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Case Report

Sudden abdominal pain in a patient with pneumonia reveals spontaneous splenic rupture in normal sized spleen ☆

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ABSTRACT

Splenic rupture is a life-threatening condition that is most frequently of traumatic etiology. Atraumatic or spontaneous splenic rupture is much rarer and less frequently reported. We present a case of an 84-year-old male patient initially hospitalized for pneumonia, who developed sudden abdominal pain and hemodynamic instability. Further investigations revealed a spontaneous splenic rupture. Histopathological examination postsplenectomy identified a splenic hamartoma, which had not been visualized on prior imaging studies.

Introduction

Traumatic splenic rupture is a well-documented and recognized complication in medical literature, commonly observed in approximately 30% of patients who undergo surgery for blunt abdominal trauma [1]. In contrast, spontaneous splenic rupture (SSR), although significantly rarer, represents a much more insidious and life-threatening condition that demands prompt recognition, stabilization, and definitive surgical intervention [2]. SSR can occur without preceding trauma or obvious underlying pathology, making it particularly challenging to diagnose and manage effectively [3]. The first documented case of spontaneous splenic rupture dates back to 1842 [4].

This case report describes an uncommon and complex instance of atraumatic splenic rupture in a patient hospitalized for pneumonia. The subsequent histopathological examination revealed an underlying splenic hamartoma, adding an additional layer of complexity to the case. The confluence of these conditions presents unique diagnostic and therapeutic challenges, underscoring the necessity for heightened clinical awareness and multidisciplinary management to ultimately improve patient outcomes.

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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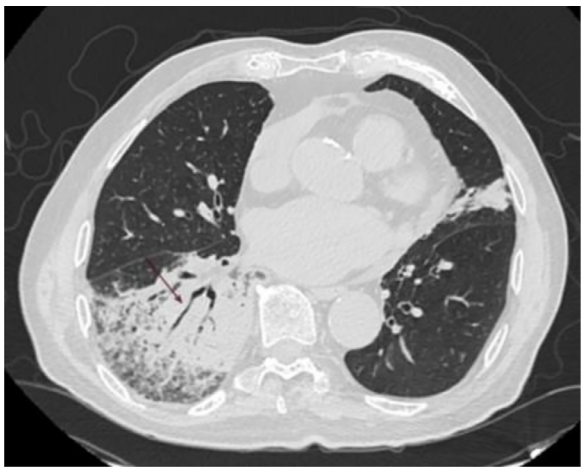

Fig. 1 – Right lower lobe consolidation. Arrow: air bronchogram.

Case presentation

An 84-year-old male with a medical history of cardiac failure secondary to hypertension, renal failure secondary to diabetic nephropathy, and oxygen-dependent respiratory failure presented to the emergency department with fever, productive cough, and dyspnea. A CT scan confirmed a diagnosis of right lower lobe pneumonia (Fig. 1), and the patient was admitted for intravenous antibiotics and supportive care.

On the ninth day of hospitalization, the patient experienced sudden onset of severe diffuse abdominal pain, accompanied by hypotension and tachycardia. Laboratory tests revealed a significant drop in hemoglobin from 13.1 g/dL to 8.8 g/dL in 24 hours. An urgent abdominal CTA showed active bleeding and hemoperitoneum, suggesting a splenic rupture (Figs. 4–6). The patient was stabilized with fluid resuscitation and blood transfusions, and an emergency laparotomy was performed. Intraoperatively, a ruptured spleen with active bleeding was identified, and a splenectomy was carried out.

Histopathological findings

Gross examination of the spleen revealed a spleen measuring 10 × 6 × 4 cm, weighing 154 g, and entirely covered by a smooth capsule. The cut section was congested with a 1 cm subcapsular hemorrhagic nodule associated with a disrupted capsule. Microscopically, the splenic architecture was intact with mild congestion of the red pulp. A 1 cm subcapsular nodule composed of irregular dilated vessels, haphazard cords, and sinuses with prominent extramedullary hematopoiesis, including numerous megakaryocytes, was consistent with a splenic hamartoma. The overlying capsule was ruptured with hemorrhage and fibrin deposition. Immunohistochemistry showed an appropriate distribution of CD5-positive T cells and CD20-positive B cells. Glycophorin and MPO highlighted erythroid and granulocyte precursors, respectively. No malignancy or infectious organisms were detected.

Discussion

SSR is rare but potentially fatal, with an unclear overall incidence. It appears to have a male predominance of 2:1 and a median age of occurrence of 45 years. Etiologies include neoplasia (30%), infectious (27%), inflammatory (20%), treatment-related (9%), mechanical (7%), and idiopathic causes. Notably, in about 9% of cases, there are 2 or more etiologicies [5].

The exact pathological mechanisms of SSR are multifactorial, involving anatomical, hematological, and immunological factors. Three main mechanisms contribute to splenic rupture: increased intrasplenic tension from cellular hyperplasia and engorgement, compression by abdominal muscles during activities like sneezing or coughing, and vascular occlusion due to reticular endothelial hyperplasia leading to thrombosis and infarction. These factors cause interstitial and subcapsular hemorrhage, eventually leading to the rupture of the distended capsule [6].

SSR secondary to cough and/or pneumonia is particularly rare, with only a few reported cases since 1980. Identified pathogens include Streptococcus pneumoniae, hemophilus influenzae, Q fever, and Legionella [5].

In this context, the patient's pneumonia likely contributed to splenic vulnerability, although the exact pathophysiological mechanisms remain unclear. Severe pneumonia can induce systemic inflammatory responses that affect the spleen, leading to splenomegaly and increased blood flow, both heightening the risk of capsular rupture [5]. Although the CT scan during the pneumonia presentation showed a normal-sized spleen (Figs. 2–3), the severe inflammatory milieu likely exacerbated the intrinsic fragility of the spleen due to the underlying hamartoma.

Splenic hamartomas are rare benign lesions composed of disorganized splenic tissue. Typically asymptomatic and often incidental findings, they can occasionally cause complica-

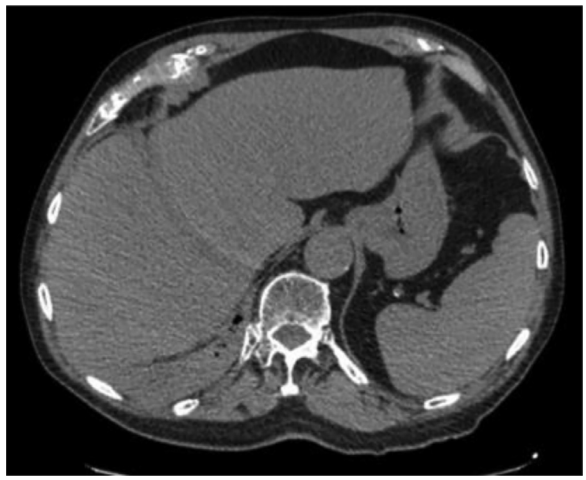

Fig. 2 – Homogeneous appearance of the normal-sized spleen with intact capsule 9 day prior to episode.

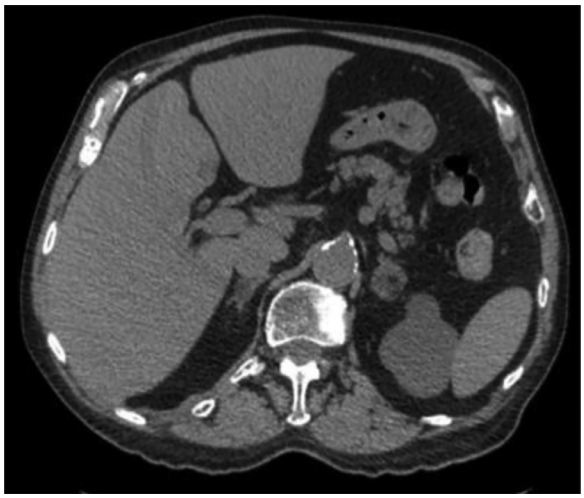

Fig. 3 – Homogeneous appearance of the posteroinferior pole of the spleen with intact capsule 9 day prior to episode.

tions like hypersplenism, cytopenias, or SSR [7]. Only few cases have been reported to date, the youngest being a 5-month-old boy, which led to the death of the child [8]. The hamartoma in this patient may have contributed to the rupture by creating a localized area of structural weakness within the splenic parenchyma.

Patients with SSR commonly present with abdominal pain, referred shoulder pain (Kehr's sign), and signs of hypovolemic shock, though chest pain is also possible. These symptoms can overlap with other abdominal pathologies, complicating diagnosis. In this case, the patient's sudden abdominal pain and hemodynamic instability warranted urgent diagnostic workup. Blood tests should include a full blood count, urea, electrolytes, creatinine, liver function tests, crossmatch, and coagulation studies. Diagnosis can be made via CT in hemodynamically stable patients or identified through bedside ultrasonography or intraoperatively [9].

Imaging modalities such as ultrasound and CT scans are crucial for prompt SSR diagnosis. In this patient, an emergent abdominal CT scan revealed hemoperitoneum and splenic rupture, necessitating immediate surgical intervention. Imaging differentiates SSR from other acute abdominal causes, impacting clinical outcomes directly.

Management of SSR involves initial stabilization with fluid resuscitation and blood transfusions, followed by definitive surgical intervention. Splenectomy remains the standard treatment, especially when the spleen is extensively damaged or the patient is hemodynamically unstable. Nonoperative management has a high failure rate, and total splenectomy is often also recommended for SSR of unknown etiology, even if all preconditions for nonoperative management are met [2].

In this patient, an emergent splenectomy was performed, controlling the hemorrhage and allowing histopathological examination. Postoperative analysis confirmed a small hamartoma, likely contributing to the rupture. This highlights the importance of thorough pathological examination in SSR cases to provide insights into the etiology and guide follow-up care, especially because a significant number of malignant disease may cause SSR.

This case emphasizes important clinical considerations. Clinicians should include SSR in their differential diagnosis for patients with acute abdominal pain and hemodynamic instability, especially when systemic infections like pneumonia are present. A multidisciplinary approach involving emergency physicians, radiologists, surgeons, and pathologists is essential. Early recognition and intervention are vital, and understanding the link between infections, splenic anomalies, and rupture risk aids clinical decisions. Lastly, "spontaneous splenic rupture" is misleading, as atraumatic ruptures usually occur in a diseased spleen or due to a pathological process. A pathological cause should always be investigated [10].

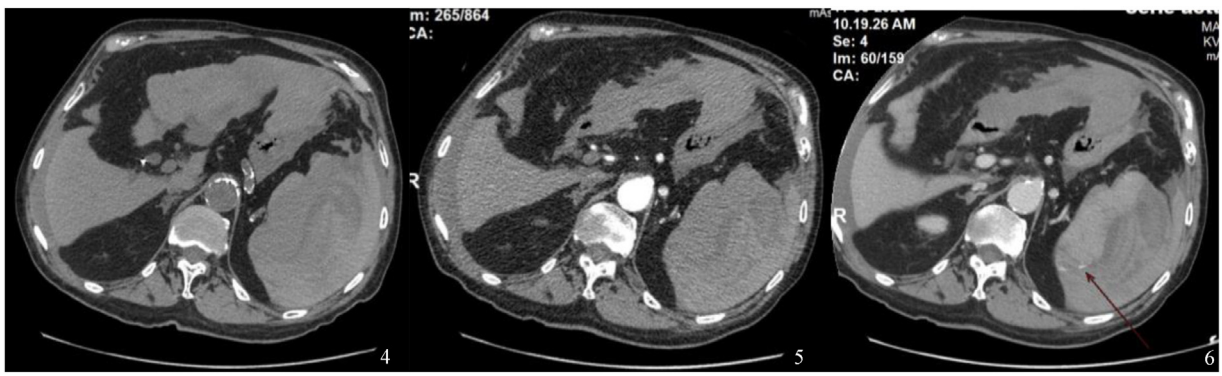

Fig. 4, 5 and 6 – Appearance of a large, heterogeneous, dense subcapsular splenic hematoma with contrast blush (arrow in Fig. 6). The splenic parenchyma is compressed and heterogeneous, with several nonenhanced hypodense areas. Appearance of moderately abundant abdomino-pelvic ascites with spontaneously hyperdense hemorrhagic components reaching the perihepatic area (Figs. 4 and 5). Appearance compatible with splenic rupture. No rib fracture within the limits of the sections. No pneumoperitoneum.

Conclusion

In our case, the splenic rupture could not have been anticipated or prevented. The patient was fortunate to experience the rupture as an inpatient, allowing for prompt diagnosis and emergency surgery. Following local guidelines regarding immunizations and antibiotic prophylaxis postsplenectomy is crucial for preventing future complications.

Patient consent

The authors confirm that a written informed consent was obtained from the patient for the publication of this case report and accompanying images.

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