Spontaneous splenic rupture associated with *Escherichia coli* bacteremia and dual antiplatelet therapy

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ABSTRACT

Splenic rupture is a life-threatening condition that usually occurs after blunt abdominal trauma. It can lead to hemorrhagic shock and death if not diagnosed quickly. Rarely, spontaneous rupture of the spleen may occur without a history of trauma. In these cases, establishing a diagnosis is challenging due to the ambiguous presentation of the conditions associated with it. Infections and hematological malignancies are the two most common causes of spontaneous splenic rupture. Here, we report a case of spontaneous splenic rupture in the setting of Gram-negative bacteremia and recent initiation of dual antiplatelet therapy.

KEYWORDS Dual antiplatelet therapy; Gram-negative bacteremia; spontaneous splenic rupture

Spontaneous splenic rupture (SSR) is a rare and life-threatening condition that occurs without a previous history of trauma. It can be classified as atraumatic-idiopathic or atraumatic-pathologic depending on the etiology identified during pathological examination. Atraumatic-pathologic, the most common presentation, is caused by hematological malignancies and infections in about half of the cases.^{1,2} With the advent of oral anticoagulants, there has been an increased report of splenic rupture associated with these medications, as well as with dual antiplatelet agents.^{3,4} Multiple mechanisms are thought to be involved in the pathophysiology of this condition. Splenomegaly has been proposed as a risk factor for SSR in patients with infectious mononucleosis. Inflammation, necrosis, micro infarcts, and subcapsular hematomas are often identified during pathological examination of resected spleens after rupture. Increased abdominal pressure (from, e.g., cough, sneeze, and defecation) and derangements in the coagulation system together with the presence of any of the factors mentioned above can prompt the formation of subcapsular hematomas and subsequent rupture.^{5,6}

CASE DESCRIPTION

A 60-year-old woman with a past medical history of hypertension, type 2 diabetes mellitus, coronary artery disease, and heart failure presented to the emergency department complaining of acute-onset left upper-quadrant pain. Vital signs at initial presentation were a blood pressure of 85/63 mm Hg, heart rate of 120 beats per minute, and temperature of 37.2°C. She reported having a fever and low back pain for the past week and frequent urinary tract infections. Her home medications included aspirin and prasugrel, both started 1 month earlier, after undergoing percutaneous coronary intervention for an inferior wall myocardial infarction. Her white blood cell count was 4000/mm³; hemoglobin, 11.8 g/dL; hematocrit, 36.2%; and platelets, 348/mm³. Computed tomography of the abdomen and pelvis showed a large subcapsular hematoma with frank hemoperitoneum, as well as evident extravasation from the spleen (*Figure 1*). The patient was found to be in hemorrhagic shock and was resuscitated appropriately with intravenous fluids.

Emergency laparotomy found approximately 600 mL of clotted blood in the abdomen, and a splenectomy was performed. Blood and urinary cultures obtained at initial pres-

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Figure 1. CT of the abdomen revealing a large subcapsular hematoma with active extravasation.

Figure 2. Hematoxylin and eosin stain showing a background of splenic parenchyma with hemorrhage and mixed inflammatory infiltrate. Bacteria are large and rod-shaped.

entation were positive for extended-spectrum beta-lactamase (ESBL) *Escherichia coli*. The patient was treated with broad-spectrum antibiotics according to culture sensitivities. Biopsy from the spleen showed extravasated blood, a mixed inflammatory infiltrate, and large rod-shaped bacteria (Figure 2). Formalin-fixed embedded tissue was sent for polymerase chain reaction testing, which came back negative for bacterial DNA. After 18 days, the patient's condition improved and she was discharged without further complications. Pneumococcal, meningococcal, and *H. influenzae* vaccines were administered before discharge.

DISCUSSION

Among infectious etiologies, malaria and mononucleosis are the two most common entities associated with SSR.² Both conditions are known to cause splenomegaly and inflammation, predisposing to rupture.⁷ SSR associated with bacterial infections such as *Salmonella* spp., *Brucella* spp., *Bartonella henselae*, and *Haemophilus influenzae* have also been described.^{8,9} However, to our knowledge, SSR in the setting of *E. coli* bacteremia has not been reported before. Spleens from birds inoculated with strains of *E. coli* showed hyperemia, expansion of the red pulp, fibrinopurulent exudates, and hemorrhage 8 hours after inoculation.¹⁰

Histopathological features of the resected spleen in our patient showed acute splenitis and the presence of elongated

forms with hematoxylin and eosin stain. No abscess collection was seen on macroscopic examination. The presence of formalin could have decreased the sensitivity of the polymerase chain reaction testing to detect any bacterial DNA. Guyard et al showed a 53% decrease in DNA quantity on formalin-fixed tissue blocks after a median storage period of 5.5 years.¹¹

Blood and urine cultures obtained during the initial presentation were positive for ESBL *E. coli*. There is a strong possibility that the recurrent urinary tract infections in this patient led to bacterial seeding of the spleen, and the subsequent inflammation along with the use of prasugrel prompted the SSR.

Splenic rupture requires immediate surgical intervention in most cases.² A conservative approach with nonoperative management and/or embolization is considered in low-grade splenic injury and hemodynamically stable patients.¹² SSR symptoms are nonspecific and can be confounded by other causes of abdominal pain. If unrecognized, SSR can lead to hemorrhagic shock and death. This case highlights two mechanisms that lead to SSR and also expands the spectrum of infectious agents associated with this condition.

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