INTRODUCTION

Babesiosis is a tick-transmitted, zoonotic disease that is endemic in the Northeastern and upper Midwestern United States. There are several species of Babesia that can infect humans, Babesia microtic being the most prevalent. [1] The disease manifestation is broad and may present as flu-like illness with anemia, thrombocytopenia and jaundice. The average incubation period is one to nine weeks. [2] We report a 71-year-old male who was infected with babesiosis following a trip to Maryland.

CASE

A 72-year-old male with a medical history of hypertension, hyperlipidemia and COPD presented to the emergency department with fevers, worsening shortness of breath and weakness. He denied orthopnea, cough, headache, abdominal pain, melena, hematochezia, urinary complaints or sick contacts. He had recently traveled to Maryland however denied any history of international travel. He presented hemodynamically unstable with blood pressure as low as 64/32 millimeters of mercury (mmHg). Heart rate was 76 beats per minute and he was not hypoxic; saturating at 96% on room air. He was febrile to 38.2 degrees Celsius (°C). He was nontoxic appearing and asymptomatic to his profound hypotension. The rest of his physical exam was largely unremarkable. Initial laboratory evaluation revealed a hemoglobin of 7.8 grams per deciliter, thrombocytopenia at 60,000 International Units per Liter [IU/L]. He had evidence of Hemolysis with an elevated total bilirubin of 1.6 milligram per deciliter [mg/dL], Lactate dehydrogenase of 805 IU/L and Haptoglobin less than 8.0 mg/dL. He had no lactic acidosis nor positive direct coombs test. He received 2 units of Packed Red Blood Cells, was resuscitated with 2 liters of Normal saline, 1 liter of lactated ringers and started on the broad-spectrum antibiotics’ piperacillin-tazobactam and vancomycin for treatment of presumed sepsis of unknown origin. The patient was subsequently admitted to the intensive care unit given persistent hemodynamic instability for closer monitoring. He briefly required Phenylephrine for vasopressive support. A peripheral smear revealed mature granulocytes, intra erythrocytic ring forms with no extra erythrocytic parasites seen. Given epidemiology, the diagnosis was felt to be babesiosis as he had not been out of the country and had no prior transfusion history to suspect malaria. Parasitemia was below 1%; no plasma exchange was initiated. He was started on atovaquone and azithromycin for babesiosis and clinically improved rapidly. Within 24 hours of admission, he was downgraded to the general medical floor and eventually discharged home after 3 days of hospital admission with plans to complete a 10-day course of treatment for babesiosis. Follow up evaluation revealed he had no sequelae from his illness.

CONCLUSIONS

Although this patient was immunocompetent with no splenic dysfunction, he still suffered a moderate Babesiosis infection. Although his parasitic load was less than 1% he experienced hemodynamic instability which is generally associated with more severe disease. Babesiosis is often misdiagnosed as a malaria-borne illness, however a Maltese cross on peripheral blood smear is pathognomonic for babesiosis. [3] Given babesiosis may present subclinical or be fulminant in immunocompromised or asplenic patients, it is important it is accurately diagnosed and treated.

REFERENCES