Ruptured subcapsular liver hematoma and pregnancy: a rare complication of severe preeclampsia: a report of a case discovered fortuitously at the Maternity Teaching Hospital of Cocody

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Summary

We report a case of spontaneous rupture of a subcapsular hematoma of the liver (SHL). It was discovered incidentally at the end of an emergency exploratory laparotomy performed due to unexplained hemoperitoneum with hypovolemic shock which occurred with severe preeclampsia. Diagnosis and therapeutic management are very difficult in sub-Saharan Africa due in part to the limitations and lack of medical equipment. The prognosis is usually marked by the death of the patient, as in our case. Through this clinical observation we wanted to show the interest in performing a liver ultrasound at any level of preeclampsia to detect liver abnormalities as soon as possible.

Key words: Subcapsular hematoma Liver (SHL); Pregnancy; Preeclampsia.

Introduction

Subcapsular hematoma of the liver (SHL) is a very serious but uncommon complication of severe preeclampsia [1]. It is defined as blood collection under the liver capsule and was described for the first time by Abercombie quoted in the Mahi *et al.* study [2]. It occurs in an unspecific clinical picture leading to a delayed diagnosis [1, 3] which is often detrimental because it leads to secondary rupture of the liver capsule which is rare and extremely serious [1, 3]. Maternal mortality is in order of 50 to 75%, and that of the fetus of 60 to 80% [3]. This prognosis is more for burden for our sub-Saharan Africa where people are particularly poor and financially deprived. Through this clinical observation we wanted to measure the severity related to the difficulties of diagnosis and management in our developing country.

Case Report

A 34-year-old female, G4/P3, consulted for preeclampsia at 32 gestational weeks with predominant abdominal pain syndrome in the upper right quadrant. The symptoms had evolved for two weeks with headache and vomiting. She received two days of antiulcer treatment and diazepam with no improvement. In her history no knowledge of hypertension during previous pregnancies or peptic ulcer was found.

On admission, chronic epigastric pain associated with excessive weight gain was noted. The medical examination highlighted serious epigastric pain associated with extreme agitation. Her blood pressure was 160/110 mm Hg, associated with massive albuminuria. Uterine size was estimated at 29 cm. The fetal heart rate was regular at 144 beats per minute. Rather

quickly the clinical signs became serious, with hypovolemic shock and extreme abdominal distension without diffuse loss of normal contours of the uterus. There was no vulvar bleeding so we considered hypovolemic hemorrhagic shock with hemoperitoneum. An emergency exploratory laparotomy was therefore carried out associated with reanimation measures. After aspiration of abundant hemoperitoneum (21), a cesarean first allowed the extraction of a dead fetus. The exploration found no uterine lesion. No uterine vascular injury was observed. However the patient continued to have active bleeding from the floor above the mesocolic shelf. The exploration showed the presence of a large hematoma in the capsule of the liver with bleeding in almost all segments of the hepatic hematoma with rupture of segments 4 and 5 (grade 3). Moreover, the liver was soaked with blood giving it a brownish appearance. The spleen and the rest of the mesocol were healthy.

Hemostatic tamponade of the hematoma with abdominal fields was carried out in vain. The hemodynamics could not be rectified because of the unavailability of blood products. The death of the patient occurred on the operating table two hours after the start of surgery.

Discussion

Frequency

SHL is rare outside of pregnancy [2]. The occurrence during pregnancy is mainly seen in association with preeclampsia and/or HELLP syndrome [2]. Its frequency varies according to reports. It is low in developed countries, estimated at 1/40,000 to 1/250,000 pregnancies [4]. This may be related to better detection and better treatment of preeclampsia. This frequency remains undervalued in underdeveloped countries, especially those in sub-Saharan Africa. Indeed, mild forms go unnoticed due to the limited practice of ultrasound (US) or abdominal scanner. Also in severe cases responsible for sudden

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death, an autopsy is performed exceptionally to make the diagnosis in our country. Sociocultural and economic beliefs are mentioned in particular to explain this. Previously, however, the lack of diagnosis was related to the poor quality of US in our country.

Contributing factors

Apart from preeclampsia, SHL can occur in direct abdominal trauma or pre-existing focal hepatic lesion (hemangioma, adenoma, focal nodular hyperplasia, hepatoma, and perihepatitis) [5] or anticoagulant treatment [2]. Few cases of spontaneous onset of SHL during pregnancy have been described [2].

Hematoma can occur at any age, with a wide choice in multiparous women aged over 30 years old [5]. It occurs in 50% of cases after 36 weeks of gestation and in 85% of the cases it appears before labor during and in 15% of cases during postpartum [1]. Also it more frequently involves the right lobe of the liver (75%), the left lobe in 14% and both lobes in 11% of cases [3]. These facts are identical to those found in our case and thus support the literature data.

Pathophysiology

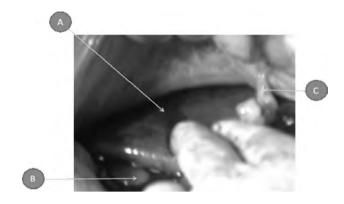
Some authors [2] incriminate the role of microtraumatism in the formation of the hematoma, which occurs in a liver already weakened by preeclampsia, such as abdominal extension. After autopsy, in fact, there is extensive necrosis of the ischemic liver, hemorrhagia, and some cases of fibrin deposits in the periportal sinusoids [2]. Disturbances in coagulation have also been implicated [2]. However in most cases, preeclampsia, is for most authors [1, 2, 5] the grounds of this complication, as indicated in our observation.

For Langer *et al.* [1] the mechanism of occurrence of the hematoma could be explained by two complementary theories. One involves acute disseminated microangiopathy causing acute coagulopathy which results in deposition of fibrin in the sinusoids and arterioles in the liver: this causes multifocal hemorrhagic necrosis. The other is uteroplacental ischemia which results in the release of vasoactive substances responsible for causing spasms of capillary doors, ischemia and hemorrhagic necrosis of the liver [1, 2].

Clinical diagnosis

All authors agree on the difficulty of diagnosis. Indeed, small liver lesions bring about a simple distension of Glisson's capsule and can explain preeclampsia in some patients; the upper right quadrant pain resolves spontaneously after delivery [2]. In severe forms the diagnosis rarely occurs before surgery [2].

SHL usually involves two successive evolutionary stages spontaneously [3, 6]. The first one corresponds to the formation of an unruptured hematoma with sudden distension of the capsule in preeclampsia, characterized by severe epigastric pain and/or right upper quadrant pain with scapular lumbar or thoracic radiation. It is often



A = Huge bleeding hematoma of the right lobe of the liver.

B = Gallbladder.

C = Liver suspensory ligament.

accompanied by painful hepatomegaly on palpation [2]. It is resistant to the usual analgesics and may be accompanied by nonspecific signs (headache, nausea or vomiting with faintness) which can delay diagnosis [1]. The pain is the best sign suggestive of a hematoma.

In the second phase intraperitoneal rupture of SHL occur can which causes hemorrhagic shock associated with signs of cardiovascular collapse [2]. Physical examination noted a distended abdomen, painful as a whole [6]. In our case the delay in diagnosis of our patient caused rupture with hemoperitoneum.

Diagnostic tests

Clinical and hepatic lesions may precede all changes with the biological value of medical imaging as a diagnostic aid [5]. In an emergency setting, US examination has established itself as essential for the diagnosis [3]. It was difficult to perform for our patient due to the emergency related to the precarious state of the patients and the rapid installation of the clinical picture as was the case for many authors [2, 5]. Abdominal US is required first as suggested by El Youssoufi et al. [3] in cases of preeclampsia and/or HELLP syndrome. It can confirm the presence of an already suspected SHL or in case of associated intraperitoneal effusion [2]. US scans evoking a SHL may even precede the onset of clinical signs such as Strauss et al. pointed out [6]. The liver scanner, allows better assessment of a liver injury, showing an oval hypodense image [6]. Mesenteric arteriography also objectifies arteriolar rupture and specifies the topography of the hepatic arterial vasculature to facilitate hemostatic action [6]. These two tests are not common in our practice. Moreover paraclinical explorations should not delay any therapeutic action in these patients.

Treatment

The management of SHL depends mainly on the integrity of the liver capsule and follows the rules of conventional liver surgery [1, 6]. Further resuscitation pre-,

per- and postoperative aims to correct high blood pressure, hypovolemia, circulatory collapse and possible alterations in coagulation [9]. Fetal extraction by cesarean section alone can stop the progression of liver damage in cases of preeclampsia and HELLP syndrome according to Langer *et al.* [1].

In case of integrity of the capsule (unbroken SHL), the authors agree on surgical abstention of the liver. Suffice it to medical treatment (blood transfusion) and an abdominal scan or US scan [7] to monitor the spontaneous regression of the hematoma [1]. Surgical treatment is indicated if there is rupture of the hematoma. Hemostasis is achieved by as conservative means as possible, namely by packing or tamponade hemostatic substances (biological glue, collagen compresses). Upon failure of conservative measures, the use of hepatic artery ligation or one of its branches has been described [1, 8]. Arterial embolization is a nonsurgical method that reduces mortality [6]. A liver transplant should be considered exceptionally because of the many problems, and our patient's only indication remained acute liver failure after control of bleeding [2]. We opted for conservative treatment because of our limited therapeutic options.

Prognosis

The prognosis of SHL is poor for most authors [1-3]. Maternal mortality is heavy, from 50 to 75% and fetal mortality ranges from 60 to 80% [3]. In one series, El Youssoufi et al. reported five maternal deaths of eight cases and four fetal deaths [3]. In our case maternal death occurred due to vain intraoperative hemostasis attempts. The poor prognosis appears to be related to three key factors for some authors [5, 6]: 1) the delay between SHL and surgery, 2) extent, and 3) especially the characteristic of the ruptured hematoma. In our case these factors were associated. Wicke et al. [8] noted no deaths with unruptured SHL cases that did not undergo surgery. However, maternal mortality was extreme (three times) in unoperated cases when the hematoma was ruptured. In all cases when a cure is obtained, it is without sequelae. The obstetric future does not seem to be compromised and a new pregnancy can be carried out under supervision [2].

Conclusion

SHL is a very serious and rare complication of preeclampsia and HELLP syndrome. The maternal and fetal mortality are heavy. The diagnosis should be considered in any pregnant woman in labor or with right upper quadrant pain and/or epigastrium pain in relation to preeclampsia. Liver US is needed urgently. It should also be part of the monitoring of any severe preeclampsia. Only such an approach will minimize risks. What to do before a SHL depends mainly on the integrity of the liver capsule. Abstention surgery is recommended by most authors.

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