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SPONTANEOUS SPLENIC RUPTURE AS AN UNCOMMON COMPLICATION OF ACUTE PANCREATITIS

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ABSTRACT

Background: Spontaneous splenic rupture (SSR) is a rare and potentially life-threatening complication of acute pancreatitis. *Case report:* We present the case of a 26-year-old female with a history of chronic alcohol use and a recent hospitalization for acute pancreatitis, who presented to the emergency department with severe abdominal pain. Contrast-enhanced computed tomography (CT) scan revealed necrotizing pancreatitis (Balthazar D) and a splenic collection. Following signs of hemodynamic instability and a critical drop in haemoglobin levels, an urgent exploratory laparotomy was performed. *Conclusion:* Splenic complications in the context of acute or chronic pancreatitis are uncommon. A high index of clinical suspicion combined with early diagnosis through CT scan or other imaging modalities is crucial to improving prognosis. Given the potential for rapid deterioration, prompt and appropriate intervention can significantly reduce the morbidity and mortality associated with splenic rupture.

KEYWORDS

Acute pancreatitis, spontaneous splenic rupture, exploratory laparotomy

LEARNING POINTS

- Spontaneous splenic rupture should be suspected in patients with acute pancreatitis who develop hemodynamic instability.
- Computed tomography scan is essential for early detection and assessment of splenic complications.
- Prompt surgical management can significantly improve outcomes in unstable patients.

INTRODUCTION

Spontaneous splenic rupture (SSR) is a rare and potentially life-threatening complication of acute pancreatitis. By 1945, only 7 cases of splenic rupture secondary to an episode of acute pancreatitis had been documented[1,2]. Splenic complications in pancreatitis are associated with high morbidity (79%) and significant mortality (8%)[3], highlighting the importance of rapid and timely recognition. Many of

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these complications—especially parenchymal ones—are diagnosed incidentally through abdominal computed tomography (CT) scan during the evaluation of pancreatitis progression[4]. The aetiopathogenic mechanism of SSR is not well understood and remains a subject of debate; any cause that leads to splenic enlargement or increased pressure within the splenic vasculature may favour rupture[5-7]. Ultimately, splenic rupture should be considered in the differential diagnosis of shock occurring during the course of acute pancreatitis.

CASE DESCRIPTION

A 26-year-old female with a history of chronic alcohol consumption and a previous hospitalization for acute pancreatitis two months earlier presented to the emergency department with postprandial abdominal pain with a belt-like distribution to both flanks, accompanied by nausea and vomiting of gastrointestinal contents, predominantly after eating.

Physical examination revealed tachycardia, tachypnoea, diaphoresis, and generalized pallor. Initial laboratory tests revealed the following: lipase 1704 U/l, haemoglobin 14.8 g/dl, leukocytes 19,260/mm^3, neutrophils 15,601/mm^3 (81%), and platelets 516,000/mm^3. Contrast-enhanced CT scan of the chest abdomen and pelvis *(Fig. 1, 2 and 3)* revealed a left pleural effusion, necrotizing pancreatitis classified as Balthazar D, a heterogeneous subcapsular splenic hematoma (32-60 HU), and free fluid in the perihepatic and perisplenic areas, the left pericolic sulcus, the pouch of Douglas, and the pelvic cavity, suggestive of hemoperitoneum (20-70 HU). Intensive fluid resuscitation and analgesia were initiated.

Follow-up studies showed a critical drop in haemoglobin to 4 g/dl, leukocytes 13,820/mm^3, and platelets 301,000/mm^3. A repeat CT scan *(Fig. 4 and 5)* showed an enlarged subcapsular splenic hematoma and free intra-abdominal fluid. Given these findings, an urgent exploratory laparotomy was performed, which revealed complete splenic rupture, hemoperitoneum (1200 cc), inflammatory plaques in the greater omentum, and necrosis of the pancreatic tail. Consequently, a total splenectomy was performed.

In the immediate postoperative period, the patient developed hypovolemic shock, requiring temporary norepinephrine support and red blood cell transfusions. Clinical progress was favourable, with oral intake initiated and close monitoring for7 days. Due to the patient's significant clinical improvement, the decision was made to discharge him from the hospital on the eighth day, with outpatient follow-up by Internal Medicine and General Surgery.

DISCUSSION

Splenic complications associated with pancreatitis include haemorrhages, abscesses, intrasplenic pseudocysts, infarctions, ruptures, and vascular injuries. Although infrequent, these conditions can severely compromise prognosis if not diagnosed and treated promptly[3].

Several pathogenic mechanisms have been proposed to explain splenic rupture in the context of pancreatitis. These include splenic vein thrombosis, formation of perisplenic adhesions, invasion or erosion of the splenic parenchyma by pancreatic pseudocysts, and acute inflammation of ectopic pancreatic tissue located within the spleen[5-7]. In the case described, splenic rupture was likely attributed to enzymatic activity released during an episode of acute pancreatitis.

Diagnosis of these complications is challenging due to the

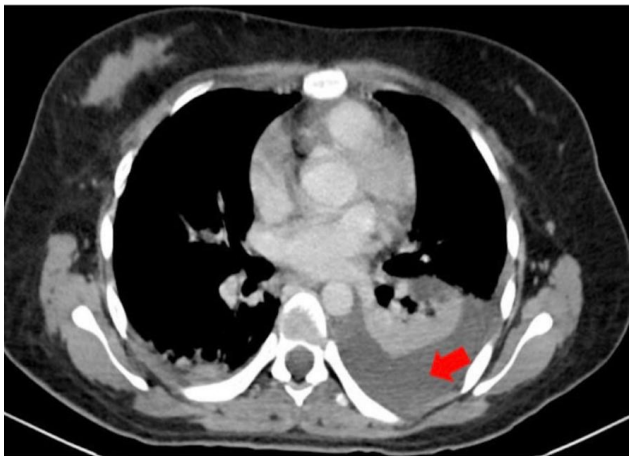


Figure 1. Non-contrast computed tomography scan of the chest, axial view: bilateral pleural effusion with left-side predominance, measuring 2.3 cm in thickness (red arrow).

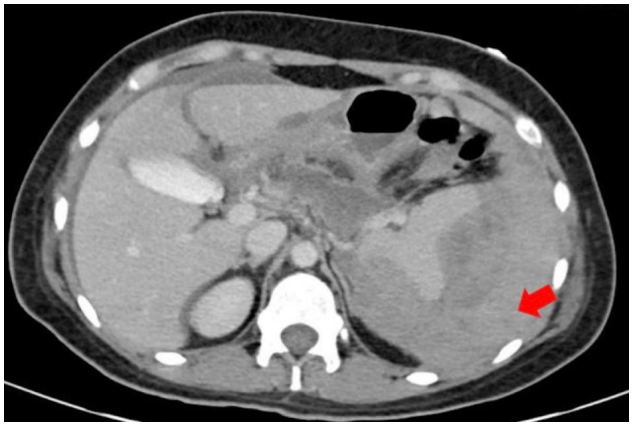


Figure 2. Contrast-enhanced computed tomography scan of the abdomen and pelvis, axial view: heterogeneous splenic collection consistent with a subcapsular hematoma (red arrow) (32–60 HU), along with perihepatic and perisplenic free fluid (red arrow).



Figure 3. Contrast-enhanced computed tomography scan, of the abdomen and pelvis, axial view: findings consistent with necrotizing pancreatitis, Balthazar grade D (red arrow).

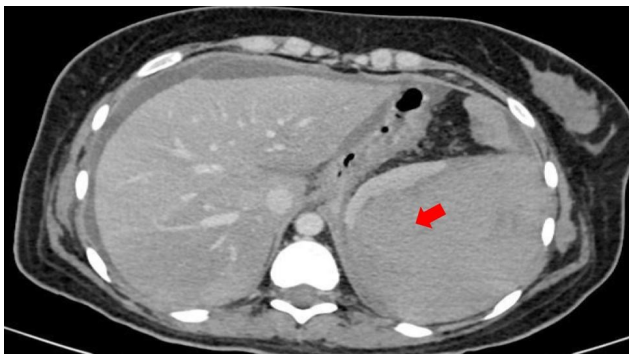


Figure 4. Contrast-enhanced computed tomography scan of the abdomen and pelvis, axial view: increased volume of the subcapsular splenic hematoma and greater amount of free fluid (red arrow).

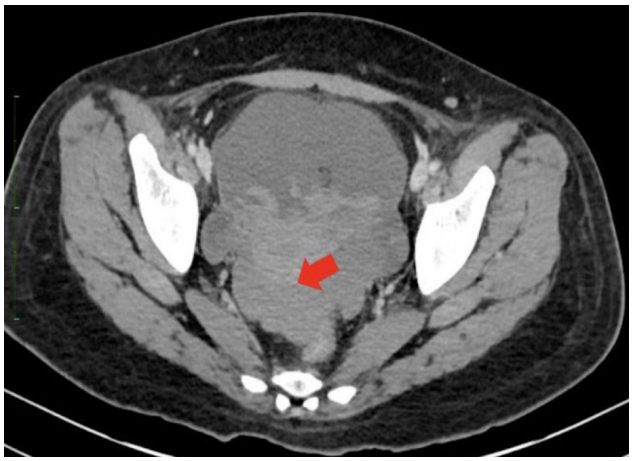


Figure 5. Contrast-enhanced computed tomography scan of the abdomen and pelvis, axial view: free fluid in the left paracolic gutter, pelvic cavity, and pouch of Douglas, suggestive of hemoperitoneum (red arrow) (20–70 HU).

nonspecific signs and symptoms. However, the presence of pain in the left upper abdominal quadrant, as well as referred pain to the left shoulder (Kehr's sign), should alert the clinician[8-9]. CT scan is the imaging modality of choice for identifying these complications and monitoring patient progress[4]. Additionally, worsening abdominal pain accompanied by a sudden drop in haemoglobin levels may be a key clinical indicator of splenic bleeding.

Renzulli et al. (2009)[2] systematically reviewed atraumatic splenic rupture, identifying causes such as neoplastic disorders (e.g., lymphoma), infectious diseases (e.g., infectious mononucleosis, malaria), haematological conditions (e.g., haemolytic anaemia), and inflammatory processes, including pancreatitis. In our case, acute pancreatitis was the likely trigger, potentially due to enzymatic erosion of the splenic parenchyma or vascular compromise, with no evidence of malignancy, infection, or haematological disorders.

The patient had no history of recent trauma, including blunt or penetrating abdominal injury, and no clinical or laboratory evidence of infections such as infectious mononucleosis or other infectious causes of splenic rupture. This was supported by clinical history and laboratory findings (e.g., absence of atypical lymphocytes or positive monospot test results) to rule out infectious mononucleosis.

Treatment fundamentally depends on the patient's hemodynamic stability. In hemodynamically stable individuals, conservative options such as close observation, image-guided percutaneous drainage, selective arterial embolization, or, in selected cases, surgery may be considered. Conversely, in unstable patients, urgent splenectomy may be necessary[4-6]. Approximately 12.5% of cases require emergency splenectomy due to clinical instability, as occurred in this case.

Although splenic complications in the context of acute or chronic pancreatitis are rare, high clinical suspicion combined with early diagnosis through CT scan or other imaging techniques is crucial to improving prognosis. Given that this condition can progress rapidly, early and appropriate intervention can significantly reduce the morbidity and mortality associated with splenic rupture.

CONCLUSION

SSR is a rare but severe complication of acute pancreatitis. Timely diagnosis and intervention are critical to patient survival.

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