

Case Report

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Liver Rupture in Preeclampsia Complicated by HELLP Syndrome, A Case Report

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Introduction

Preeclampsia remains a common pregnancy disorder with a high maternal and neonatal mortality and morbidity. It affects 2–5% of pregnancies, and occurs most commonly at term. HELLP syndrome, which is characterized by elevated liver enzymes, and a low platelet count, is a known severe complication of preeclampsia-eclampsia. This syndrome is associated with an increased risk of both maternal and fetal adverse outcomes, and its management is often challenging.

Preeclampsia and HELLP syndrome usually occur in the third trimester and less frequently in the late second trimester or in the postpartum period. Hepatic rupture is a severe complication associated with HELLP syndrome. The incidence of hepatic rupture is 1 pregnancy out of 4500-22500 and occurs in <2% of cases of HELLP syndrome [1-3]. It results from liver engorgement due to extravasation of fluid from porous hepatic microvasculature as a result of abnormal fibrin deposition in the hepatic sinusoids. Sensitization of the reticuloendothelial system of the liver by preeclampsia may render it unable to clear the fibrin thrombi from the circulation resulting in infarction with vascular disruption leading to intrahepatic hemorrhage and parenchymal destruction [1, 2, 4]. We present a case of postpartum hepatic rupture that required both surgical and radiological procedures in order to be solved.

Case Presentation

A 30-year-old Caucasian woman was admitted to our department at 29 weeks and 5 days of gestation with hypertension, proteinuria and severe intrauterine growth restriction (IUGR). The pregnancy was dated by her last menstrual period, which was consistent with first trimester sonographic measurements. Upon admission, the patient's systolic and diastolic BPs ranged from 160 to 180 mm Hg and from 91 to 105 mm Hg, respectively. She denied any headache, nausea, vomiting, right upper quadrant or epigastric pain, visual disturbances or scotoma, chest pain, or palpitations.

The physical examination on admission revealed an afebrile, hypertensive status, no evidence of peripheral edema or any neurological deficits and normal deep tendon reflexes. The initial laboratory evaluation revealed hemoglobin 10.6 g/dL, platelet count 284,000, creatinine level 0.6 mg/dL, lactate dehydrogenase (LDH) 631 Units, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) of 23 Units/L and 37 Units/L, respectively. AT III was 63%. The patient was admitted for evaluation and control of hypertension. Proteinuria was 4274 mg/24 hours.

After adequate counselling, an expectant management with nifedipine was performed. On day 2 a slight increase of AST, ALT and LDH was recorded (53 Units/L, 63 Units/L and 891 Units respectively). On day 3 a sudden worsening of clinical condition and of blood tests was recorded and the patient showed intractable hypertension and epigastric pain; blood test revealed an important increase of AST (295 Units/L), ALT (444 Units/L), LDH (2720 Units/L) with signs of haemolysis and initial impairment of coagulation. An emergency caesarean section was performed and the surgery was uneventful.

On day 4 patient referred an intractable epigastric pain and blood test revealed an important increase of AST (798 Units/L), ALT (1194 Units/L), a platelet count of 98.000 and a haemoglobin 9.1 g/dL. Ultrasound examination demonstrated an important haemoperitoneum and a subsequent explorative re-laparotomy was performed under general anesthesia. An haemoperitoneum of 1 litre was drained; the uterine suture and pelvic organs were normal with no signs of active bleeding; the Pfannenstiel incision was extended to detect the source of bleeding via midline incision up to the xiphoid process resulting in a T-incision. A 10 cm laceration on the right lobe of the liver from a subcapsular haematoma was observed; to initially reduce blood loss, the liver was packed using abdominal swabs. Finally, the rupture was closed using Z-stitches with nonabsorbable monofilament sutures, hemostatic agents and with positioning a 2 surgical drainages.

On day 6, An emergency computed tomography (CT) scan of the abdomen and pelvis was carried out due to a persistent low hemoglobin count which revealed hemoperitoneum and multiple areas of active extravasation from the right hepatic lobe. Emergent visceral angiography revealed multiple foci of contrast medium extravasation in the peripheral branches of the right hepatic artery. The portal vein was widely patent. The proximal right hepatic artery was selectively catheterized with a 5-F RC1 catheter and embolized with metallic coils. Postembolization arteriography showed successful “pruning” of all peripheral right hepatic artery branches and cessation of bleeding. Two days later, a follow-up CT scan of the abdomen and pelvis revealed extensive hemoperitoneum near right hepatic lobe as well as a large intrahepatic haematoma; no signs of active bleeding but extensive right pulmonary atelectasis with pleuric effusion were noted. The patient remained in the intensive care unit for 5 days. A Subsequent CT scan performed 17 days later and an arteriography 38 days later showed a progressive regression of haematoma and the haemoperitoneum; the hepatic vasculature was always patent.

The patient was discharged at day 44 in good conditions with almost normalized blood tests. Ultrasonographic examination 1 month after discharge showed an ipoechoic area in right hepatic lobe due to the devascularization performed with arteriography; no signs of haemoperitoneum or pleuric verse were noted.

Discussion

Usually HELLP Syndrome occurs between 28 and 36 weeks of gestation, but it can also occur postpartum or even during labor [5]. It is associated with a maternal mortality rate of 3.3% and hepatic hematoma and rupture are its rare complications. When hepatic hematomas are present, or when hepatic rupture develops, mortality and morbidity rise significantly (total maternal mortality rate is 10–22%, while fetal deaths are described in 31% and up to 80% of cases) [6].

Preeclamptic and eclamptic patients are found to accumulate sinusoidal fibrin in the liver, especially in those who develop HELLP syndrome. This accumulation leads to obstruction of the liver sinusoids, leading to ischemia, hepatic necrosis, intraparenchymal and subcapsular hemorrhage and in rare cases, rupture of parenchyma and capsule. The right lobule is generally affected [7].

Clinically hepatic hematomas are characterized by pain in the right hypochondrium or epigastrium (70–90% of cases), though other symptoms may be associated. Given their rarity and variable presentation, delays in diagnosis are frequent, which may pose serious consequences.

Unfortunately, the patient’s clinical history cannot precisely predict the likelihood of liver trauma. Often laboratory findings only show slight changes in values [8].

The typical upper abdominal pain in HELLP syndrome occurs in most patients even without liver trauma. Whereas, the distinction between common HELLP syndrome symptoms and the possible combination of HELLP syndrome with hepatic trauma could be difficult; the early use of ultrasound examination is very useful because can detect major abnormalities, such as hematomas, ruptures or free intra-abdominal fluid [9].

Management mainly consists in eliminating the causal factor and controlling hemorrhage. The most important factor that lead the flow chart or treatment is the hemodynamic status and the severity

of the hepatic lesion. In case of a hemodynamically stable patient with hepatic hematoma, the hematoma does not require specific surgical treatment. In case of a hemodynamically unstable patient or with diagnosis of hepatic rupture, an active approach with surgery should be immediately performed [6].

Regarding the management of pregnancy, without signs of fetal compromise or impaired maternal circulation, a conservative approach with short-time re-evaluation could be taken into consideration. Prolongation of pregnancy, is indicated in few cases, such as in a very early preterm birth in order to administer antenatal steroid treatment. In most cases an immediate delivery via cesarean section and further surgical treatment is necessary.

Regarding surgical management, the approach should respect the same principles of trauma surgery [6]. The course of action involves controlling the hemorrhage and, after hemostasis, proceeding with the definitive treatment in the same or subsequent surgery. Usually Most patients can be cured with a conservative approach (liver packing with abdominal swabs, with or without omental packing, hemostatic sutures and hemostatic agents) and only in very few cases hepatectomy with or without cholecistectomy or liver transplantation is required [6, 10]. Centres with interventional radiology and transcatheter arterial embolization can be an alternative to or in combination with surgical procedures. Embolization significantly decreases maternal mortality and morbidity when performed by well-trained specialists. In our case arteriography avoided a re- laparotomy with an important increase of maternal morbidity [11].

The cornerstone for the management of hepatic rupture is the prompt diagnosis and the immediate evaluation of haemodynamic status of patient; usually a timely intervention proves decisive [6].

For this reason seems to be useful to extend the field of ultrasound examination beyond the limited obstetrical focus in patients with upper abdominal pain as a standard procedure to improve the correct diagnosis and initiate prompt treatment of liver hematoma and rupture. Ultrasound could be a first line examination in those cases of upper abdominal pain and magnetic resonance could be considered for patients with pathological findings at ultrasound.

Conclusion

It is fundamental to remember when treating hepatic rupture that this rare complication can be a severe complication of HELLP syndrome and a combination of surgical treatment and aggressive supportive management could effectively reduce mortality rate.

The cornerstone of prognosis is early diagnosis. Those rare cases should be managed in tertiary health care centres where more experienced medical teams are available.

References

1. Masato Yoshihara, Michinori Mayama, Mayu Ukai, Sho Tano, Yasuyuki Kishigami, et al. (2016) Fulminant liver failure resulting from massive hepatic infarction associated with hemolysis, elevated liver enzymes, and low platelets syndrome. J. Obstet. Gynaecol. Res 42: 1375-1378.
2. Ellington SR, Flowers L, Legardy-Williams JK, Jamieson DJ, KourtisAP (2015) Recent trends in hepatic diseases during pregnancy 524: e1-e7.
3. Sibai BM, Ramadam MK, Utsa I, Salama M, Mercer BM, et al. (1993) Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). Am J Obstet Gynecol 169: 1000-1006.

4. Arias F, Mancilla-Jimenez R (1976) Hepatic fibrinogen deposits in preeclampsia: immunofluorescent evidence. *N Engl J Med* 295: 578-582.
5. MF van Oostwaard, L van Eerden, MW de Laat, C JJ Duvekot, d JJHM Erwich, et al. (2017) Maternal and neonatal outcomes in women with severe early onset pre-eclampsia before 26 weeks of gestation, a case series. *BJOG* 124: 1440-1447.
6. Raumanns J, Behrendt W, Lehnen H (1992) Spontaneous liver rupture as a rare complication of the HELLP syndrome. *Anaesthesist* 41: 386-390.
7. Francisco Melo Bento, Maria Ines Leite, Antonio Melo, Catarina Moura, Luis Amaral, et al. (2019) Hepatic rupture in HELLP syndrome. *Journal of Surgical Case Reports* 10: 1-3.
8. Masato Yoshihara, Michinori Mayama, Mayu Ukai, Sho Tano, Yasuyuki Kishigami, et al. (2016) Fulminant liver failure resulting from massive hepatic infarction associated with hemolysis, elevated liver enzymes, and low platelets syndrome. *J. Obstet. Gynaecol. Res* 42: 1375-1378.
9. Grand'Maison S, Sauvé N, Weber F, Dagenais M, Durand M, et al. (2012) Hepatic rupture in hemolysis, elevated liver enzymes, low platelets syndrome. *Obstet Gynecol* 119: 617-625.
10. Till Kaltofen, Johanna Grabmeier, Tobias Weissenbacher, Klaus Hallfeldt, Sven Mahner, et al. (2019) Liver rupture in a 28-year-old primigravida with superimposed pre-eclampsia and hemolysis, elevated liver enzyme levels, and low platelet count syndrome. *J Obstet Gynaecol Res* 45: 1066-1070.
11. J Kelly, DJ Ryan, N O'Brien, WO Kirwan (2019) Second trimester hepatic rupture in a 35 year old nulliparous woman with HELLP syndrome: a case report. *J. Obstet. Gynaecol. Res* 45: 1066-1070.