Babesiosis Microti is a parasitic alveolate that is usually transmitted by Ixodes scapularis ticks. In the United States, the endemic areas of Babesiosis include the Northeast and Upper Midwestern regions [1]. Symptoms include fever, malaise, fatigue, vomiting, and jaundice [1]. Current therapy primarily consists of a combination of azithromycin and atovaquone. Clindamycin and quinine may be administered in severe cases. For its emerging health risk and importance of recognizing Babesiosis outside of its endemic area, and that worldwide, clinicians must be aware of the several presenting manifestations of Babesiosis Microti. It was initially thought that the patient suffered from a malaria-borne illness. Diagnostic workup for each of these cases similar to ours, included complete blood workup, basic metabolic panel, hemolysis workup revealed low haptoglobin, high serum lactate dehydrogenase, and high ESR. While in the ICU he started on atovaquone, clindamycin, and azithromycin. After the initial blood transfusion, his hemoglobin increased from 6.7 g/dl to 7.9 g/dl. The patient complained of occasional malaise and weakness at times, but tolerated his meals and slept well. His chest X-ray was normal and a computerized tomography (CT) scan of the abdomen revealed some mild peri-portal edema suggestive of inflammation of the liver along with hepatomegaly (Figure 1 and Figure 2, respectively). Status post initial blood transfusion the patient’s hemoglobin level reduced again from 7.9 to 6.8 g/dl. Another unit of blood was prepped and transfused. This brought the hemoglobin level to 7.8 g/dl and the level remained stable, and continued to rise. The patient’s symptoms improved and he was discharged to complete a 7-day course of oral clindamycin and azithromycin.

Case Report

The patient is a 29-year-old Hispanic male who presented at the emergency department (ED) with fever of four days duration. Patient initially presented to the hospital with fever of 102-103 degrees. He reported he was weak and felt like he had the flu. Patient provided a past medical history of travelling to an area that has high concentration of Ixodes tick carrying Babesia Microti. It was initially thought that the patient suffered from a malaria-borne illness. Diagnostic workup for each of these cases similar to ours, included complete blood workup, basic metabolic panel, hemolysis workup revealed low haptoglobin, high serum lactate dehydrogenase, and high ESR. While in the ICU he was started on atovaquone, clindamycin, and azithromycin. After the initial blood transfusion, his hemoglobin increased from 6.7 g/dl to 7.9 g/dl. The patient complained of occasional malaise and weakness at times, but tolerated his meals and slept well. His chest X-ray was normal and a computerized tomography (CT) scan of the abdomen revealed some mild peri-portal edema suggestive of inflammation of the liver along with hepatomegaly (Figure 1 and Figure 2, respectively). Status post initial blood transfusion the patient’s hemoglobin level reduced again from 7.9 to 6.8 g/dl. Another unit of blood was prepped and transfused. This brought the hemoglobin level to 7.8 g/dl and the level remained stable, and continued to rise. The patient’s symptoms improved and he was discharged to complete a 7-day course of oral clindamycin and azithromycin.

Introduction

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<table>
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<tr>
<th>Image Captions</th>
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<tr>
<td>A: Chest X-Ray</td>
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<td>B: CT Scan of the Abdomen</td>
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<td>C: Peripheral Blood Smear</td>
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Discussion

Clinicians need to be aware of Babesiosis in endemic and non-endemic parts of the country. A detailed travel history is crucial for diagnosis and treatment (Kunimoto et al.,). While most cases of Babesiosis may appear to be subclinical, however, symptomatic cases are more likely in asplenic patients (Kunimoto et al.,). Our patient presented with symptoms and gave a past medical history of splenectomy. When symptomatic, patients may present with nonspecific symptoms such as headache, muscle aches, fever, and fatigue (Kunimoto et al.,). In asplenic patients such as our reported case current treatment consists of atovaquone and azithromycin or clindamycin and quinine as an alternative treatment for severe disease along with blood transfusion (Kunimoto et al.,).

Conclusion

Clinicians should have a heightened awareness of Babesiosis as it can present in nonendemic areas. Thorough travel history should be elicited during initial interviewing of the patient. As such the differential diagnosis of Babesiosis should still be considered even if seen outside its incubation period. Coinfection with other Ixodes-borne pathogens in any patient with Babesiosis must be thoroughly investigated while working up for the Babesia primary infection as the clinical course can rapidly deteriorate. Severe disease may occur in immunocompromised hosts as seen in our patient with a past surgical history of splenectomy.

References


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