Atraumatic splenic rupture was attributed to intra-cystic haemorrhage and hypersplenism in a patient with cirrhosis and portal hypertension: A case report

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Abstract
Liver cirrhosis with splenomegaly and portal hypertension has been described in the medical literature as increasing the risk of splenic rupture. We report a case of atraumatic splenic rupture in a male with liver cirrhosis associated with splenomegaly, which was further complicated by hypersplenism and intra-cystic haemorrhage in the spleen. The 56-year-old man was hospitalized because of sudden onset of intermittent pain in the left quadrant abdomen with no history of trauma. Upon admission, the patient presented no fever, palpable abdominal tenderness, splenomegaly, and hypersplenism. Enhanced computed tomography revealed a splenic subcapsular haematoma connected to a cystic mass located at the splenic hilum and free fluid in the abdomen, which is indicative of splenic rupture. The patient underwent immediate laparotomy and splenectomy followed by proper management. Post-surgery diagnostic microscopy indicated liver steatosis, perivenular fibrosis, and regenerative nodules, which are suggestive of cirrhosis. The patient was discharged from the hospital with an uneventful recovery.

Keywords
Atraumatic splenic rupture, cirrhosis, splenomegaly, hypersplenism, intra-cystic haemorrhage

Case presentation
A 56-year-old male was referred to our emergency room with sudden onset of intermittent abdominal pain in the left quadrant of the abdomen. The patient had a history of liver cirrhosis and splenomegaly. Upon admission, the patient presented with no fever, palpable abdominal tenderness, splenomegaly, and hypersplenism. Enhanced computed tomography revealed a splenic subcapsular haematoma connected to a cystic mass located at the splenic hilum and free fluid in the abdomen, which is indicative of splenic rupture. The patient underwent immediate laparotomy and splenectomy followed by proper management. Post-surgery diagnostic microscopy indicated liver steatosis, perivenular fibrosis, and regenerative nodules, which are suggestive of cirrhosis. The patient was discharged from the hospital with an uneventful recovery.
upper quadrant. The pain started 5 days prior with no obvious traumatic cause. The patient had no clinical history of hepatitis, hypertension, coronary heart disease, diabetes, or any type of surgery, but he had a long-term history of excessive alcohol consumption with an average of 100 g of ethanol per day. During physical examination upon admission, he presented no fever with a body temperature of 36.2°C, a pulse rate of 80/min, and blood pressure of 170/90 mmHg. Abdominal palpation revealed tenderness in the left upper quadrant but no rigidity or spasm. The spleen was palpable at 10 cm below the left coastal arch. The result of the abdominal shifting dullness exam was negative. Diagnostic paracentesis yielded nonclotting blood.

The patient’s laboratory tests showed a white blood cell (WBC) count of $2.03 \times 10^9/L$, red blood cell (RBC) count of $3.46 \times 10^9/L$, haemoglobin level (Hb) of $109 g/L$, and platelet count of $71 \times 10^9/L$, which are indicative of hypersplenism. Enhanced computed tomography revealed splenic subcapsular attenuation of echogenicity connected to a hypoechoic cystic mass (~3 cm) located at the splenic hilum, as well as free fluid in the abdomen, which are suggestive of intra-cystic haemorrhage–induced splenic rupture and haemoperitoneum (Figure 1).

An exploratory laparotomy was performed due to the suspicion of splenic rupture and haemoperitoneum. During the surgery, ~2300 mL of fluid mixed with blood clots was collected from the peritoneum. The liver exhibited an irregular or nodular surface, blunt edges, and medium hard texture, which are suggestive of liver cirrhosis. The enlarged spleen ($35 \times 28 \text{ cm}$) presented subcapsular haematoma (>40% of the surface area) and laceration (~4 cm in depth). The intraoperative portal venous pressure was measured as $35 \text{ cm H}_2\text{O}$. The ruptured spleen was removed, and a liver biopsy ($5 \text{ mm} \times 5 \text{ mm}$) was obtained for histopathological purposes. During the splenectomy, the patient received a blood transfusion containing 800 mL of packed RBCs, 300 mL of plasma, and 1 unit of platelets.

The patient had an uneventful recovery and was discharged at 10 days post-surgery. Upon discharge, his laboratory test exhibited favourable outcomes (WBC $7.11 \times 10^9/L$, RBC $4.87 \times 10^9/L$, Hb $148 g/L$, and platelets $424 \times 10^9/L$). Post-surgery microscopy of the liver revealed macrovesicular steatosis, perivenular fibrosis, and fibrous bands subdividing the liver into regenerative nodules, which is indicative of cirrhosis (Figure 2(a) and (b)). The spleen presented intra-cystic haemorrhage and a cyst covered with an endothelial layer, but there was no calcification or fibrosis (Figure 2(c)). There was also congestion and dilation of the sinusoids, active germinal centres, and increased numbers of macrophages in the spleen pulp (Figure 2(d)).

**Discussion**

The majority of atraumatic splenic ruptures (over 90%) are pathologic, which is generally considered to be subsequent to pathological changes in the spleen. A few cases of splenic rupture were previously reported in patients with hepatitis-related liver cirrhosis and portal hypertension, including one rare case of spontaneous rupture of splenic hamartoma which was not encapsulated and composed of sinusoidal spaces and therefore was mostly affected by portal hypertension. In this case, it was apparent that the patient presented portal hypertension and severe splenomegaly due to alcoholic cirrhosis, and the laboratory test upon admission suggested typical hypersplenism. Post-surgery diagnostic microscopy also revealed active germinal centres and congestive changes in the splenic parenchyma. Moreover, computed tomography revealed subcapsular haematoma connected to intra-cystic haemorrhage. In general, nonparasitic cysts of the spleen are rare but usually benign, and non-traumatic haemorrhage in those cysts are even uncommon when the sizes are small. Surgical management has also been recommended for splenic
cysts with diameter larger than 4–5 cm, because they increase the risk of complications.\textsuperscript{14,15}

This case was complicated by a few unfavourable conditions that potentially increased the risk of splenic rupture. The first is the increased intra-splenic tension due to cellular hyperplasia and engorgement in splenomegaly, which may lead to splenic rupture. The spleen may be compressed by forceful diaphragmatic contraction or abdominal musculature during physiological activities, such as sneezing, coughing, and defaecation. Second, liver cirrhosis generates portal hypertension, which may give rise to splenic congestion and vascular occlusion caused by reticular endothelial hyperplasia, which in turn results in thrombosis and infarction. This is often observed in patients with splenomegaly and is involved in interstitial and subcapsular haemorrhage, as well as further stripping of the capsule. The distended capsule finally gives way and causes splenic rupture. Third, enhanced computed tomography revealed intra-cystic haemorrhage in the spleen, which may lead to subcapsular haematoma. Splenomegaly caused by severe portal hypertension is common in other patients with cirrhosis, who normally exhibit severe thrombocytopenia and leukopenia. This could worsen subcapsular haemorrhage due to coagulation dysfunction, and expanding subcapsular haematoma eventually tears the capsule. Notably, the cystic mass was located at the splenic hilum, where it increased the risk of haemorrhage due to portal venous hypertension. We suspect this to be the most direct cause of splenic rupture in our case.

Conclusion

Emergency splenectomy is required for splenic rupture. Recently, ultrasonography and computed tomography have helped improve the discovery of incidental splenic cysts. Operative indications for splenic cysts should include not only the possibility of malignant disease but also the risk of splenic rupture under certain circumstances, even when the cyst is small. We consider the pre-existing liver cirrhosis, portal hypertension, and hypersplenism to be risk factors for intra-cystic haemorrhage that eventually lead to splenic rupture.

Declaration of conflicting interests

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Ethical approval

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