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Case report

IDIOPATIC LIVER RUPTURE: AN ITALIAN CASE REPORT

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ABSTRACT

The liver can be damaged from an impact (such as in a car accident) or from penetrating trauma (such as a stab or gunshot wound). Liver lesions range from relatively small collections of blood (hematomas) to large deep lacerations. Since the liver is supplied with many large blood vessels, the main problem following a liver injury is severe bleeding. Almost all bleeding from a liver injury occurs in the abdominal cavity. Spontaneous rupture of the liver is a rare occurrence. This is often associated with underlying pathological conditions (pregnant women with HELLP syndrome, liver pathologies such as adenoma, hepatic lymphoma, hepatocellular carcinoma HCC, macronodular cirrhosis, hemangioma, metastatic tumors and peliosis hepatis) or following traumatic insults. The authors report a rare case of spontaneous rupture of the liver that occurred in a 72-year-old man without underlying pathologies predisposing this condition, in the absence of evident traumatic lesions in the abdominal area and with a near- negative pathological history of trauma (falls, road accidents, etc.).

1. Introduction

Non-traumatic liver rupture is a rare but possible occurrence. However, this condition is associated with high morbidity and mortality [1] because of the difficulty in diagnosis. Pregnant women with HELLP syndrome are more prone to hepatic rupture, but it can also occur with liver pathologies [2] (adenoma, hepatic lymphoma, hepatocellular carcinoma HCC, macronodular cirrhosis, hemangioma, metastatic tumors and peliosis hepatis) [3,4] and, very rarely, in isolation. The condition has a worldwide incidence between 2.3% ad 26% and a mortality rate of 25% to 100% [3]. Here we report a rare case of severe hemorrhagic shock following spontaneous liver rupture subjected to autopsy.

2. Case report

A 72-year-old man was admitted into the emergency room with sudden-onset abdominal pain.

On physical examination the patient had a heart rate of 80 per minute, a blood pressure of 133/87 mmHg and 99% oxygen saturation. He complained of tenderness in the left upper quadrant. Laboratory results showed a hemoglobin of 13.10 g/dl, white blood cell count of 12700/uL, a total bilirubin level of 0.90 mg/dl, a direct bilirubin level of 0.90 mg/dl, an alanin transaminase level of 36 U/L, and an aspartate aminotransferase level of 24 U/L. The ultrasound of the upper abdomen revealed nothing of significance, and he was discharged with a diagnosis of alithiasic cholecystitis. Three days later, the patient presented with abdominal pain and fever (39°C), investigation revealed a slight decrease in hemoglobin levels (12,30 g/dl) and white blood cell count (10600/uL) with a contemporary increase of total bilirubin level (2,40 mg/dl), alanin transaminase level (83 U/L) and aspartate aminotransferase level (116 U/L). The consultation of the general surgeon concluded for acute cholecystitis with indication for hospitalization. The clinical condition continued to deteriorate, and his blood pressure declined gradually in the hours following admission to values of 80/60 mmHg, emergency blood tests revealed hemoglobin values of 9.0 g/dl.

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The patient underwent blood transfusions and an abdominal TC scan with contrast medium was performed showing intraparenchymal and subglissian collections with blood content, thickened walls as from hemorrhagic abscesses. In addition, the patient had an exponential growth of liver enzymes: an alanin transaminase level of 2008 U/L, and an aspartate aminotransferase level of 3460 U/L. Further haemodynamic instability (blood pressure of 60/40 mmHg) and fall in haemoglobin (5.90g/dl) forced the health workers to emergency surgery as the patient was in hypovolemic shock. Laparotomy showed that the right hepatic lobe had a deep laceration with copious hemorrhage between VII and VIII hepatic segments. This was sutured. A few hours after the operation the patient died of hemorrhagic shock from intra-hepatic bleeding. The autopsy allowed to recognize liver abundantly covered with hemostatic material; the sickle cell, coronary and right triangular ligaments are dissected, points of haemostasis at the level of the inferior vena cava; the right hepatic vein offers consideration of a laceration and a suture with a blue thread; portal vein containing abundant blood material. The hepatic segments 4-5-6-7-8 have no Glisson's covering and covered with haemostatic material with evidence of laceration on the posterior hepatic surface (segments 7-8) in which 4 metal clips and 2 sutures are highlighted. In the context of the same lesion, more in depth, a further suture point is found. External body examination and autopsy rule out trauma injuries. The patient died of a severe haemorrhagic shock, resulting from liver lesions of natural pathological origin. Significant impacts (e.g., a car accident) can damage the liver, as can penetrating trauma (e.g., knife wound, gunshot wound). Liver lesions range from subcapsular hematomas, to small capsular lacerations, to deep parenchymal lacerations, to crush injuries and vascular avulsion. The main immediate consequence is bleeding. The bleeding can be small or severe, depending on the nature and degree of the injury. Many small tears, particularly in children, stop bleeding spontaneously [5]. Large lesions bleed extensively, often causing hemorrhagic shock. Mortality is significant in high-grade liver injury. Manifestations of severe abdominal haemorrhage, including haemorrhagic shock, abdominal pain, tenderness, and distention are usually clinically evident [6].

As first described by Abercrombie in 1844 as a complication of pregnancy, spontaneous hepatic rupture is a rare event [7]. Spontaneous liver rupture may also occur in association with hepatic malignancies and hemangiomas. The condition may also be related to coagulation anomalies and hypertensive disorders [8]. Hepatic rupture rarely occurs as an idiopathic occurrence.

Spontaneous liver rupture affects the right side in 75% of cases, the left side in 11% and both sides in 14% of cases [9]. In the case presented, the patient had no underlying hepatic pathologies and the external examination of the corpse did not show the presence of traumatic lesions in the abdominal region (ecchymosis, excoriations). During the autopsy investigation segments 7 and 8 of the right hepatic lobe were involved.

The diagnostic phase of this condition is often difficult. Suspicion of liver rupture can be raised by history, physical examination, and basic laboratory tests. [10]. Hepatic injury is associated with a significative distortion of many important metabolic functions and it can be studied by determining serum concentration of some analytes such as alanine aminotransferase (AST), aspartate aminotransferase (ALT), alkaline phospatase (ALP), bilirubin and albumine [11].

Instrumental tests such as ultrasound or CT scan can provide confirmation to the suspected diagnosis.

Finally, angiography can be useful so that it can be both diagnostic and therapeutic.

The patient in our case was characterized at the first admittance for normal hemoglobin and AST/ALT values, however with high levels of direct bilirubin. Any form of obstruction of the bile ducts - complete or partial - can lead to an increase in conjugate bilirubin values [12]. Based on a negative ultrasound, the patient was then discharged with a diagnosis of alitiasic cholecystitis.

In the following three days the laboratory pattern was characterized by an increase in the values of direct bilirubin and AST / ALT being the results of the abdominal CT suggestive for intraparenchymal blood collections so that, due to a hypovolemic shock, the patient underwent surgery.

This clinical presentation is in line with what is suggested by the literature: generally, subcapsular haemorrhage precedes idiopathic liver rupture. Henny et al described a biphasic presentation of the condition distinguishing a pre-acute phase and an acute phase [13]

The pre- acute phase - starting about one month before the liver rupture - is characterized by the appearance of general abdominal pain and malaise. The acute phase, in relation to the stretching of hepatic capsule, is defined by a more intense pain and a vascular collapse. The liver receives blood from two main areas: the hepatic artery and the portal vein. The two vessels carry about 1.5 liters of blood per minute to the liver, nearly 75% of which comes from the portal vein. Compared to the rest of the body, liver has a significant amount of blood flowing through it - it is estimated that 13% of the body's blood is in the liver at any given time.

Including the evolution of the clinical presentation, the etiology of the spontaneous hepatic rupture remains difficult to diagnose. In this case, we were unable to find underlying liver pathology or inflammatory processes that could have led to hepatic rupture, nor any external injuries ascribable to traumatic injuries (bruises) being negative for the next anemnesis due to traumas (falls, accidents etc.). This proves that spontaneous liver rupture should be considered, not just in patients with known predisposing factors.

The treatment of this unusual condition depends on the aetiology. Tamponade of haemorrhage is the preferred method in cases of diffuse losses, in cases of ruptures and focal bleeding embolization and / or resection are preferred [8].

3. Discussion

Spontaneous hepatic rupture not correlated to pregnancy, to the presence of a liver tumor or traumatic injuries remains a rare event, requiring a multidisciplinary approach consisting in clinical monitoring, angiographic approach and possibly surgical intervention. The diagnosis of the condition is not immediate and can be made on the basis of clinical and instrumental examination. Laboratory tests can be useful too, however, the etiology of the spontaneous hepatic rupture appears difficult to define in most cases. A thorough medical history and a correct examination of the patient could highlight rare predisposing conditions such as, for example, amyloidosis.

In our case, we were unable to identify the real cause of liver bleeding. The decision is debatable [19]. If the perforation of a Jejunoileal causes only localized peritonitis and the patient remains stable, medical management with intravenous antibiotics, bowel rest and percutaneous CT-guided collection aspiration may be suitable and avoids the need for surgery [15]. For patients with peritonitis not responsive to non-surgical treatment, and for those with generalized peritonitis, the treatment of choice is prompt laparotomy with intestinal resection and primary anastomosis [16]. Resection may have to be limited to include only the segment containing the perforated diverticulum in order to prevent recurrences and short bowel syndrome [3]. Diagnostic laparoscopy can result in avoiding unnecessary laparotomies.

In the end, the period between clinical presentation and diagnosis seems to be the biggest determinant of prognosis [17].

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