Splenic infarction in alcoholic hepatitis

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ABSTRACT

Splenic infarctions have been reported in cirrhotic patients with comorbidities (e.g., primary biliary cirrhosis), but there is little to no mention of spontaneous splenic infarction in alcoholic hepatitis. This condition is notable, as splenic function has been shown to decrease in alcoholic liver disease, but the exact pathophysiology has been unclear. We describe a relatively young cirrhotic woman who developed splenic infarctions in the setting of alcoholic hepatitis.

KEYWORDS Acute hepatitis; alcoholic liver disease; cirrhosis; splenic infarction

Splenic infarction is generally described as either having a cardiovascular etiology or arising in hypercoagulable states. To date, only case reports exist describing splenic infarctions in patients with cirrhosis. In this report, we describe a young cirrhotic woman presenting with alcoholic hepatitis who was subsequently found to have splenic infarctions.

CASE DESCRIPTION

A 40-year-old white woman with known hepatic steatosis secondary to alcohol abuse and irritable bowel syndrome presented to our hospital with worsening jaundice, nausea, and vomiting. Her alanine aminotransferase was 54 U/L; aspartate transaminase, 246 U/L; alkaline phosphatase, 589 U/L; total bilirubin, 14.0 mg/dL; international normalized ratio, 1.9; and ethanol level, 240 mg/dL. Her white blood cell count was 20,900/ μ L with 92% neutrophils. At this point, despite a Maddrey score >50 , we decided to forego corticosteroids given her infection. After a 5-day course of intravenous ceftriaxone, however, her white blood cell count decreased to 18,400/ μ L.

Because of the patient's worsening abdominal symptoms after a course of antibiotics, a right upper quadrant ultrasound was done, which showed only hepatic steatosis. Computed tomography with contrast showed cirrhosis with progressive moderate hepatomegaly as well as borderline splenomegaly with mild ascites (*Figure 1a*). She began to have small amounts of tarry stool, and an esophagogastroduodenoscopy yielded

nothing of clinical interest. No infectious process was discovered even as abdominal pain, leukocytosis, and total bilirubin levels continued to worsen. Antinuclear antibody, Gliadin IgA, tissue transglutaminase, human immunodeficiency virus, and hepatitis panels were negative. Blood cultures remained negative, but our infectious diseases service recommended treating her with empiric meropenem.

With her severe abdominal pain and white blood cell count $>50,000/\mu$ L even after a 7-day course of meropenem, a contrast CT scan was repeated (10 days after initial CT imaging). At this time, lesions in the spleen concerning for infarction were seen (*Figure 1b*). The decision was made to manage her conservatively, and her condition improved until discharge. Upon discharge, her white blood cell count was 13,000/ μ L. Her abdominal pain and nausea had resolved to a comfortable level. Review of past electrocardiograms showed normal sinus rhythm. A CT scan 6 weeks after discharge showed resolution of these infarctions.

DISCUSSION

In a patient who presented with alcoholic hepatitis, with a CT scan showing cirrhotic morphology, we have described splenic infarctions that had not been noted on previous imaging. She was ultimately able to be managed conservatively. A mere handful of case reports exist describing splenic infarctions in cirrhotic patients with portal hypertension. In fact, of 152 patients with splenic infarctions in one study, only 3 cases were found to be secondary to cirrhosis or portal

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Figure 1. (a) Initial CT scan. (b) CT scan 10 days later showing splenic infarctions.

hypertension.¹ Most patients ≤ 40 years had an associated hematologic disorder, while those >41 years often had an embolic event. Left upper quadrant abdominal pain was the most common complaint in all patients, regardless of age or etiology. Leukocytosis was present in 49% of patients.

It is currently hypothesized that splenic infarctions in cirrhosis are due more to hemodynamic changes than organic occlusion of splenic vasculature. Splenomegaly results from portal hypertension, and this is believed to increase splenic oxygen requirements disproportionate to the body's oxygen-delivering capacity. Consequently, the spleen suffers anoxic injury and subsequent infarction.²

The acuity of splenic infarction in our patient is notable, developing at some point in the 10 days between her CT scans. Judging by the timeline of her imaging and clinical picture, we believe the burden of her alcoholic hepatitis coupled with her urinary tract infection and then melena further compromised her oxygen-delivering capacity and she developed these infarctions. The nature of resolution of these infarcts is also notable; in one case report of splenic infarction in a cirrhotic patient, spontaneous resolution was not seen, even after 6 months.² Spontaneous resolution of splenic infarction has, however, been described in patients with cardioembolic splenic infarctions.^{3,4}

The patient's prolonged leukocytosis without evidence of active infection is likely related more to leukemoid reac-

tion than splenic infarction. Mitchell et al found that patients with alcoholic hepatitis who developed leukemoid reactions generally had white blood cell counts $>40,000/\mu\text{L}$, and the average was about $73,000/\mu\text{L}$.⁵ They noted a remarkably high mortality rate, with patients dying a mean of 32 days after admission, regardless of Maddrey score.⁵ Our patient was able to be discharged home after symptom resolution.

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