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CASE REPORT

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Non-traumatic liver rupture due to a perforated gastric ulcer

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Abstract The case of a 57-year-old woman with a fatal liver rupture due to a necrotizing perihepatic abscess caused by a perforated gastric ulcer is presented. The ulcer had been treated successfully by surgical intervention 8 days before. The autopsy revealed a large perihepatic abscess and multiple ruptures of Glisson's capsule with a large subcapsular hematoma and underlying lacerations of the liver parenchyma. The patient had no history of previous abdominal trauma and the known etiological factors for spontaneous liver rupture were excluded by the autopsy findings or by clinical and laboratory data. No liver penetration by the gastric ulcer was found at autopsy and there were no clinical signs or symptoms for an infection or any degenerative or inflammatory diseases. Histologically abundant vegetable fibers, identified as stomach contents and a dense infiltrate of lymphocytes and granulocytes were found in the perihepatic abscess next to Glisson's capsule. Below Glisson's capsule there were hemorrhages, focal hepatocellular necrosis and a mixed cell inflammatory infiltration. In the present case, preceding perforation of the gastric ulcer with leaking of gastric acid into the peritoneal cavity resulted in peptic digestion of Glisson's capsule. Vascular lesions of the affected parts of Glisson's capsule and the liver parenchyma underneath resulted in intrahepatic hemorrhage and an increase in intrahepatic pressure with subsequent liver rupture. To the authors' knowledge no similar case of spontaneous liver rupture due to perforation of a gastric ulcer has been reported previously.

Key words Liver rupture · Non-traumatic gastric ulcer · Perforation · Complication · Perihepatic abscess

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Introduction

Spontaneous, non-traumatic liver rupture is a rare and uncommon event, mainly seen in patients with malignant or benign liver tumours [3, 8, 13, 15, 19], hepatic amyloidosis [2, 14], peliosis hepatis [1, 18], pre-eclampsia and eclampsia of pregnancy [11, 17]. Despite immediate surgical treatment the prognosis of hepatic rupture remains grave and the mortality rate is high [4, 10, 15]. Here we report a case of spontaneous, non-traumatic liver rupture due to perforation of a peptic ulcer.

Case report

A 57-year-old woman was admitted to hospital with abdominal pain in the right upper quadrant, nausea and vomiting, but with no signs of gastrointestinal bleeding. Chest radiography, electrocardiography and ultrasonography of the abdomen showed no anomalies. Laboratory data and hematological investigations were also within normal limits. On arrival, the patient denied consent to any

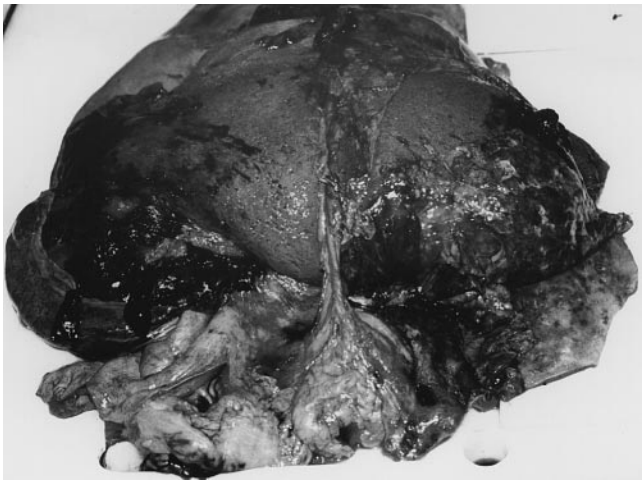

Fig. 1 Multiple ruptures of Glisson's capsule, lifted off the surface of the liver with subcapsular hemorrhages and parenchymal lacerations. Right: perihepatic abscess

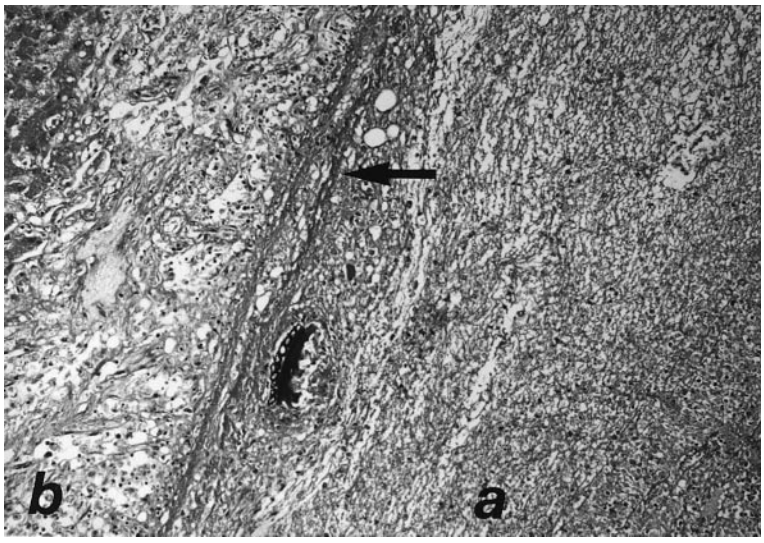

Fig. 2 Stomach content in the perihepatic abscess (**a**) next to Glisson's capsule (*arrow*). Subcapsular liver parenchyma (**b**) (PAS × 150)

invasive diagnostic test but agreed to computer tomography of the abdomen which revealed a perforated gastric ulcer. As the pain in the right upper quadrant intensified and abdominal signs progressed to generalized peritonism 24 h after admission, consent was finally obtained for an emergency laparotomy. Laparotomy revealed more than 1 L of muddy exudate and a perforated gastric ulcer 0.8 cm in diameter, located prepyloric at the lesser curvature. The ulcer was excised and sutured. There was no evidence of adhesions inside the peritoneal cavity and no fibrin structures or any other signs of peritonitis were found. A benign gastric ulcer was identified by a pathology examination of the specimen. Postoperatively, the patient was admitted to the intensive care unit, where she continued to require supportive ventilation for the following 7 days without regaining consciousness. On the eighth day after admission, the patient developed hypotension. Hematological investigations revealed a hemoglobin level of 9.2 g/dl, a hematocrit level of 31% and a white blood cell count of 20.3×10^9/L. A further emergency laparotomy was performed, revealing several liters of blood in the peritoneal cavity with sequestra of liver parenchyma within. The origins of the bleeding were parenchymal ruptures on the surface of the right lobe and on the bottom side of the left lobe of the liver. No suture insufficiency or any hints for active bleeding was found in the area of the former ulcer resection. Careful examination failed to reveal the cause of liver rupture. The hemorrhage was uncontrolled, even after ligation of the hepatic artery and finally the abdomen was closed with packs in situ. A transfusion of nine units of red cell concentrate was given, but despite intensive care the patient's condition failed to improve sufficiently for further surgical treatment with progressive insufficiency of hepatic and renal function. Death occurred from multiple organ failure a few hours after surgery.

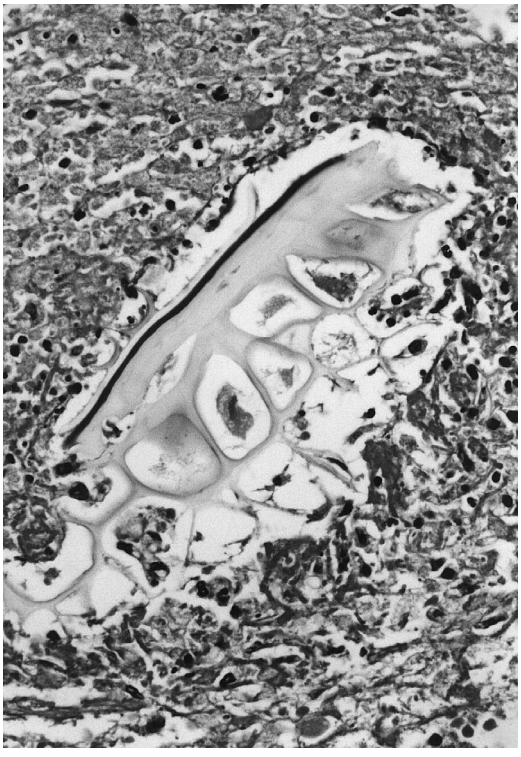

Fig. 3 Higher power view of a stomach content in the perihepatic abscess with infiltration of inflammatory cells (PAS × 235)

Autopsy findings

The enlarged liver (weight 2450 g) was surrounded by a large perihepatic abscess with adhesions to Glisson's capsule. In addition, a subphrenic abscess was found adjacent to the surface of the right lobe of the liver. Glisson's capsule showed multiple ruptures and was raised from the surface of the liver with a large subcapsular hematoma. On the right lobe of the liver underneath the ruptures of Glisson's capsule, multiple parenchymal lacerations were found up to 3.0 cm in depth (Fig. 1). Cross-sections of the liver parenchyma revealed multiple hemorrhagic foci ranging from 0.2 to 3.5 cm in diameter with pale yellow lesions in the marginal regions. There was no portal vein obstruction and the major hepatic veins were free of thrombi. Examination of the stomach revealed a regular operation field without perforation or active bleeding and without suture insufficiency. There were neither signs of a persistent or recurrent gastric ulcer in the gastric walls nor any hints for generalized peritonitis. Apart from bilateral pleural effusions (600 ml right, 300 ml left) and congestion of the lungs (1460 g combined weight), no other macroscopic abnormalities could be found.

Histological findings

Histological examination of the perihepatic abscess next to Glisson's capsule showed extensive necrosis with focal areas of granulomatous inflammation consisting of cellulose-containing particles that were identified as stomach contents (Figs. 2, 3). Besides a dense infiltrate of lymphocytes, granulocytes, foam cells and a distinct fibrin network (PTAH staining), fibroplasia but no mature fibrous tissue (van Gieson staining) could be detected in the abscess. Underneath Glisson's capsule, edematous liver parenchyma with

focal hepatocellular necrosis and microvesicular fatty transformation of hepatocytes, hemorrhages, an extended fibrin network and a mixed-cell inflammatory infiltration, predominately granulocytes were found. In the marginal regions of the hemorrhagic foci, attenuated and dissociated liver cell cords due to varying degrees of hepatocellular degeneration, sinusoidal dilatation, bile stasis and a sharp granulocytic demarcation of the hemorrhagic foci were detected. However, no evidence of fibrosis could be demonstrated. The undegenerated liver parenchyma was found to be congested and hyperemic and hepatic vein thrombosis was not present.

Discussion

The etiology of spontaneous, non-traumatic liver rupture is difficult to diagnose in vivo. Opportune life-saving surgery often fails, therefore, in most cases a definitive diagnosis can only be established through postmortem examination [2, 6, 8, 9].

The patient had no history of previous abdominal trauma other than the recent laparotomy and none of the known etiological factors for spontaneous liver rupture such as anabolic agents, oral contraceptives or pregnancy [7, 9, 11] could be demonstrated. Carcinomatosis of Glisson's capsule or the presence of an underlying liver tumour was exluded by macroscopic and histological examination. Congo red stain showed no amyloidosis. Periodic acid-Schiff (PAS) staining eliminated the possibility of the presence of a fungus. Peliosis hepatis was ruled out histologically by the lack of cystic lesions and spaces and no fibrosis could be found. There was no evidence of portal vein obstruction or hepatic vein thrombosis at autopsy and there were neither histological nor clinical signs for an infection or any previously existing degenerative or inflammatory diseases. Disseminated intravascular coagulopathy as a rare cause of spontaneous liver rupture [6] was excluded by laboratory data.

Although gastric ulcers may penetrate and perforate virtually every organ in close proximity to the stomach [5, 12, 16, 20], in this case, a liver penetration by the gastric ulcer was excluded by laparoscopy and autopsy findings. In the present case, an open perforation of the gastric ulcer with gastric acid leaking into the peritoneal cavity resulted in peptic digestion of Glisson's capsule. This resulted in development of a perhepatic abscess with subsequent necrosis, edema and vascular lesions of the affected parts of Glisson's capsule and the liver parenchyma underneath with an increase in intrahepatic pressure causing venous obstruction. The combination of a normal arterial inflow and a poor venous outflow, causing intrahepatic congestion and further edema with following intrahepatic hermorrhage via rhexis, resulted in an enlargement of the liver with final rupture of Glisson's capsule and hemoperitoneum. The histologically detectable abundant stomach contents in the perihepatic abscess next to Glisson's capsule proved that the perforated gastric ulcer was the cause of the liver rupture. The presence of lymphocytes, granulocytes, foam cells, patchy infiltrates of giant cells around the stomach contents and fresh granulation tissue with fibroplasia but no development of mature fibrous tissue in the perihepatic abscess is consistent with a time span of 8 days since gastric ulcer perforation. The sharp granulocytic demarcation of the hemorrhagic foci and the varying degrees of hepatocellular degeneration reflect the rapid parenchymal collapse following hemorrhages through rhexis. These degenerations and inflammatory changes correspond to a survival time of approximately 12 to 24 h after the onset of intrahepatic bleeding, thus excluding the possibility of injury caused by laparotomy, e.g. by a surgical retractor, one week before.

To our knowledge, this is the first report of a spontaneous, non-traumatic liver rupture due to the perforation of a gastric ulcer. It illustrates, that in extremely rare instances, gastric acid originating from a perforated gastric ulcer can lead to liver rupture via digestion of Glisson's capsule, resulting in intrahepatic hemorrhage and sufficient rise of intrahepatic pressure with subsequent liver rupture.

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