SPONTANEOUS RUPTURE OF A LIVER SUBCAPSULAR HAEMATOMA DURING PREGNANCY

RUPTURA ESPONTÂNEA DE HEMATOMA SUBCAPSULAR HEPÁTICO NA GRAVIDEZ

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ABSTRACT

We report the case of a 37 years-old nulliparous woman who developed HELLP (Hemolysis Elevated Liver Enzymes Low Platelets) syndrome with intense upper abdominal pain during the 27th week of pregnancy. A subcapsular right liver lobe haematoma and a large peritoneal effusion were identified on ultrasound. Fetal extraction was carried out by caesarean section, during which liver exploration revealed a ruptured subcapsular haematoma which was coagulated with Argon LASER. Recovery was slow, and re-absorption of the haematoma was documented by serial imagiological exams. Although rare, ruptured subcapsular liver haematoma should be considered in pregnant women presenting with upper abdominal pain and signs of hemorrhagic shock.
INTRODUCTION

Hepatic rupture during pregnancy is a rare but dramatic occurrence with a high risk of maternal and fetal mortality (it can reach 50%). Generally occurring in the 3rd trimester or in the puerperium (20%)\(^1\), it is associated with severe preeclampsia or HELLP (Hemolysis Elevated Liver Enzymes Low Platelets) syndrome, affecting up to 1/4500 pregnancies. Spontaneous hepatic rupture is preceded by subcapsular haematoma formation that leads to right upper quadrant pain due to distension of the hepatic capsule. After the rupture, the development of a hemoperitoneum justifies the peritoneal signs and hypovolemic shock that may occur. If blood loss is extensive hypertension can be borderline or absent at presentation, becoming visible only after volemic correction.

Blood analysis may show anaemia, thrombocytopenia, hypofibrinogenemia, elevation of liver blood tests and prolonged coagulation tests\(^1\).

Once the diagnosis is confirmed by ultrasound, computerized tomography (CT) or nuclear magnetic resonance (NMR), an immediate surgical exploration with fetal extraction is mandatory.\(^2\)
A 37-year old nulliparous in the 27th week of pregnancy was transferred to the obstetric emergency room of Coimbra’s University Hospitals (CUH) with a HELLP syndrome. Prenatal care had been provided since the first trimester by her family doctor and the pregnancy was uneventful until admission.

She presented with epigastric pain, hypertension (170/100 mmHg) and the laboratory tests performed revealed an haemoglobin of 10.1 g/dl, an hematocrit of 29 %, 43000/uL platelets, 8.5 ug/ml of fibrin degradation products, 3.0 g/L of fibrinogen, no relevant changes in coagulation times, an SGPT/ALT of 421 U/L, 276 U/L of SGOT/AST, 454 U/L of LDH, a total bilirubin of 2.6 mg/dl, a direct bilirubin of 1.5 mg/dl and normal urinalysis. Obstetrical ultrasound revealed a normal fetus with an estimated weight of 870 g.

After the introduction of anti-hypertensive drugs, magnesium sulphate and steroid (dexamethasone) therapy, blood pressure decreased to normal values (100/70 mm Hg) in the first hours after admission, but lab tests showed no improvement 6 hours later. Some minutes later there was an increase in the abdominal pain, localized to the right upper quadrant, with abdominal guarding. An abdominal ultrasound scanning was performed showing a subcapsular liver haematoma involving the right lobe and extensive hemoperitoneum.

Consequently, it was decided to perform an immediate abdominal exploratory laparotomy with delivery by caesarean section. Once the abdominal cavity was exposed 1700 ml of free hemoperitoneum was drained, followed by hysterotomy and delivery of a baby girl weighing 680 g, who was intubated at birth and admitted to the neonatal intensive care unit. Liver exploration showed an extensive haematoma involving the diaphragmatic face of the right lobe with rupture of Glisson’s capsule at its anterior edge (Figure 1). With the cooperation of a general surgeon Argon LASER coagulation of the bleeding area was performed. During surgery, transfusion of only 2 U of packed red cells and 6 U of plasma were necessary.
Post-operative recovery was slow but favourable. After close monitoring for two days at the Intensive Care Unit, hospital stay was prolonged for ninety days. Serial ultrasounds and CT scans (Figures 2 and 3) were performed to monitor the resorption of the liver haematoma.

On the 71st post operative day, the child was discharged from the neonatal intensive care unit, weighting 2090 g, after a favourable clinical evolution.
DISCUSSION

Rupture of subcapsular liver haematoma during pregnancy is rare, with an incidence ranging from 1:15000\(^1\) to 1:45000\(^4\) live births. About 0.9% patients with HELLP syndrome develop subcapsular hematoma\(^5\). In pregnancies complicated with hypertension, liver haematoma is probably due to fibrin clots deposition (arising from endothelial dysfunction with activation of the intravascular coagulation) in the hepatic arterioles and sinusoids and also to periportal haemorrhagic necrosis, inciting capsule distension\(^2,6,7\). The presence of microscopic hepatopathy in these cases is probably underestimated, since random sampling of the hepatic surface in preeclamptic women has shown variable degrees of haemorrhage (ranging from hemorrhagic spots to subcapsular haematoma)\(^8\).

The free hemoperitoneum resulting from capsule rupture causes peritoneal signs (abdominal guarding) and can lead to hypovolemic shock. Often, hypertension is absent or borderline, becoming evident only after volume replacement\(^1\).

Blood analysis may show anaemia, thrombocytopenia, hypofibrinogenemia, elevation of liver blood tests and prolonged coagulation tests\(^1\).

The differential diagnosis should include fatty liver of pregnancy, placental abruption with coagulopathy, thrombotic thrombocytopenic purpura and haemolytic-uremic syndrome\(^1,3\) and differentiation should be based on clinical, analytic and imagiological changes.

Once confronted with a suspicious case, imaging is indicated, either by abdominal ultrasound, NMR or CT scan\(^1\).

Treatment should begin with volume replacement and coagulopathy correction if needed. Immediate surgical exploration with simultaneous delivery should be performed. Local liver haemostasis can be achieved by hepatic suture, cauterization, Argon LASER, temporary tamponade, hepatic artery ligation or even segmental hepatectomy\(^1,4\). More recently there have been reports of successful management of these situations with selective arterial embolization\(^9,10\).
Treatment should be as conservative as possible (a hemodynamically stable patient with an unruptured hematoma at imaging may be followed by imaging without intervention\(^9\)), leaving the more aggressive interventions to particularly severe cases\(^2\).

The postoperative period may require close monitoring in an intensive care unit and serial imaging exams to define the dimensions and progression of the haematoma and to identify further ruptures. The management of these situations should be multidisciplinary, thus leading to a mortality rate reduction\(^2\). Nevertheless, maternal mortality can reach 60% and is mostly due to massive haemorrhage and coagulopathy\(^1,3,4\).

In surviving woman, further morbidity can be due to respiratory distress syndrome, pulmonary oedema, acute hepatic failure\(^1\), acute renal failure, disseminated intravascular coagulation and multiple transfusions\(^4\).

Although rare, this diagnosis should be considered every time a pregnant woman presents with upper abdominal pain and signs of haemorrhagic shock, not only in those pregnancies with hypertension but also in otherwise apparently uncomplicated pregnancies.

In this particular case the prompt suspicion and diagnose made possible a fast intervention in the control of the haemorrhage.

The favourable outcome in the end (although after a long period of time), contrasts with the two other cases occurred in our institution in the past ten years, both culminating with the death of the patients. In one of these cases, the mother was sent to our department already in shock, and died rapidly. Diagnosis was made upon the autopsy report. The other case occurred in the third puerperal day and also had a catastrophic evolution.

From our experience, we must retain that it is of great importance to promptly consider this complication, because rapid identification and multidisciplinary intervention can save both mother and child lives.
REFERENCES


Legends:

**Figure 1** – Discontinuity area in the anterior edge of the subcapsular liver haematoma (white arrow).

**Figure 2** - Enhanced abdominal CT before (a) and after (b) intravenous iodine contrast, shows a lentiform hypodense collection, anterolateral to the right lobe, deforming the liver contour, with 17 cm – subcapsular haematoma. Some spontaneous hyperdense areas are identified (60 UH) in the interior, compatible with recent haemorrhage.

**Figure 3** - Abdominal CT performed before (a) and after (b) intravenous iodine contrast. Coronal reconstruction. Subcapsular liver haematoma.
Figure 2a

Figure 2b
Figure 3