Delayed Splenic Rupture: A Myth or A Reality

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Introduction

The spleen is the most commonly injured organ in blunt abdominal trauma.[1] Most splenic injuries manifest immediately after trauma, however, delayed splenic rupture (DSR) may occur days to weeks following a blunt abdominal trauma. It has been debated whether delayed splenic rupture is simply a delayed diagnosis of a missed contained splenic rupture or rather an initially latent splenic lesion that may have been undetected by conventional imagery and evolved later to rupture.[2,3]

The majority of case reports describing DSR have relied mainly on clinical evaluation without obtaining an initial computed tomography (CT) scan of the abdomen to confirm what is genuinely delayed. Very few articles have reported a truly delayed splenic rupture with a normal CT scan upon admission.[4]

Case Report

A 42-year-old female presented with abdominal pain that started 2 weeks prior following a non-collision, deceleration motor vehicle accident in which she was wearing her seatbelt. Her history included gastric bypass four years prior and systemic lupus erythematosus (SLE).

On initial examination, the patient was stable and in no acute distress. The abdominal examination revealed a mild left upper quadrant tenderness and a palpable spleen, but no evidence of trauma. Further findings included a butterfly rash.

The patient's hemoglobin was 6.9 g/dl and hemoccult testing was positive. A CT scan of the abdomen with intravenous contrast obtained on admission (see Figure 1A) was unremarkable, except for the splenic size, which was in the upper limits of normal. After four units of packed red blood cells were transfused, her hemoglobin increased to 11.0 g/dl. She was scheduled for esophagogastroduodenoscopy and colonoscopy. She subsequently developed worsening nausea and emesis with retching followed by increasing left upper quadrant (LUQ) pain without evidence of hemodynamic compromise. A repeat hemoglobin revealed a drop to 7.0 g/dl.

The patient denied hematemesis, hematochezia, or melena. A LUQ ultrasound revealed free abdominal fluid, and a stat repeat abdominal CT scan with intravenous and oral contrast (see Figure 1B) demonstrated intraperitoneal free fluid and capsular changes consistent with splenic rupture.

Surgery was consulted. The decision was made to proceed with splenic artery embolization. Embolization was successful, with five coils deployed in the distal splenic artery. The follow-up hemoglobin remained stable. The workup of splenomegaly, including viral serology and clotting studies, was normal. The rest of her hospital course was uneventful, and she was discharged a few days later.

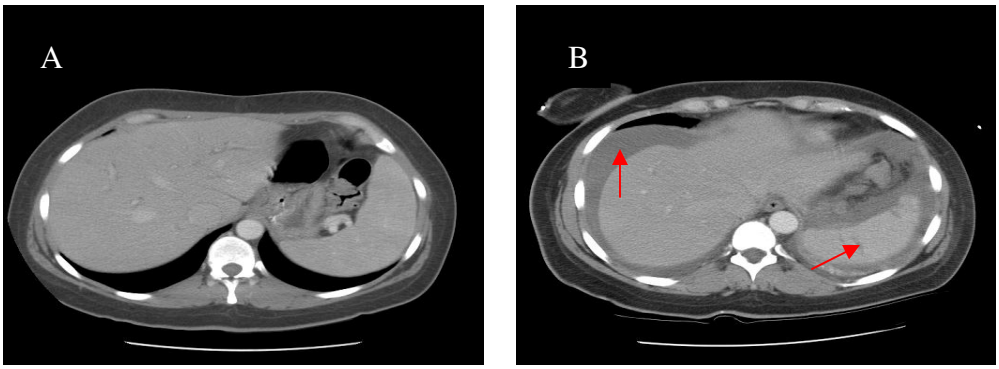

Figure 1. (A) CT scan of the abdomen upon admission without evidence of splenic rupture. (B) Repeat abdominal CT scan two days later revealed free intraperitoneal fluid, hypodensity in the spleen, and capsular changes consistent with splenic rupture (see arrows).

Discussion

Delayed splenic rupture can be explained by either a subtle splenic lesion which is not visualized and/or a false negative CT scan. Subtle splenic lesions that can progress to splenic rupture include subcapsular hematoma, pseudocyst, and pseudoaneurysm.[1] Sources of false negatives include: 1) artifact or interference from the surrounding tissues which make the injury difficult to detect, 2) an early CT scan taken before a subcapsular hematoma has bled enough to be detected on CT, 3) technical performance of the machine, 4) diluted oral contrast, and 5) variability in the intravenous contrast protocol used.[4]

This case exemplified splenic rupture as a true clinical entity. Contributing factors most likely included the recent decelerating motor vehicle accident and vomiting/ retching with abdominal adhesions from previous gastric bypass surgery which may have affected positioning and exerted traction on the spleen. Additionally, underlying SLE also may have contributed to DSR through pathologic changes within the spleen.

Of note, many cases of DSR occur along with underlying diseases such as end stage renal disease, amyloidosis[5], rheumatoid arthritis[6], chronic lymphocytic leukemia[7], and sarcoidosis[8]. Although not all of these studies reported a normal initial CT scan, these cases supported the hypothesis that certain co-morbid conditions can favor the occurrence of DSR by making the spleen more fragile[7] and the small splenic lesions more prone to progress later to frank splenic rupture.

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