

Rupture of hepatic hematoma: a rare diagnosis during pregnancy

Rotura de hematoma hepático: um diagnóstico raro na gravidez

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Abstract

The rupture of an hepatic hematoma is a rare but catastrophic complication of pregnancy. Pre-operative diagnosis requires a high index of suspicion, however, in the majority of cases the diagnosis is only intra-operative. Due to the rarity of this complication, there are no standard recommendations on the management. 38 years-old primigravida admitted at 32 weeks pregnant with a diagnosis of preeclampsia in association with dyspnea and retrosternal and epigastric pain, and subsequent imagological suspicion of hepatic hematoma. Submitted to cesarian section with intraoperative confirmation of the hematoma, treated conservatively. Transference to the Intensive Care Unit with clinical and laboratorial improvement.

Keywords: Hepatic hematoma; Rupture; Pre-eclampsia; pregnancy.

Resumo

A rotura de um hematoma hepático é uma complicação rara, mas catastrófica da gravidez. O diagnóstico pré-operatório requer um elevado índice de suspeição clínica, no entanto, na maioria dos casos o diagnóstico é intraoperatório. Devido à raridade do diagnóstico, não existem recomendações *standard* quanto à abordagem. Primigesta de 38 anos com 32 semanas de gravidez internada por pré-eclâmpsia associada a quadro de dispneia e dor retrosternal e epigástrica, com subseqüente suspeita imagiológica de hematoma hepático. Realizada cesariana urgente com confirmação intraoperatória do hematoma, tendo-se optado por abordagem conservadora e transferência para a unidade de cuidados intensivos, com evolução clínica e analítica favorável.

Palavras-chave: Hematoma hepático, Rotura, Pré-eclâmpsia, Gravidez.

INTRODUCTION

The rupture of a subcapsular hepatic hematoma is a rare (incidence ranges between 1/250000 and 1/40000 births, and 1/2000 and 1/50 births if associated with pre-eclampsia/HELLP) but catastrophic complication of pregnancy, occurring most of the time in association with severe pre-eclampsia or primarily Hemolysis, Elevated Liver enzymes and Low Platelets (HELLP) syn-

drome^{1,2,3,4}. Other causes of hepatic rupture are underlying conditions, such as intrahepatic hemangiomas, adenomas or malignancies, as well as coagulation disorders, and can even rarely occur spontaneously^{3,5}. Some of the main risk factors are advanced maternal age and multiparity¹. This complication happens mostly after the 28-36th week of pregnancy, but it can occur also in the immediate postpartum period or even during labor^{2,6}. The mechanism of hepatic rupture is based on an ischemic insult with intravascular fibrin deposition that leads to hepatic sinusoidal obstruction and intrahepatic vascular congestion, that consequently results in increased

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hepatic pressure as well as necrosis and hemorrhage, which ultimately can culminate in capsular rupture^{2,7}. The signs and symptoms include pain in the right hypochondrium, epigastric pain or shoulder-referred pain, nausea and/or vomiting, headache, and clinical manifestations of hemodynamic instability in the most severe cases^{1,2,3,6}. Pre-operative diagnosis requires a high index of suspicion, considering the variety and low specificity of symptoms¹. It is therefore necessary in most cases a confirmation by imaging, usually ultrasound, as a good screening tool, available and quick to perform; computerized tomography (CT) scan or magnetic resonance imaging (MRI) are usually used as a second line technique to characterize an already known hematoma, being the MRI reserved for the clinically stable patients^{2,3,8}. However, in the majority of cases the diagnosis is only intra-operative, during an urgent/emergent cesarean section due to maternal or fetal decompensation in case of rupture, which always carries a higher risk^{2,6}. The prognosis is poor because of the high maternal and fetal morbimortality associated, depending of multiples factors such as timing of rupture, liver injury grade, availability of therapeutic options and also patient baseline comorbidities^{2,9}. Due to the rarity of this complication, besides the need for emergent cesarean delivery, there are no standard recommendations on the specific management, but critical care can be guided by trauma guidelines, with *American Association for the Surgery of Trauma* (AAST) scoring system being useful in choosing between surgical and non-surgical approach⁹. It is also important to notice the relevance of a multidisciplinary team that can achieve an early diagnosis and adequate treatment^{3,4}. Nowadays, the best results described in stable patients with low-grade injuries were achieved with conservative surgical treatment, mostly liver packing and hemostatic agents, however, more invasive surgical techniques, such as resection, hepatic artery ligation, embolization, electrocoagulation or hemostatic sutures, may be necessary in more complex injuries or for bleeding control in unstable patients, depending of the expertise and local context^{2,3,4,5,6}.

CLINICAL CASE

38 years-old primigravida (pregnancy through intrauterine insemination) with known non medicated hy-

perthyroidism and no surgical history, was admitted to a tertiary hospital at 32 weeks and 6 days of pregnancy with a diagnosis of preeclampsia – new onset hypertension in association with proteinuria. Of notice that, two days before the hospitalization, the patient had sought medical attention in another obstetric emergency room due to a general malaise and retrosternal pain, where she was found with elevated blood pressure but no other analytical alterations (including cardiac enzymes), being discharged and treated with nifedipine and salicylic acid. Upon admission she was hypertensive (blood pressure 158/98 mmHg) and dyspneic (SpO₂ 98%), complaining of retrosternal and epigastric pain radiating to the right hypochondrium, but with no headache, visual impairment or new onset of peripheral edemas. Blood tests revealed a moderate elevation of liver enzymes (AST 119 U/l, ALT 116 U/l) and anemia (Hb 10.4 g/dl) with no further alterations (normal platelet count 331×10^9). Electrocardiogram as well as troponins were normal. Cardiotocography revealed normal baseline fetal heart frequency and decreased variability, as well as irregular uterine contractions; ultrasound showed fetal biophysical profile 8/8.

In this context, fetal lung maturation and tocolysis with atosiban was initiated as well as magnesium sulfate and antihypertensives according to the department protocol. 13 hours after hospital admission, because of persistence of the pain not responding to the analgesic therapy, associated with analytical worsening with a decrease in hemoglobin levels and raise in hepatic enzymes with normal platelet count (Hb 8 g/dl, platelets 250×10^9 , AST 228 U/l, ALT 267 U/l), an abdominal ultrasound was therefore performed, which revealed an heterogeneity area in the left hepatic lobe, with an anechogenic subcapsular area with 6 centimeters of major diameter and an area of heterogeneous density with 8 centimeters of major diameter, suggesting an intrahepatic hematoma as well as a subcapsular hematoma (Figure 1), in association with moderate free fluid, mainly in perihepatic space and right parietocolic gutter.

These findings led to the decision to perform an urgent cesarean section with a midline infraumbilical incision, without completion of fetal lung maturation, together with the general surgery team, during which the intraoperative confirmation of the diagnosis was achieved: subcapsular hepatic rupture with no active hemorrhage

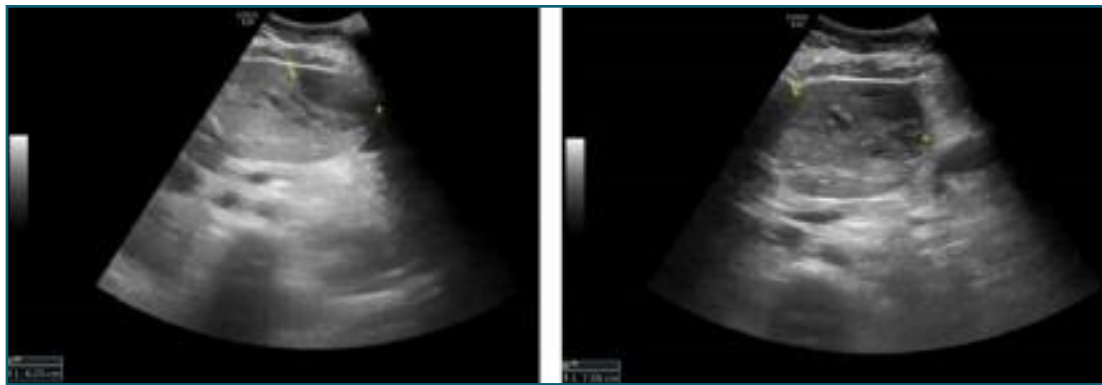


FIGURE 1. Abdominal ultrasound revealing an heterogenous area in left hepatic lobe, with an anechogenic subcapsular area with 6 cm of major diameter suggesting a subcapsular hematoma (on the left) and an area of heterogeneous density with 8 cm of major diameter suggesting an intrahepatic hematoma (on the right).



FIGURE 2. Intraoperative evidence of subcapsular hepatic rupture with no active hemorrhage (darker area limited by black line in the image).

(Figure 2) and associated hemoperitoneum with estimated blood losses of 200 cc. Given the absence of active bleeding, the general surgery team chose a conservative treatment method, placing a hemostatic agent *Tachosil*® (Takeda Austria GmbH, Linz, Austria) on the rupture site. The surgery went on without complications. The new-

born (weight 1730 gr and Apgar Index 9-10-10) was transferred to the Neonatal Intensive Care Unit, and our patient to the Surgical Intensive Care Unit, with continuous infusion of magnesium sulfate and labetalol.

There were no complications during the immediate postoperative period, with extubation and suspension of labetalol on the first postoperative day, followed by transfer on the 4th postoperative day to intermediate care. On the 7th postoperative day, after clinical and laboratorial improvement (resolution of anemia after 3 units of red blood cells transfusions with minimum hemoglobin of 7.9 g/dL and improving of hepatocellular pattern with maximum AST and ALT of 243 U/l e 267 U/l respectively), as well as tensional control with quadruple oral anti-hypertensive medication (methyldopa, nifedipine, carvedilol and captopril), the patient was moved to the Obstetrics department. On the 10th postoperative day, blood tests revealed an worsening of cholestatic pattern (GGT increase to a maximum of 257 U/l as well as ALP increase to a maximum of 375 U/l on 14th postoperative day, with normal bilirubin levels and stable AST, ALT and LDH), which lead to repeating abdominal ultrasound, that reveals the known hematoma in reabsorption and was evaluated by a gastroenterologist, being admitted an association with the hepatic rupture given the time correlation and spontaneous resolution in the next days. On the 19th postoperative day, the patient was discharged, clinically stable (without antihypertensive medication) and with hepatic blood tests almost in the normal range (AST 17 U/l, ALT 27 U/l, GGT 284 U/l, ALP 229 U/l, LDH 192

U/I and total bilirubin 0.15 mg/dL), referred to post-discharge appointments (Obstetrics, General Surgery, Intensive Care and Endocrinology).

DISCUSSION

Although rupture of a hepatic hematoma in a patient with pre-eclampsia is a rare event, maternal and fetal mortality rates remain high, still exceeding 20%, despite advances in management, hepatic surgery and critical-care unit assistance^{10,11}. In this context, the pertinence of this case resides in the fact of the diagnosis being made through the clinical examination and ultrasound, instead of the most common and later intraoperative diagnosis. This clinical report warns about the importance of a high index of suspicion in the face of a patient with pre-eclampsia and severe abdominal pain or non-responding to analgesic therapy pain, allowing an early diagnosis and reducing the need for emergent intervention due to hemodynamic instability or fetal distress, with clear benefits on maternal and newborn outcomes.

Besides that, this case highlights how crucial a multidisciplinary team is, including professionals from the areas of Obstetrics, General Surgery, Anesthesiology, Intensive Care Medicine, Radiology and Immunohemotherapy, in the workup of a complex and challenging case such as this one. Even after successful immediate treatment of hepatic rupture, there may be direct and indirect hepatic sequelae and these patients remain at high risk of complications, such as infection and necrosis, being mandatory to be aware of postoperative challenges that may arise during follow-up^{10,11}.

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AUTHOR CONTRIBUTIONS

Sara Sardinha Abrantes: Writing of manuscript, review and editing. Noemi Curzel: review and editing. Rute Branco: review and validation. Mariana Miranda: review and validation. Teresa Diniz Costa: review and validation.

CONFLICT OF INTEREST STATEMENT

None declared.

INFORMED CONSENT

Written informed consent for publication of the case was obtained from the patient.

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