

Undiagnosed Subcapsular Liver Hematoma Rupture in HELLP Syndrome Unveiled during Emergency Cesarean Delivery: Case Report

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DOI: <https://doi.org/10.36347/sjmcr.2025.v13i03.020>

| Received: 02.02.2025 | Accepted: 09.03.2025 | Published: 13.03.2025

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Abstract

Case Report

Background: HELLP syndrome (Hemolysis, Elevated Liver Enzymes, and Low Platelets) is a severe obstetric complication that affects approximately 1% of pregnancies but occurs in 10–20% of cases with preeclampsia. One of its most serious complications is spontaneous hepatic hematoma, which can lead to life-threatening liver rupture in rare cases. Early diagnosis and timely intervention are crucial for improving maternal and fetal outcomes. **Case Presentation:** We report the case of a 35-year-old gravida 3 para 2 woman at 35 weeks of gestation who presented with persistent upper abdominal pain. She was diagnosed with HELLP syndrome, and an emergency cesarean section was performed due to acute fetal distress. During surgery, the patient developed hemodynamic instability, prompting an exploratory laparotomy, which revealed a ruptured hepatic subcapsular hematoma with active bleeding. Surgical management involved peri-hepatic packing and massive transfusion. Postoperatively, the patient was closely monitored in the intensive care unit, where she showed progressive clinical and biochemical improvement. She was successfully discharged on the twelfth postoperative day and exhibited a favorable recovery at follow-up. **Conclusions:** Although rare, hepatic hematoma and capsular rupture are life-threatening complications of HELLP syndrome. Given the nonspecific clinical presentation, a high index of suspicion is essential for early diagnosis. Imaging studies should be promptly performed in suspected cases, and when rupture occurs, immediate surgical intervention is necessary to optimize outcomes.

Keywords: HELLP Syndrome, Hepatic Hematoma/Rupture, Preeclampsia, Obstetric Complication, Emergency Cesarean Section.

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INTRODUCTION

HELLP syndrome (Hemolysis, Elevated Liver Enzymes, and Low Platelets) is a serious obstetric complication that affects approximately 1% of pregnancies. However, its prevalence rises significantly to 10–20% among women diagnosed with preeclampsia [1]. One of the most severe complications associated with HELLP syndrome is spontaneous hepatic hematoma, which has been observed in nearly 39% of affected patients. In rare cases (0.5–2%), these hematomas can progress to liver capsule rupture, posing a substantial risk of mortality, with maternal and fetal fatality rates reported at 17% and 38%, respectively [2]. Diagnosing hepatic hematoma and capsular rupture in HELLP syndrome remains challenging due to its nonspecific clinical presentation. Consequently, in any

pregnant patient with HELLP syndrome, even subtle symptoms suggestive of liver involvement should prompt immediate evaluation and intervention.

We report a case of a 35-year-old woman with HELLP syndrome who was suspected and finally diagnosed with spontaneous hepatic rupture during an emergency cesarean delivery. Early diagnosis and rational clinical judgment led to a better outcome.

CASE REPORT

A 35-year-old woman, gravida 3 para 2(G3P2) at 35 weeks of gestation, with no significant medical history or complications in her previous pregnancies. She presented to the emergency with persistent upper abdominal pain for four days, which had intensified over

Citation: Chikhi Brahim, Hmadate Ilyass, Aarjouni Youssef, Fakri Ahmed, Bensghir Mustaph, Balkhi Hicham. Undiagnosed Subcapsular Liver Hematoma Rupture in HELLP Syndrome Unveiled during Emergency Cesarean Delivery: Case Report. Sch J Med Case Rep, 2025 Mar 13(3): 420-425.

the past six hours. She reported no history of hypertension during pregnancy. Upon admission, her vital signs were within normal limits: blood pressure at 135/84 mmHg, heart rate at 80 bpm, temperature at 36.8°C and normal glycemia. On physical examination, tenderness was noted in the upper abdomen. A urine dipstick test revealed significant proteinuria (3+) with lower limb edema. The fetal heart rate was recorded at 150 bpm, with occasional palpable uterine contractions.

Laboratory tests revealed a platelet count of $56 \times 10^9/L$, red blood cell count of $4.37 \times 10^{12}/L$, and hemoglobin level of 10.1 g/dL. The prothrombin level was measured at 75%, while liver enzymes were

elevated, with alanine aminotransferase (ALT) at 195 UI/L and aspartate aminotransferase (AST) at 100 UI/L. Total bilirubin was 18 mg/L, with direct bilirubin at 5 mg/L. Lactate dehydrogenase (LDH) was recorded at 467 U/L, haptoglobin at 0.4g/L, creatinine at 7 mg/L, and urea at 0.18 g/L. Serum potassium was within normal limits at 4.2 mmol/L. Thyroid function testing showed a normal ultra-sensitive TSH level of 2.2 mIU/L. Based on this clinical presentation, a diagnosis of HELLP syndrome was established.

The patient underwent an abdominal ultrasound that showed no abnormalities (**Figure 1**).



Figure 1: Liver ultrasound of the parturient in the emergency department. The size and shape of the liver were normal, the liver surface was smooth, and the liver margin was sharp, which indicated that there was no subcapsular liver hematoma upon admission

Shortly after admission to the obstetrics unit, Doppler assessment revealed fetal bradycardia, accompanied by episodes of hyperactive fetal movements. In light of the confirmed diagnosis of HELLP syndrome and signs of acute fetal distress, an emergency cesarean section was promptly indicated to ensure maternal-fetal safety.

Upon entering the operating room, the parturient was immediately monitored which showed a blood pressure of 97/58 mmHg, a heart rate of 129 beats per minute and oxygen saturation at 99% on room air. As the decision to proceed with surgery was made, we promptly notified the transfusion department to prepare red blood cells, platelets, and fresh frozen plasma. A hemostasis test was performed, and the neonatologist arrived to prepare for the newborn's resuscitation before the parturient was transferred to the operating room.

General anesthesia was prepared and conducted using a rapid sequence intubation (RSI) technique. Equipment for difficult intubation, including a videolaryngoscope, a bougie and supraglottic airway

devices, was prepared in advance. After the loss of consciousness, cricoid pressure was applied, and endotracheal intubation was performed using a videolaryngoscope due to anticipated airway difficulties. Once successful intubation was confirmed by capnography and bilateral lung auscultation, mechanical ventilation was initiated, and the cesarean section began immediately.

Two minutes after anesthesia induction, the fetus was delivered with a birth weight of 1620 g. However, the newborn exhibited no spontaneous breathing or crying. Immediate neonatal resuscitation was initiated tracheal intubation was performed. At 4 minutes of life, the Apgar score was recorded at 4 (1 point each for heart rate, skin color, reflex response, and muscle tone). With continued resuscitative measures, the neonate's condition improved, and the Apgar score reached 10 by the 10th minute. The newborn was subsequently stabilized and transferred to the neonatology department for ongoing monitoring and specialized care.

However, during the closure of the cesarean section incision, the obstetrician noticed a significant and persistent flow of non-clotting blood from the surgical site. The patient's condition deteriorated rapidly, with her blood pressure dropping to 82/43 mmHg and her heart rate escalating to 146 beats per minute. The results of the hemostasis test revealed a severely reduced platelet count of $36 \times 10^9/L$, a hemoglobin level of 7.5 g/dL, and a prothrombin level of 65%. Given the parturient's preoperative upper abdominal pain and the confirmed diagnosis of HELLP syndrome, there was a strong suspicion of liver capsule rupture. To stabilize the patient, we promptly administered 1g of tranexamic acid to enhance coagulation. A continuous infusion of noradrenaline was initiated through a peripheral line to support hemodynamic stability. Simultaneously, a second external jugular line was secured to facilitate the rapid transfusion of 2 units of packed red blood cells along with 1000 mL of saline solution for volume resuscitation. Recognizing the need for more advanced hemodynamic monitoring and better vascular access, a central venous catheter was placed in the right internal jugular vein, followed by the insertion of a right radial arterial line to enable continuous blood pressure monitoring and arterial blood gas analysis.

Recognizing the severity of the situation, the surgeon extended the incision to a median laparotomy for better access and control of the bleeding. Upon exploration, a large subcapsular hematoma measuring approximately 10 cm in diameter was discovered on the right lobe of the liver. The hematoma had ruptured, leading to active hemorrhage from the liver capsule. The surgeon applied a peri-hepatic gauze packing, which was maintained until no active bleeding was observed. Given the risk of postoperative hemorrhage, an abdominal drainage tube was placed to monitor ongoing blood loss and detect any early signs of rebleeding. In response to significant blood loss, massive transfusion protocol was initiated, the patient received 4 units of packed red blood cells, 4 units of platelet concentrate, and 4 units of fresh frozen plasma. Fluid resuscitation was supplemented with nearly 1000 mL of Ringer's solution. As a result, the patient's hemodynamic status improved markedly, and the need for noradrenaline support was successfully weaned following hemorrhage control and transfusion. Urine output was closely monitored, totaling approximately 500 mL, indicating adequate end-organ perfusion. Following successful surgical intervention

and stabilization, the patient was transferred to the intensive care unit for close monitoring, and further management of her critical condition.

On the first postoperative day, a bedside ultrasound revealed hypoechoic material surrounding the liver and a small amount of free fluid in the upper right abdominal cavity. Laboratory tests showed the following values: ALT 180.4 U/L, AST 225.0 U/L, total bilirubin (TB) 22 mg/L, direct bilirubin (DB) 7 mg/L, hemoglobin 10.1 g/dL, platelet count $46 \times 10^9/L$, prothrombin at 51%, and fibrinogen at 4.06 g/L. The abdominal drainage tube output was recorded at 190 ml. While in the ICU, the patient received comprehensive supportive care, including broad-spectrum antibiotics, tranexamic acid to enhance coagulation, and transfusions of red blood cells, fresh frozen plasma, and platelet concentrate to correct ongoing coagulopathy. Additionally, intravenous nutrition was administered to support recovery. By the fourth postoperative day, her condition had stabilized significantly. The peri-hepatic gauze packing was carefully removed, confirming the absence of active bleeding. The patient was successfully extubated and transferred from the ICU back to the obstetrics department for further recovery. Liver function markers showed gradual improvement, reflecting progressive healing. On the seventh postoperative day, follow-up blood tests indicated significant recovery, with ALT reduced to 55.4 U/L, AST to 79.1 U/L, LDH to 180.5 U/L, hemoglobin rising to 11.0 g/dL, RBC count at $3.19 \times 10^{12}/L$, platelets increasing to $120 \times 10^9/L$, and prothrombin activity improving to 87%. A computed tomography (CT) scan still showed the presence of a subcapsular hematoma with mild gas accumulation, though no signs of active bleeding were observed (**Figure 2**). Meanwhile, the volume of abdominal drainage fluid progressively declined. By the tenth postoperative day, drainage had nearly ceased, allowing for safe removal of the abdominal drainage tube. With continued clinical improvement, the patient was discharged in stable condition on the twelfth postoperative day. At the 30-day follow-up, the patient reported no significant discomfort, and her overall health had returned to baseline. A follow-up abdominal ultrasound performed three months post-surgery showed that the subcapsular hematoma had been almost completely absorbed, confirming a favorable recovery (**Figure 3**).



Figure 2: Computed tomography (CT) scan of the parturient on postpartum day 7. Subcapsular hematoma on the surface of the right lobe of the liver. a: Subcapsular hematoma of the liver; b: Abdominal gas accumulation; c: Abdominal drainage tube

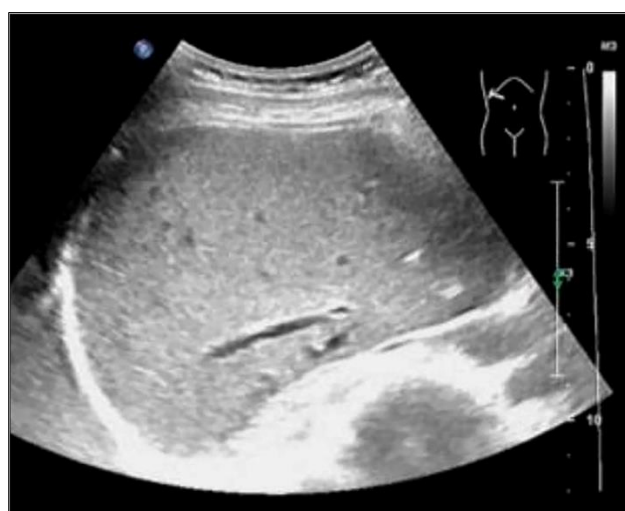


Figure 3: Liver ultrasound of the patient, performed 3 months after surgery. The liver surface was smooth, the liver margin was sharp, and no obvious lesions were found where the hematoma had been detected by computed tomography scan during hospitalization, suggesting that the patient's subcapsular liver hematoma and gas accumulation had been completely absorbed

DISCUSSION

HELLP syndrome is a rare but life-threatening pregnancy complication. Many investigators consider HELLP syndrome as a form of preeclampsia, and guidelines about HELLP syndrome are almost always included in the category of hypertensive disorders of pregnancy (HDP). However, the relationship between the two diseases is still controversial. In fact, 30% of cases defined as HELLP syndrome have no HDP, and some of the women with HELLP syndrome do not exhibit typical preeclampsia [3]. Multipara, older age (over 35 years old), obesity, HELLP syndrome history, and severe preeclampsia are high risk factors for HELLP syndrome. The pathogenesis of HELLP syndrome is still

unclear, but immunological dysfunction, vasospasm, endothelial injury, or vascular fibrin deposition might be involved in the disease progression [4]. Symptoms of HELLP syndrome can be atypical, which make it difficult to be discovered and diagnosed. Most common symptoms are right upper quadrant or epigastric pain and headache, followed by nausea and vomiting, visual changes, malaise, and swollen feet and ankles [5, 6].

HELLP syndrome is a rare but potentially life-threatening complication of pregnancy. While many researchers classify it as a variant of preeclampsia, it remains a subject of debate. Current guidelines typically include HELLP syndrome within the category of

hypertensive disorders of pregnancy (HDP). However, the exact relationship between these conditions is still unclear. Notably, approximately 30% of HELLP syndrome cases occur without HDP, and some affected women do not exhibit the classic signs of preeclampsia [3]. Several risk factors increase susceptibility to HELLP syndrome, including multiparity, maternal age over 35, obesity, a history of HELLP syndrome, and severe preeclampsia. Although its precise pathogenesis remains uncertain, mechanisms such as immune system dysfunction, vasospasm, endothelial injury, and fibrin deposition in blood vessels have been proposed to contribute to disease progression [4]. The symptoms of HELLP syndrome are often non-specific, making early diagnosis challenging. The most frequently reported symptoms include right upper quadrant or epigastric pain and headache, followed by nausea, vomiting, visual disturbances, general malaise, and peripheral edema [5, 6].

As highlighted in our case, hepatic capsular rupture is a serious complication of HELLP syndrome. The clinical presentation can be misleading, with symptoms such as abdominal pain, epigastric pain, anemia, right shoulder pain, or, in severe cases, hypovolemic shock. Given these risks, any HELLP syndrome patient exhibiting one or more of these symptoms should be carefully evaluated for potential liver hematoma [1], particularly in the presence of significantly reduced platelet counts [2]. Imaging studies are crucial for diagnosis. In our case, the initial abdominal ultrasound performed in the emergency department did not reveal any subcapsular hematoma. However, during hospitalization and preoperative preparation, a follow-up ultrasound was not conducted. By the time the patient was taken to the operating room, her blood pressure had already dropped, a critical detail that was overlooked. At that point, an undiagnosed subcapsular hematoma with active bleeding may have already developed. It has been documented that increased intra-abdominal pressure from vomiting, constipation, trauma, or compression can trigger hematoma formation and rupture [7]. Therefore, in any suspected case of subcapsular liver hematoma, an immediate ultrasound should be performed for confirmation. If needed, more sensitive imaging techniques such as computed tomography (CT) or magnetic resonance imaging (MRI) should be employed to assess the extent of the hematoma and detect liver rupture [8].

For hemodynamically stable patients, conservative management may be appropriate, including supportive care, fluid resuscitation, and transfusion of blood products [9]. However, in cases where the hematoma progresses, hemodynamic instability occurs, or liver rupture is confirmed, surgical intervention becomes necessary. Surgical options include packing, compression hemostasis, and suturing of the liver capsule and parenchyma. Another viable approach is to

limit blood flow to the affected area, such as through surgical ligation of the hepatic artery [10]. Transcatheter arterial embolization is also an effective and minimally invasive alternative, but potential ischemic complications, including liver dysfunction, must be considered [11]. In extreme cases, where there is extensive hepatic necrosis and irreversible liver failure, liver transplantation may be required, although its benefits remain controversial [12].

CONCLUSIONS

Although rare, hepatic hematoma and hepatic capsular rupture in women with HELLP syndrome require sufficient attention. Imaging examinations can assist in diagnosis, especially for women with symptoms or signs. When hepatic capsular rupture or hemodynamic instability occurs, decisive surgical treatment is necessary. In selected cases, liver transplantation might also benefit patients.

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