

CASE REPORT

An Unusual Case of Severe Preeclampsia in the Postpartum Period with Hemoperitoneum

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ABSTRACT

Background: HELLP syndrome, consisting of hemolysis, elevated liver enzymes, and low platelet count, is a rare complication of occurring in 10 to 20% of women with severe preeclampsia and in 0.2 to 0.6% of total pregnancies. Delivery is the only definitive treatment, with maternal condition usually improving in the postpartum period.

Hemoperitoneum in a woman with preeclampsia is very rare, usually secondary to hepatic hematoma with rupture. While the prevalence of intrahepatic hematoma in these women has been reported to be as high as 39%, capsular rupture is rare, occurring up to 12% in women with capsular hematoma, carrying with it high mortality for both the mother and fetus.

Aim: We report an unusual case of hemoperitoneum secondary to liver hematoma in severe preeclampsia in the postpartum period that was conservatively managed leading to good maternal outcome.

Case report: We report a case of postpartum onset of severe preeclampsia in a 25-year-old woman complicated by hepatic dysfunction with coagulopathy and hemoperitoneum. This was secondary to a contained subcapsular hematoma and was conservatively managed resulting in a good outcome.

Conclusion: Prompt recognition with careful clinical examination, laboratory investigations and imaging, supportive treatment, and early fetal delivery remain the cornerstone in the management. Although reaching the diagnosis was initially challenging in our case study, both mother and baby had a good outcome with an appropriate supportive treatment.

Clinical significance: Most cases with liver hematomas may lead to hepatic rupture with hemodynamic instability, necessitating an aggressive operative approach. However, conservative management with close monitoring of hemodynamic status can be undertaken if the patient is hemodynamically stable with good outcome, as illustrated in this case.

Keywords: Hemoperitoneum, Postpartum, Preeclampsia, Pregnancy.

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BACKGROUND

HELLP syndrome, consisting of hemolysis, elevated liver enzymes, and low platelet count, is a rare complication of severe preeclampsia. It was first reported by Weinstein in 1982.¹ It occurs in 10 to 20% of women with severe preeclampsia and in 0.2 to 0.6% of total pregnancies.² Delivery of the baby is the only definitive treatment, with the maternal condition usually improving in the postpartum period. Risk factors include multiparity, advanced maternal age, and previous personal and family history of preeclampsia/HELLP syndrome.³

The overall mortality rate for women with HELLP syndrome is 1.1%,⁴ and up to 38% experience adverse maternal outcomes including disseminated intravascular coagulation (DIC), placental abruption, acute renal failure, pulmonary edema, and subcapsular hematoma.⁵ Perinatal deaths have been reported in about 7.7 to 60% of cases.⁶

Hemoperitoneum in a woman with preeclampsia is very rare. This is usually secondary to hepatic hematoma with rupture. While the prevalence of intrahepatic hematoma in women with HELLP syndrome has been reported to be as high as 39%,⁷ capsular rupture is rare, occurring in 1 in 40,000 to 1 in 250,000 deliveries⁸ and 0.53 to 2% of women with HELLP syndrome.⁸ It occurs in about 12% of women with capsular hematoma,⁷ with high mortality rates for both the mother (17%) and the fetus (38%).⁹ Prompt recognition and management are critical to reduce maternal and fetal morbidity and mortality.

We report a case of severe preeclampsia in the postpartum period complicated by subcapsular hematoma with hepatic dysfunction, coagulopathy, and hemoperitoneum.

CASE REPORT

A 25-year-old lady attended KK Women's and Children's Hospital (KKH) for routine antenatal care at 34 weeks of gestation in her second pregnancy. She had one previous

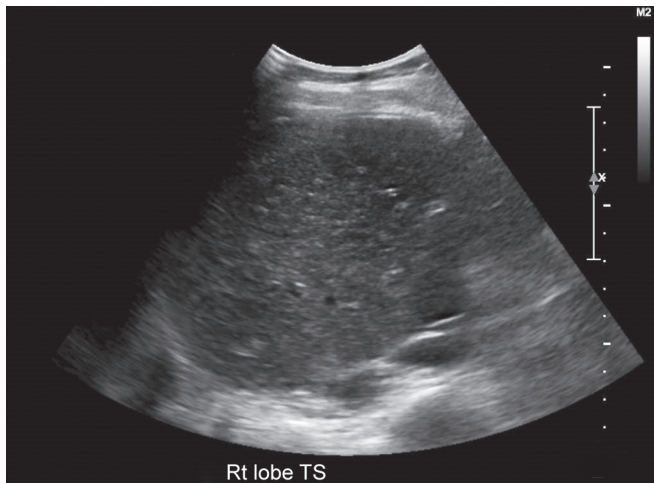


Fig. 1: Hepatic architecture. Mild coarsening of hepatic echotexture is noted on ultrasound scan due to hepatic fibrosis

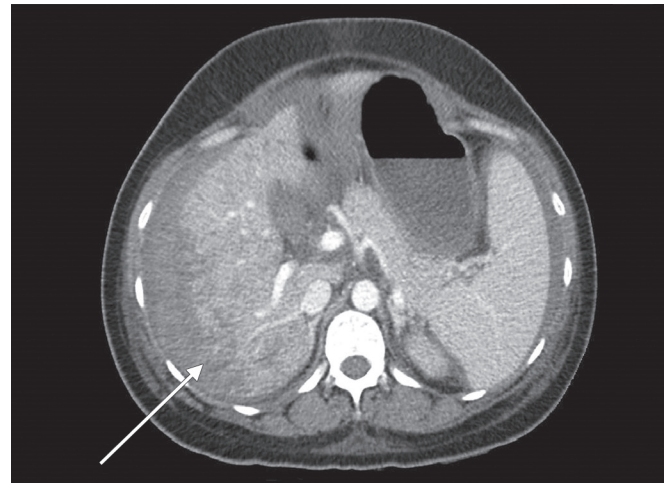


Fig. 2: Subcapsular hematoma. Severe preeclampsia can cause coagulopathies that could lead to subcapsular hematomas as noted on this CT scan image

preterm vaginal delivery at 28 weeks of gestation following preterm premature rupture of membranes. Her baby died 2 weeks after birth due to prematurity. She had no significant past medical or surgical history apart from thalassemia minor.

In this pregnancy, she was booked with a private obstetrician at 10 weeks of gestation. She had an uneventful antenatal period until 34 weeks when she transferred her care to KKH. The rest of her antenatal period at KKH was unremarkable. She had an uneventful vaginal delivery at 39 weeks and 4 days of gestation. The baby's birth weight was 3,160 gm and his Apgar scores were 9 and 9 at 1 and 5 minutes of life respectively.

One hour after delivery, the patient complained of sudden-onset epigastric pain. This was not associated with blurring of vision or headache. Her heart rate was 79 beats per minute, blood pressure was 177/106 mm Hg, and her oxygen saturations were normal. On examination, she did not have any signs of jaundice. Her reflexes were normal with no clonus. She had epigastric tenderness with no abdominal distension or signs of an acute abdomen.

Her investigations revealed 3+ albumin on urine dipstick analysis, urine protein creatinine ratio of 11.3, serum uric acid level of 433 $\mu\text{mol/L}$ (normal range 140–450), low hemoglobin of 8.5 gm/dL (normal range 12–16), raised total bilirubin of 26 $\mu\text{mol/L}$ (normal range 3–20), elevated alanine transaminase of 157 U/L (normal range 0–55), and aspartate transaminase of 213 U/L (normal range 5–34). Serum creatinine, platelets, and coagulation profile were normal.

A provisional diagnosis of preeclampsia/HELLP syndrome was made. She was transferred to the intensive care unit. Nifedipine 20 mg was commenced orally to control her blood pressure. Magnesium sulfate infusion was started to prevent eclampsia. Repeated blood tests

showed thrombocytopenia, worsening transaminitis, and coagulation derangement. An ultrasound scan of the abdomen showed hepatomegaly, mild coarsening of the hepatic echo texture, increased periportal echogenicity, and diffuse edema of the gallbladder wall that raised the possibility of hepatitis or third spacing with moderate ascites (Fig. 1).

A gastroenterologist's review was sought. Further investigations revealed normal serum lipase, amylase, and hepatitis screen. Random serum glucose (8 nmol/L, normal range 3.9–6.0) and lactate dehydrogenase (1289 u/L, normal range 120–260) were elevated and haptoglobin was <0.10 gm/L (normal range 0.37–2.70). Peripheral blood smear showed hypochromic microcytic red blood cells, some polychromatic macrocytes, poikilocytes, few target cells, and metamyelocytes. Her liver function and coagulation profile were closely monitored and serially trended for a day during her stay in our institution.

Her epigastric pain worsened, with radiation to her left hypochondrial region along with increasing abdominal distension and abdominal petechial rash. An urgent computed tomography (CT) scan of abdomen and pelvis revealed multiple areas of active extravasation along hepatic capsular surface and a subcapsular hematoma with possible extension into peritoneal cavity. Large amount of ascites with bilateral pleural effusions and atelectasis of lungs was noted (Fig. 2).

Liver function progressively worsened and she developed DIVC [fibrinogen 0.47 gm/L (normal range 1.80–4.80), activated partial thromboplastin time 52.1 seconds (normal range 29.7–45.6), prothrombin time 26.3 seconds (normal range 11.6–14.2), international normalized ratio 2.47]. Two units of fresh frozen plasma, 1 unit of cryosupernatant, 10 units of cryoprecipitate, and 5 units of packed cell were transfused.

She was then transferred to the high dependency unit of another tertiary general hospital for further management. Serial hepatic artery angiogram revealed hemoperitoneum and perihepatic hematoma with no signs of active bleeding. She was conservatively managed with close monitoring of hemoglobin, platelets, liver functions, and coagulation profile. She received two more units of packed cell for a drop in hemoglobin from 7.2 to 5.4 gm/dL, which subsequently stabilized at 8 gm/dL without a need for any further transfusions. Her liver function and platelet counts improved. She was discharged on 14th postnatal day in stable condition.

DISCUSSION

In total, 70% of HELLP syndrome occurs in the antenatal period and 20 to 30% occurs in early postpartum period, with about 10% occurring in the first week postdelivery.¹⁰ Patients usually complain of nonspecific symptoms, such as malaise, epigastric/right upper quadrant pain, or nausea/vomiting, mimicking other hepatic conditions, such as acute fatty liver in pregnancy, obstetric cholestasis, and viral hepatitis.

Abercrombie¹¹ first described liver hematomas in pregnancy in 1844. Symptoms are nonspecific including acute abdominal/epigastric pain radiating to right shoulder, nausea/vomiting, and abdominal distension that makes early diagnosis challenging. Hemodynamic instability with hypovolemic shock may be present in cases of hepatic rupture.

Rupture of liver is almost always preceded by parenchymal hematoma usually developing in the right lobe of the liver.⁶ Occasionally, hepatic hematomas can be associated with renal hematomas.¹² Exact pathophysiology and cause of intrahepatic hematoma are unknown. There is a suggestion that it could be secondary to fibrinoid thrombi formed from DIVC in the liver sinusoids causing necrosis and periportal hematoma.³ Minor trauma and uncontrolled hypertension may further increase the risk of capsular rupture due to overdistension of the Glisson's capsule.

Diagnosis is aided by ultrasound, CT, or magnetic resonance imaging. In our case, CT scan was done to visualize the entire abdomen to rule out other causes of abdominal distention and pain.

Treatment of liver capsular hematoma with rupture and hemoperitoneum requires a multidisciplinary team involving obstetricians, neonatologists, anesthetists, and gastroenterologists. It requires aggressive fluid resuscitation and infusion of blood products. In patients who are hemodynamically unstable, surgical intervention including drainage, packing, hepatic artery ligation, and partial hepatectomy of affected segment or liver

transplant may be required.⁹ In selected cases, they can be managed conservatively with or without transcatheter hepatic artery embolization. Increasingly, conservative treatment is becoming the mainstay of treatment due to a better understanding of pathophysiology of HELLP syndrome and preeclampsia along with advances in diagnostic imaging techniques. Postnatally, it is important to follow the patient up with serial imaging till the hematoma resolves.

Mortality in patients with liver hematomas is most commonly associated with coagulopathy and exsanguination.^{13,14} In our case, though she had coagulopathy, she had a good outcome with conservative management as she responded well to supportive treatment.

Hemoperitoneum secondary to rupture of hepatic hematoma has been reported in the literature before,¹⁵⁻¹⁷ with surgical treatment being the mainstay of management. Only one case was being conservatively managed.

CONCLUSION

Prompt recognition with careful clinical examination, laboratory investigations, and imaging, followed by aggressive resuscitation, supportive treatment, and early fetal delivery remains the cornerstone in the management of hemoperitoneum secondary to liver hematoma rupture in patients with severe preeclampsia. In this case, although reaching the diagnosis was initially challenging, due to appropriate supportive treatment, both mother and baby had a good outcome.

CLINICAL SIGNIFICANCE

Most cases with liver hematomas may lead to hepatic rupture with hemodynamic instability necessitating an aggressive operative approach. However, conservative management with close monitoring of hemodynamic status can be undertaken if the patient is hemodynamically stable with good maternal outcome, as illustrated in this case.

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