

Spontaneous Liver Capsule Rupture in Preeclampsia: A Rare and Complex Case - Report and Literature Review

Gunjan Bahuguna*, Tamizian Onnig, Shuchi Dixit, Sinduja Palanisamy, Anuja Joshi

University Hospitals of Derby and Burton NHS Foundation Trust, Derby, United Kingdom

Citation: Gunjan Bahuguna, Tamizian Onnig, Shuchi Dixit, Sinduja Palanisamy, Anuja Joshi. Spontaneous Liver Capsule Rupture in Preeclampsia: A Rare and Complex Case – Report and Literature Review. Int Clinc Med Case Rep Jour. 2025;4(11):1-7.

Received Date: 10 November 2025; Accepted Date: 12 November 2025; Published Date: 14 November 2025
*Corresponding author: Gunjan Bahuguna, University Hospitals of Derby and Burton NHS Foundation Trust,
Derby, United Kingdom

Copyright: © Gunjan Bahuguna, Open Access 2025. This article, published in Int Clinc Med Case Rep Jour (ICMCRJ) (Attribution 4.0 International), as described by http://creativecommons.org/licenses/by/4.0/

ABSTRACT

Liver hematoma or rupture is a rare but serious complication of severe preeclampsia, presenting with upper abdominal pain, nausea, vomiting, and deranged liver function tests. Early diagnosis using ultrasound, CT, or MRI is crucial, though in acute cases imaging may not be possible. Management depends on patient stability: stable patients may be managed conservatively, while unstable patients often require damage control surgery with liver packing and hemostatic agents, sometimes supplemented by interventional radiology or, rarely, liver transplant. Rapid resuscitation, correction of coagulation and electrolyte abnormalities, and a multidisciplinary approach involving obstetricians, surgeons, interventional radiologists, and intensivists are key to save a life. Early recognition and prompt intervention, as illustrated in the case of a 37-year-old primigravida with hepatic rupture.

Keywords: Liver hematoma; Liver rupture; HELLP; Severe preeclampsia; Damage control surgery

Abbreviations: HELLP - Hemolysis, Elevated Liver Enzymes, Low Platelet Count, PET – Preeclampsia, CG – Cardiotocography, MDT - Multidisciplinary Team, FAST - Focused Assessment with Sonography for Trauma, CT - Computed Tomography, MRI - Magnetic Resonance Imaging

INTRODUCTION

The incidence of hypertensive disorders in pregnancy in the world is about 10% (Fox et al., 2019). HELLP syndrome which stands for hemolysis elevated liver enzymes low platelet count is a serious complication of preeclampsia, associated with cerebral haemorrhage, pulmonary edema, liver and renal dysfunction, and placental abruption leading to fetal compromise (Vigil-De Gracia and Ortega-Paz, 2012). Spontaneous hepatic hematoma or rupture is a rare event in severe preeclampsia.

International Clinical and Medical Case Reports Journal

Case Report (ISSN: 2832-5788)



Liver hematoma or rupture of liver capsule can present with nausea, vomiting, and upper abdominal pain in the backdrop of preeclampsia. Raised liver enzymes, along with abdominal imaging can guide diagnosis. Ultrasound is a quick tool to identify liver hematomas or free intraperitoneal fluid in the abdomen, suggesting hemoperitonuem.

Management of a hepatic hematoma/ rupture involves a multidisciplinary approach, incorporating conservative management, interventional radiology, damage control surgery, and definitive surgical repair based on the patient's clinical presentation. However, there is no clear consensus on the best approach.

The aim of this case report is to highlight the importance of team-based management in liver hematoma rupture, leading to a successful outcome.

CASE REPORT

A 37-year-old primigravida presented at 38+4 weeks of pregnancy with multiple episodes of vomiting, abdominal pain, and headache. Her pregnancy had been straightforward until then, though she was on the serial scan pathway for low PAPP-A levels. Her BMI was 34 and she did not have any raised blood pressure during her antenatal assessment.

On admission, she had a raised blood pressure of 154/114. Abdominal examination showed a soft but tender abdomen. She was given Paracetamol 1 gm for pain relief and a stat dose of oral labetalol 200 mg. Blood tests for PET were requested, and 2 units of blood were cross-matched. Thirty minutes post labetalol, blood pressure came down to 125/115 and urine dipstick showed 2+ proteinuria. Laboratory blood results showed haemoglobin of 122g/l, WBC 21, neutrophils 19, and platelets 139, INR 0.87, and blood gas arterial lactate 4.4mmol/l, Liver function tests were deranged, with bilirubin 29 micro mol/l, ALP 252 U/L, ALT 595 U/L. Creatinine was elevated at 111 micromol/L and CRP was 1.7mg/L. She continued to have abdominal pain and then the CTG was pathological (baseline of 150, variability until 5, absent accelerations, occasional decelerations, about 4 to 5 uterine contractions in 10).

An emergency caesarean section was planned for the CTG concerns. With a working diagnosis of severe PET, she was started on intravenous magnesium sulphate while awaiting theatre and blood results. However, while awaiting theatre, acute fetal bradycardia of 71 occurred and she had a category 1 caesarean section under general anaesthesia. The procedure was uneventful, and baby had 3 loops of tight cord around the neck, with no placental abruption. There was intermittent relaxation of the uterus, hence tranexamic acid, hemabate were given and a pelvic drain was inserted because of blood-stained peritoneal fluid and coagulopathy risk. The estimated blood loss was 800 ml. One-hour post-caesarean section she had 200mls of fresh blood in the drain. Increasing drain output and rising respiratory rate with normal blood pressure (which was low for her as her initial BP was high) were noted. On examination, her abdomen was soft with no distension and lochia was normal. Because of increasing abdominal pain, respiratory rate, dropping blood pressure and increased drain



output (about 400ml) a decision to return to theatre for a midline laparotomy to allow access to the upper abdomen and to find the cause of bleeding was made. The patient provided informed consent for the procedure. Four units of blood were cross matched before she was taken to the theatre. An on-call vascular consultant was called in, and intraoperatively a tear of 4x1cm was found on the liver capsule. The tear was on the tip of the right lobe of the liver, along with a very edematous liver (Figure 1). About 300ml of clots were evacuated from the upper abdomen. Bleeding was controlled by direct pressure and surgiseal. Once active bleeding was controlled, the abdomen was closed. The estimated blood loss was 1,665 ml. the patient was subsequently transferred to the ITU for close observation.

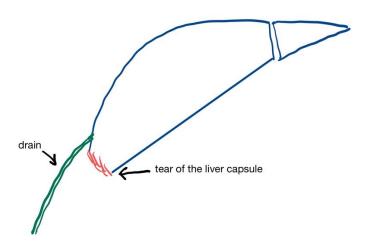


Figure 1: Diagrammatic representation of intraoperative events

After about an hour, bleeding from the abdominal wound was noted with 150ml bloody drain output. Observations were continued, blood was requested and a relook laparotomy was contemplated. Meanwhile, she was hypotensive, BP of 76/54, heart rate of 76, Sats of 99, and RR 25. Drain output increased to 850 ml. Red blood cells and FFP were administered. She had a relook laparotomy in view of deteriorating observations and increased drain output. Intraoperatively, continuous slow ooze from the lacerated right lobe of the liver, the right paracolic gutter, generalised ooze was noted. Variset and two medium abdominal packs were applied to the liver, and one medium abdominal pack was applied onto the right paracolic gutter to control the oozing. Estimated blood loss was 1,500ml. She was moved to the ICU for post-operative monitoring and continued on broad-spectrum IV antibiotics.

The aim was to have a re-re look laparotomy after 24 hours once there were no signs of bleeding and the clotting parameters had normalised. At that point, the total blood loss was 3965ml. Seven units of red blood cells, 4 units of FFP, 1 unit of platelet, and 1 unit of cryoprecipitate had been given in total. A CT abdomen and pelvis was done prior to the return to the theatre to make sure there was no bleeding. CT abdomen and pelvis showed large subcapsular perihepatic hematoma with no evidence of contrast extravasation to suggest haemorrhage. A liver laceration was noted in segment 7. Postoperatively, her observations remained stable and she remained intubated, supported with noraderaline. Blood showed Hb of 68, WBC 18, platelet 97, Creat 138, ALT 2214, and CRP 194. She was given 2 more units of red blood cells.



After an MDT discussion, she was transferred to hepato-biliary centre for removal of abdominal packs. She made an excellent recovery and Table 1 shows the trend of blood results. She had a debrief seven weeks post-event. In terms of implications for future pregnancies, she is at high risk of preeclampsia again and is to be on Aspirin from 12 weeks of pregnancy to reduce the risk.

Table

	22/ 5 05:28	21/ 5 05:41	20/ 5 16:46	20/ 5 02:40	19/5 21:35	19/5 17:24	19/5 10:33
hb	72	68	79	96	94	122	152
wbc	15	18	15	20	16	21	29
platelet	94	97	90	107	74	137	198
Bil	6	7	11	30	25		29
ALT	1412	2214	2710	1513	646		595
creat	91	138	136	115	121	135	111
CRP	231	194	134	38	16		1.7
INR	0.92	0.93	0.95		0.97		0.87

DISCUSSION

Incidence

According to the latest MBRACE report, hypertensive disorders of pregnancy is one of the leading causes of maternal deaths in the UK. Preeclampsia occurs in 2-5% of pregnant women [1]. HELLP which stands for hemolysis, elevated liver enzymes and low platelet count is one of the complications of preeclampsia seen in 10-15% of women (Vigil-De Gracia and Ortega-Paz, 2012). Hepatic rupture accounts for less than 2 % of the complications associated with HELLP syndrome [2].

Pathophysiology behind hepatic capsule rupture

Aberration of the normal physiology of placental formation is a risk factor for preeclampsia, and preterm pregnancies ^[3]. Fibrin deposition in the sinusoids of the liver, necrosis and haemorrhage of the liver parenchyma leads to the rupture of the liver capsule ^[2]

Clinical presentation of liver hematoma/ rupture



Rupture of the liver capsule can present with upper abdominal pain, which can be severe and sudden in presentation as observed in our patient. Pain can typically localised in the epigastric or the right hypochondrium, along with nausea and vomiting. The right lobe of the liver is most commonly affected as seen in a review of 180 patients by [4]. In a review of 93 cases by [5] most of the cases had a platelet count less than 1 x 10 5/cmm, however, whether low platelets lead to hepatic rupture wasn't clear. Deranged liver function tests with raised alanine transaminases are caused by hepatic necrosis and haemorrhage. However, there can be atypical presentations making diagnosis difficult.

Circulatory collapse with low blood pressure in the background of preeclampsia can be misleading, emphasising the need for a thorough clinical assessment to identify the underlying cause.

Diagnosis of liver hematoma/ rupture

In most cases, a FAST scan can pick up free fluid inside the abdomen and point towards hemoperitoneum. Abdominal ultrasound, CT scan, or an MRI can be used to visualise liver hematomas and hemoperitoneum. In a systematic review by ^[5], liver rupture/hematoma was diagnosed before surgery in 54.8% (51/93) of cases, while 39.7% (37/93) were discovered intraoperatively. Only one case was diagnosed post-mortem. This suggests that in most cases, it was possible to diagnose a liver hematoma/ rupture before going for a planned procedure. However, in an acute setting, resuscitation of the patient is imperative and sometimes due to paucity of time imaging is not possible or accessible.

Conservative management of liver hematoma

The management depends on the clinical presentation of the patient. If the patient is stable, an enclosed hematoma can be managed conservatively with the correction of haemoglobin and coagulation parameters. This was successfully achieved in 18.2% of cases (17/93) in the systematic review of 93 cases by ^[5]. Opting for conservative management gives us the choice of steroid injections to hasten fetal maturity in case a preterm birth is anticipated.

The use of interventional radiology to selectively emboli the hepatic artery has been seen in literature with a high success rate, achieving haemostasis in 10 out of 12 cases (83.3%) ^[5].

Surgical management of liver hematoma/ rupture

In an unstable patient, damage control surgery is often employed to control ongoing bleeding using techniques such as liver or omental packing (Kaltofen et al., 2019) and the use of haemostatic agents including surgiseal onto the bleeding site. However, a relook laparotomy is warranted for the removal of the abdominal packs once the patient stabilises. In a systematic review of 93 cases by ^[5], this approach was successfully implemented in 51.6% of patients (48/93).

International Clinical and Medical Case Reports Journal

Case Report (ISSN: 2832-5788)



In a case report by ^[6] a massive liver hematoma involved the entire right lobe of the liver. Perihepatic packing was done which was followed by selective angioembolization of the right hepatic artery. However, she had a necrosis of the right lobe of the liver which necessitated a partial hepatectomy. This highlights that, although angioembolization can be effective, it carries potential complications.

An interesting case by ^[7] demonstrated massive hepatic infarction which was successfully treated by a liver transplant. In a review by Gracia et al (Vigil-De Gracia and Ortega-Paz, 2012) involving 180 cases of hepatic rupture (1992-2012) use of interventional radiology or liver transplant was associated with the lowest maternal mortality rate, at 8.3%.

Aggressive correction of acidosis, electrolyte, and coagulation parameters is imperative to support the management. Broad-spectrum antibiotics also help in preventing serious infections and sepsis in an unwell patient. The timeline of management is also particularly important in an acutely unstable patient, the first goal is to control the ongoing bleeding by a damage control surgery and then going ahead to a definitive surgery after stabilisation.

CONCLUSION

Liver hematoma/ rupture is a rare complication of severe preeclampsia. Rapid resuscitation and efforts by a multidisciplinary team involving obstetricians, vascular surgeons, interventional radiologists, hepatobiliary specialists, and anaesthetists can lead to excellent results. Early diagnosis and prompt management hold the key for maternal survival.

REFERENCES

- Poon LC. et al. "The International Federation of Gynecology and Obstetrics (FIGO) initiative on preeclampsia: A pragmatic guide for first-trimester screening and prevention," International Journal of Gynecology and Obstetrics, 2019;145(S1): 1–33.
- 2. <u>EV, et al. "Liver Rupture in Preeclampsia Complicated by HELLP Syndrome, A Case Report," Journal of Gynecology Research Reviews & Reports, 2023;1–3.</u>
- 3. <u>Labarrere CA. et al. "Failure of physiologic transformation of spiral arteries, endothelial and trophoblast cell activation, and acute atherosis in the basal plate of the placenta," American Journal of Obstetrics and Gynecology, 2017;216(3): 287.e1-287.e16.</u>
- 4. <u>Vigil-De Gracia P, Ortega-Paz L. "Pre-eclampsia/eclampsia and hepatic rupture," International Journal of Gynecology and Obstetrics. John Wiley and Sons Ltd, 2012;186–189.</u>
- 5. <u>Dubey S, Rani J. "'Hepatic rupture in preeclampsia and HELLP syndrome: A catastrophic</u> presentation," Taiwanese Journal of Obstetrics and Gynecology. Elsevier Ltd, 2020; 643–651.
- 6. <u>Millan CA, Forero JC. "Right hepatectomy after spontaneous hepatic rupture in a patient with preeclampsia: A case report," International Journal of Surgery Case Reports, 2017;39: 250–252.</u>



- 7. <u>Mikolajczyk AE. et al. "Massive Hepatic Infarction Caused by HELLP Syndrome," ACG Case Reports</u>
 <u>Journal, 2017;4(1): e81.</u>
- 8. Fox R. et al. "Preeclampsia: Risk factors, diagnosis, management, and the cardiovascular impact on the offspring," Journal of Clinical Medicine. MDPI. 2019.
- 9. <u>Kaltofen T. et al. "Liver rupture in a 28-year-old primigravida with superimposed pre-eclampsia and hemolysis, elevated liver enzyme levels, and low platelet count syndrome," Journal of Obstetrics and Gynaecology Research, 2019;45(5): 1066–1070.</u>