped	91	200	150–370
D-Dimer, Quant (ng/ml(FEU))		2539.7			<500
Fibrinogen		648			180–430
LDH-LD		544	395	476	100–190