Spontaneous Splenic Rupture Caused by *Babesia microti* Infection

David P. Kuwayama* and Renato J. Briones*

*Babesia* has not been previously associated with spontaneous splenic rupture. We describe an otherwise healthy 61-year-old man with symptomatic *babesiosis* whose spleen ruptured during hospitalization. Although this complication is rare, practitioners who commonly treat patients with *babesiosis* should be aware of its potential occurrence.

Human infection with species of the protozoan genus *Babesia* was first identified in 1957 [1], and it has since been described in North America, Europe, Asia, and Africa, although the majority of cases occur in western Europe and the northeastern United States. In the United States, infection is usually caused by the species *Babesia microti* and is transmitted by the bite of the tick *Ixodes scapularis* [2].

The majority of patients infected with *B. microti* exhibit no symptoms. The development of symptomatic disease is typically restricted to 3 subgroups of patients: elderly individuals, immunocompromised patients, and asplenic patients. In such patients, *B. microti* parasites can multiply to levels sufficient to cause hemolytic anemia, thrombocytopenia, and atypical lymphocytosis. The resulting clinical picture spans a broad spectrum of severity. Mild symptoms include fever, malaise, nausea, anorexia, and weakness. More-severe disease can cause organ dysfunction, including mental status change, pulmonary edema, renal failure, hepatosplenomegaly, and jaundice. At its most severe, *babesiosis* causes septic shock, multisystem organ failure, and death. In the United States, 5%–6.5% of all patients who develop symptomatic infection with *B. microti* die due to the infection [3, 4].

Although *B. microti* infection can cause splenic enlargement, splenic rupture has not previously been recognized as a complication of *babesiosis*. We describe here a man in otherwise excellent health, with no history of trauma, whose spleen spontaneously ruptured on day 4 of hospitalization for treatment of symptomatic *babesiosis*. Emergency laparotomy and splenectomy were required to restore hemodynamic stability.

**Case report.** The patient was a 61-year-old man from southern New Jersey with no medical history and a surgical history notable only for dural sinus disease. He received no routine medications and had no known drug allergies. He was a nonsmoker and only rarely drank alcohol. He was married without children and worked a professional desk job in an office. He denied any recent foreign travel but admitted to spending a significant amount of time outdoors, both on golf courses and in heavily wooded areas. The patient had no history of significant blunt or penetrating trauma.

Six days before admission to the hospital, the patient began to experience fever, chills, headache, and malaise. These symptoms progressively worsened, with the patient’s temperature spiking as high as 39.7°C. The day before hospital admission, he was seen by his primary care physician, who ordered bloodwork and prescribed a 5-day course of oral sulfamethoxazole-trimethoprim (800 mg/160 mg twice daily). The following day, on discovering that the patient was experiencing thrombocytopenia (table 1), the physician referred him to the emergency department.

On evaluation in the emergency department, the patient appeared to be moderately ill and exhibited shaking chills. Vital signs were notable for a striking temperature of 41.3°C, blood pressure of 111/67 mm Hg, heart rate of 93 beats per min, and oxygen saturation of 95% on room air. Physical examination revealed mildly icteric skin without petechiae or bruising, no palpable lymphadenopathy, clear lung fields, and normal heart sounds; the findings of an abdominal examination were benign without palpable hepatosplenomegaly. A complete blood count with leukocyte differential was performed (table 1). Given the high fevers, shaking chills, thrombocytopenia, and atypical leukocyte differential, a peripheral thin blood smear was ordered to screen for malaria and other intra-erythrocytic parasites. The smear had findings positive for the presence of intra-erythrocytic inclusions, including tetrad forms, consistent with *B. microti* infection; the level of parasitemia was estimated to be 5%.

The patient was admitted to the medical service with a diagnosis of acute symptomatic *babesiosis*. He was administered continuous intravenous fluids and initiated a 7-day course of antibiotic therapy with oral atovaquone (750 mg twice daily), azithromycin (500 mg once on day 1, then 250 mg daily from...
Table 1. Laboratory data for a patient with spontaneous splenic rupture caused by *Babesia microti* infection.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Reference range</th>
<th>Day before hospitalization</th>
<th>Day of hospitalization</th>
<th>Day after hospitalization</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC count, cells/mm³</td>
<td>4800–10,800</td>
<td>5600</td>
<td>4400</td>
<td>3700</td>
</tr>
<tr>
<td>Neutrophil percentage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absolute</td>
<td>37–73</td>
<td>...</td>
<td>63.0</td>
<td>...</td>
</tr>
<tr>
<td>Bands</td>
<td>0–1</td>
<td>...</td>
<td>33.0</td>
<td>...</td>
</tr>
<tr>
<td>Lymphocyte percentage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absolute</td>
<td>20–55</td>
<td>...</td>
<td>19.6</td>
<td>...</td>
</tr>
<tr>
<td>Atypical</td>
<td></td>
<td>...</td>
<td>2.0</td>
<td>...</td>
</tr>
<tr>
<td>Monocyte percentage</td>
<td>2.5–10</td>
<td>...</td>
<td>16.7</td>
<td>...</td>
</tr>
<tr>
<td>Eosinophil percentage</td>
<td>0–6</td>
<td>...</td>
<td>0.2</td>
<td>...</td>
</tr>
<tr>
<td>Basophil percentage</td>
<td>0–2</td>
<td>...</td>
<td>0.5</td>
<td>...</td>
</tr>
<tr>
<td>Hemoglobin level, g/dL</td>
<td>13.0–18.0</td>
<td>14.1</td>
<td>14.2</td>
<td>11.0</td>
</tr>
<tr>
<td>Platelet count, platelets/mm³</td>
<td>130,000–400,000</td>
<td>51,000</td>
<td>39,000</td>
<td>33,000</td>
</tr>
<tr>
<td>Haptoglobin level, mg/dL</td>
<td>43–212</td>
<td>...</td>
<td>...</td>
<td>&lt;35</td>
</tr>
</tbody>
</table>

**NOTE.** Abnormal values are indicated by boldface type.

day 2 through day 7) and doxycycline (200 mg twice daily). By day 2 of hospitalization, the shaking chills and headache had resolved; however, the patient continued to exhibit daily low-grade cycling fevers, and leukopenia and thrombocytopenia persisted. Because the patient’s clinical trajectory was considered to be generally positive, no additional thin blood smears were performed.

On hospital day 4, soon after having used the bathroom, the patient developed sharp left upper quadrant pain. With the exception of having exerted a normal amount of Valsalva pressure to pass a stool, the patient had not engaged in any kind of strenuous or rough activity and had not experienced any kind of blunt force trauma. Over the course of the next hour, the patient’s abdominal pain worsened, and he became diaphoretic and hypotensive. An emergent CT of the abdomen and pelvis with intravenous contrast (figure 1) revealed a large amount of complex intra-abdominal fluid, consistent with hemoperitoneum, and a linear lucency through the inferior pole of the spleen, suggestive of a splenic laceration.

Because of the patient’s rapidly deteriorating clinical status and the radiologic findings, he was taken emergently to the operating room for exploration. By the time that the patient arrived in the operating room, his abdomen was grossly distended. At laparotomy, 4 liters of blood were evacuated from the peritoneal cavity. The source of hemorrhage was identified as an actively bleeding grade IV laceration in the inferior pole of the spleen. On inspection and palpation, the spleen was only slightly enlarged but was highly abnormal in texture, with a soft, diffusely nodular and friable parenchyma. It was believed that such parenchyma would not reliably hold stitch, so the decision was made to perform splenectomy instead of splenorrhaphy.

The patient tolerated the procedure well, although he required intraoperative transfusion of 4 units of packed RBCs and 2 units of fresh frozen plasma to maintain hemodynamic stability. After the procedure, the patient was extubated without difficulty and transferred to the intensive care unit for monitoring, where he remained hemodynamically stable. Low-grade fever persisted, and the patient’s preoperative oral antibiotic regimen was resumed on day 2 after the procedure. On day 3 after the procedure, the patient’s fever ceased, and he experienced the return of bowel function; he was transferred out of the intensive care unit and initiated a regular diet. By day 5 after the procedure, he was ambulating without difficulty and had been afebrile for 2 days, and his platelet count had returned to normal levels. The patient was discharged home on day 6 after the procedure. Of note, throughout his hospitalization, no thin blood smears were performed after the initial diagnostic thin blood smear. The patient was in excellent condition at a routine 30-day follow-up visit.

Histologic examination of the removed spleen revealed significant red pulp hyperplasia containing plasma cells, lymphocytes, immunoblasts, and large numbers of histiocytes exhibiting erythrophagocytic activity. The white pulp was unremarkable. In searching for *Babesia* organisms, the pathologist made note of “suspicious” red cells but did not directly visualize intra-erythrocytic organisms. Because the gross specimen had been submitted in formalin, the pathologist was unable to perform touch imprint examination.

**Discussion.** No reports exist in the literature describing spontaneous splenic rupture as a result of infection with *Babesia* species; this appears to be the first documented case. A report in the lay press from 2005 described a man in Minnesota who underwent emergent splenectomy for spontaneous rupture also
Figure 1. CT of the upper abdomen showing a splenic laceration (arrow) with hemoperitoneum

attributed to babesiosis [5]; however, this case does not appear to have been reported in a medical journal.

Several infectious agents have been reported to cause spontaneous splenic rupture, including Plasmodium species [6], cytomegalovirus [7], Epstein-Barr virus [8], Coxiella species [9], Bartonella species [10], murine typhus [11], Staphylococcus species [12], and Streptococcus species [13]. Interestingly, splenic rupture resulting from bloodstream infection does not always appear to be associated with splenomegaly [13]. In our patient, the spleen was roughly normal in size; the most striking feature of the spleen was a highly abnormal parenchyma, which was soft, nodular in texture, and extremely friable. Our clinical impression was that rupture resulted, not from splenomegaly and elevated intracapsular pressure, but from degradation of parenchymal integrity.

Although neither intra-erythrocytic inclusions nor parasitic intravascular cytoadherence were directly visualized in the splenic tissue, the pathologist believed that such findings were rendered unlikely by the thickness and quality of tissue sections. We still believe that it is reasonable to attribute this patient’s splenic rupture to Babesia species infection because of the peripheral blood smear results, the degree of red pulp inflammation, and the absence of other potential etiologies.

Although spontaneous splenic rupture is clearly a rare complication of Babesia species infection, physicians who commonly encounter or treat babesiosis should be aware of the potential for its occurrence. Our case suggests that the absence of palpable splenomegaly in patients with Babesia species infection does not exclude the possibility of subsequent splenic rupture. Patients being treated for Babesia infection as out-patients should be explicitly instructed to present to the emergency department if they develop new-onset or worsening left upper quadrant pain. Because prompt diagnosis and treatment of splenic rupture can be life-saving, health care practitioners should have a low threshold of suspicion for this diagnosis, especially in patients with known infection who develop sudden hemodynamic instability.

Acknowledgments


References