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Case Report

# Subcapsular Liver Hematoma Complicating HELLP Syndrome in a 27 Week Gravid Patient: Induction of Labor for Conservative Non-Operative Management

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## Abstract

Subcapsular liver hematoma (SLH) is a rare but life-threatening complication of hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome. We present a case of conservatively managed SLH in association with HELLP syndrome via induction of labor followed by close postpartum surveillance. This case report highlights the importance of maintaining a high suspicion for SLH in women with HELLP syndrome presenting with nonspecific abdominal pain. It additionally emphasizes the need for early diagnostic imaging based on symptoms rather than laboratory results alone, and why expectant management is preferred over premature surgical intervention when liver capsule remains intact to minimize maternal mortality.

***Keywords:*** *Subcapsular Liver Hematoma (SLH); Hemolysis, Elevated Liver Enzymes, and Low Platelets (HELLP); Induction of Labor*

## Introduction/Background

Hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome is a unique variant of preeclampsia involving hepatocellular damage and occurring in only 0.2 - 0.6% of all pregnancies [1]. Subcapsular liver hematoma (SLH) is a rare but life-threatening complication of HELLP syndrome occurring in < 1% of HELLP patients [2] but holding a mortality rate of 17 - 59%. Prompt diagnosis of SLH and intact liver capsule at the time of diagnosis are paramount to maternal survival. Initial presentation can vary and findings are often nonspecific which can make recognition of the condition difficult. Once the liver capsule has ruptured, a patient will often present in hemorrhagic shock and the condition becomes much harder to treat. It is important that practitioners maintain a high suspicion of SLH in HELLP syndrome patients to optimize patient outcome and recognize the diagnosis prior to liver capsule rupture.

## Case Presentation

A 25-year-old gravida 1 para 0 Caucasian woman at 27 weeks and 3 days gestation presented to triage with shortness of breath and lower extremity edema that started 12 hours prior to arrival. Her symptoms were constant, existed at rest, and worsen when she laid flat. She also mentioned bilateral upper abdominal pain, right-mid upper back pain, and a "GI bug" consisting of nausea, vomiting and diarrhea that onset a few days ago. She stated she was feeling the baby move however it was less than usual. She denied any preterm contractions,

leak of fluid, or vaginal bleeding. Her pregnancy had been complicated by asthma with no recent exacerbations, anxiety requiring no medications, and obesity (body mass index of 38 kg/$m^2$).

Upon initial evaluation, the patient was visibly short of breath with a blood pressure in the 140 - 150s/90 - 100s mmHg which was new for her pregnancy. She was tachycardic (100 - 110 bpm), tachypneic (40 breaths/min), and hypoxic ($O_2$ sat 91% on room air). On physical examination her lungs were clear however she was significantly tender to palpation in the right upper quadrant in her abdomen without rebound or guarding. Fetal strip was Category II due to intermittent variable decelerations which resolved spontaneously without resuscitation. Moderate variability and regular accelerations were present throughout fetal tracing.

A preeclampsia panel was ordered (Table 1) revealing anemia, thrombocytopenia, transaminitis, and leukocytosis. Electrocardiogram showed sinus tachycardia, and bedside Focused Assessment with Sonography for Trauma scan revealed maternal ascites. Pulmonary Computed Tomography (CT) angiogram successfully ruled out a pulmonary embolism, however it did show small bilateral pleural effusions and a perihepatic complex fluid collection in the anterior liver which was suspicious for a subcapsular hematoma.

| Lab Values | HD#1 | HD#2 | HD#3 | HD#4 | HD#5 | HD#6 | HD#7 | 16 days PP |
|---|---|---|---|---|---|---|---|---|
| Hemaglobin (g/dL) | 9.3 | 8.2 | 7.8 | 7.1 | 7.1 | 7 | 6.9 | 8.7 |
| White blood cells (x10^9/L) | 20 | 19.8 | 22 | 18.9 | 15.4 | 14.5 | 15.7 | 9.4 |
| Platelets (x10^9/L) | 138 | 149 | 251 | 269 | 276 | 321 | 387 | 543 |
| Creatinine (umol/L) | 0.7 | 0.5 | 0.5 | 0.5 | 0.5 | 0.5 | 0.5 | 0.5 |
| LDH (UA) | 871 | 309 | | | | | | |
| Alkaline phosphatase (IU/L) | 195 | 174 | 161 | 226 | 203 | 175 | 155 | 140 |
| ALT (IU/L) | 438 | 293 | 263 | 262 | 185 | 132 | 96 | 22 |
| AST (IU/L) | 191 | 90 | 126 | 121 | 62 | 33 | 20 | 22 |
| PT (seconds) | 9.5 | 9.8 | 9.5 | | | | | |
| INR | 0.8 | 0.9 | 0.9 | | | | | |
| PTT (seconds) | 27 | 27.6 | 19.1 | | | | | |
| Fibrinogen (mg/dL) | 515 | | 496 | | | | | |
| Urine Protein/Creatinine | 2.15 | | | | | | | |

***Table 1:*** *Laboratory values trended from hospital day 1 through postpartum day 16 demonstrate HELLP syndrome with elevated liver enzymes, lactate dehydrogenase, and low platelets. Complete resolution and normal values were observed on postpartum day 16.*

Given the above findings, the patient was diagnosed with HELLP syndrome complicated by SLH and the decision was made to initiate transfer to a tertiary care center for further management. Given decreased fetal movement and Category II fetal tracing, a biophysical profile was ordered and resulted 8/8. Prior to transfer, the patient was given her first dose of betamethasone for fetal lung maturity and started on magnesium sulfate for seizure prophylaxis and neuroprotection.

Upon arrival at the receiving facility, a CT scan confirmed the diagnosis of a right lobe SLH measuring 27.8 cm x 18.0 cm x 4.4 cm as well as multiple small adjacent liver lacerations. Fetal tracing strip remained Category II and cervical exam was closed, 0% effacement, -3 station. Induction was initiated with a cervical foley and Pitocin due to increased risk of disseminated intravascular coagulation (DIC) with surgery considering maternal disease. Four units of packed red blood cells were typed and cross-matched in the event of acute maternal decline. Twelve hours after induction was initiated, a healthy infant was delivered vaginally weighing 860g and reassuring APGAR scores.

Following the delivery, gastroenterology and general surgery were both consulted and recommended expectant management with close supervision. Serial labs were ordered every six hours and showed significant improvement by postpartum day 5. Patient was cleared for discharge with close follow-up. By postpartum day 10 her hematology and liver profile had normalized. Serial right upper quadrant ultrasounds demonstrated slow but continuous improvement of her SLH, measuring 22.4 cm by 2 weeks postpartum and 12.9 cm by 6 weeks postpartum. Monthly imaging was continued until complete resolution was noted.

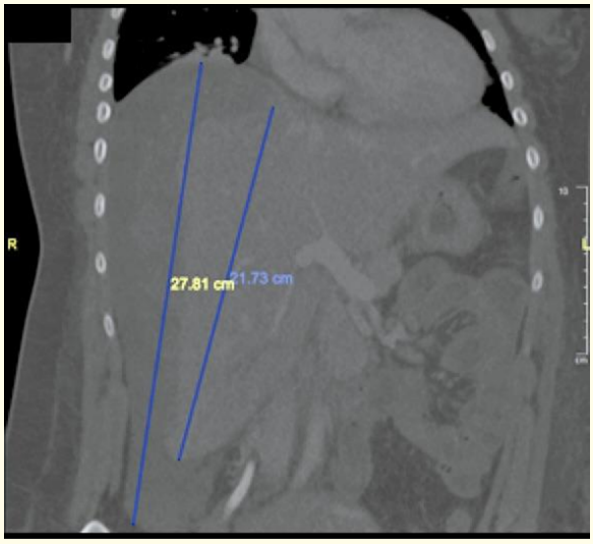


***Figure 1:*** *CT scan demonstrating the subcapsular liver hematoma from a para sagittal view.*

## Discussion

### Presentation, diagnosis, and differential for HELLP syndrome

Preeclampsia is estimated to complicate 5 - 8% of all pregnancies worldwide [3]. HELLP syndrome is a unique severe variant of preeclampsia that involves hepatocellular damage. While HELLP syndrome is rare, occurring in only 10 - 20% of patients with preeclampsia [2] and just 0.2 - 0.6% of pregnancies worldwide [1], its high rates of maternal morbidity and mortality make early diagnosis and treatment imperative to optimizing patient outcome. Formal diagnosis can be made in patients who meet the following criteria: hemolysis (lactate dehydrogenase > 600 U/L), thrombocytopenia (< 100,000 platelets/mcL), and transaminitis (alanine transaminase and/or aspartate transaminase > 70 U/L or 2x their normal value).

Over 90% of patients eventually diagnosed with HELLP syndrome will initially present with generalized malaise and viral-like syndrome. Such nonspecific symptoms often persist for several days prior to formal diagnosis. Other common symptoms on initial presentation include right upper quadrant or epigastric pain (40 - 90%), nausea and vomiting (29 - 84%), headache (33 - 61%), and/or visual changes (17%) [4]. Only 70% of HELLP patients will present in the antepartum period. Of the 30% of women who develop HELLP syndrome postpartum, 80% of those will have been diagnosed with a milder form of preeclampsia prior to delivery [4]. Onset of postpartum symptoms can take place anywhere from a few hours to seven days following delivery; however, the most frequent timing of diagnosis is within the first 48 hours postpartum. Practitioners should consider re-evaluating patients previously diagnosed with preeclampsia for

HELLP syndrome if new symptoms are to arise postpartum. In addition to the more common symptoms listed above, HELLP syndrome can also have an atypical presentation with 15% of patients lacking either hypertension or proteinuria [5]. Nonspecific findings ranging from GI, hematologic, or respiratory in nature combined with the absence of hypertension or proteinuria makes misdiagnosis with acute fatty liver of pregnancy (AFLP), acute cholecystitis, pancreatitis, or pulmonary embolism a real concern.

Complications of HELLP syndrome include but are not limited to DIC (15%), placental abruption (9%), acute pulmonary edema (6%), acute renal failure (3%), liver hemorrhage, liver failure, or SLH (1%), and lastly acute respiratory distress syndrome, sepsis, or stroke (< 1%) [4]. Patients with concomitant placental abruption, peripartum hemorrhage, or SLH are more likely to present with DIC than patients with HELLP syndrome alone.

## Subcapsular liver hematoma (SLH)

SLH, while rare, is considered a life-threatening complication of HELLP syndrome. Patients will often present with severe right upper quadrant or epigastric pain because of hepatic hemorrhage, parenchymal necrosis, and eventual stretching of Glisson's capsule. The extent of hepatic histopathologic findings does not always correlate with the severity of lab abnormalities, therefore SLH cannot be ruled out in a preeclamptic patient just because she has normal liver enzymes. Early imaging should always be considered in patients presenting with new onset right upper quadrant or epigastric pain unexplained by other etiologies, even in the absence of transaminitis, since onset of pain can often precede abnormal lab findings.

Conversely, women with preeclampsia will present with hepatic involvement as their severe feature in 10% of cases, the most common presentation being elevated liver enzymes without right upper quadrant or epigastric pain [2]. Severe transaminitis (≥ 1,000 - 2,000 IU/L) is uncommon in HELLP syndrome unless it is complicated by infarct or hematoma. A value that high should prompt imaging as well as evaluation for alternative diagnoses such as AFLP, abruption, DIC, viral hepatitis, and thrombotic thrombocytopenic purpura [4]. In addition to the standard complete blood count, basic metabolic panel, and liver function test, clinicians should also consider checking an lactate dehydrogenase (LDH) to further evaluate for SLH and/or liver infarct when pain is present. While LDH is not considered diagnostic, if elevated it can further support such suspicion. An LDH < 400 IU/L is more common with SLH, vs an LDH ≥ 10,000 - 20,000 IU/L which is seen with hepatic infarct [4].

There is no consensus on the imaging modality of choice when diagnosing SLH, however early recognition is crucial for reducing maternal mortality. Ultrasound allows for rapid diagnosis of hemoperitoneum at bedside in the unstable patient, however CT and Magnetic Resonance Imaging are more sensitive and therefore preferred for evaluating the extent of a hematoma and detecting liver rupture. It is important that clinicians choose the imaging method most readily available to minimize the risk of rupture.

Hepatic rupture, when present, is considered a surgical emergency and requires a skilled specialist to treat. Patients will often present with additional symptoms such as nausea and vomiting, shoulder pain, headache, peritoneal signs, and hypovolemic shock. Elevated blood pressure may be absent since major hemorrhage has already taken place.

## Management

In a hemodynamically stable patient with an unruptured SLH, conservative management with volume replacement, blood transfusions, correction of coagulopathy, and serial labs is recommended. Clinicians may also consider percutaneous embolization of the hepatic arteries. Close monitoring in the intensive care unit is required since rapid decline is likely if rupture occurs. Repeat ultrasound or CT should be performed to monitor the size of the hematoma and imaging should be followed until complete resolution. If labs and size of hematoma are stable, the patient may be discharged with close outpatient follow-up. Complete resolution of a SLH may take several months vs hepatic infarct which has often resolved by the time of re-imaging following delivery.

If rupture is suspected, surgical management with an experienced team in liver surgery is recommended. Surgical management may include evacuation of hematoma and temporary tamponade by packing with gauze, hepatic artery ligation, embolization of hepatic artery, Pringle Maneuver, or hepatic resection. Liver transplant should be considered in the presence of a refractory liver hemorrhage or rapidly progressing acute liver failure. Non-surgical management in hemodynamically stable patients with hepatic rupture and HELLP syndrome has been reported. Grand'Maison., *et al*. (2012) described the use of hepatic artery embolization in 6 of 9 cases of hepatic rupture associated with HELLP syndrome. Specifically, they reported 2 patients with hepatic rupture managed conservatively without intervention, 2 patients managed solely with hepatic artery embolization, and 3 patients managed with a combination of surgery and hepatic artery embolization. Compared with traditional surgical intervention, management including hepatic artery embolization significantly reduced maternal and fetal mortality rates [7].

## Conclusion

SLH is considered a life-threatening complication of HELLP syndrome. Clinicians must maintain a high suspicion for SLH in HELLP patients or patients presenting with right upper quadrant or epigastric pain. Prompt imaging should be ordered even if laboratory findings are normal and if SLH is noted, expectant management is preferred over surgery if the liver capsule remains intact.

## Declaration of Patient Consent

The authors certify they have obtained the verbal patient consent. The patient has given her verbal consent for her images and other clinical information to be reported in the journal. The patient understands her name and initial will not be published and due efforts will be made to conceal her identify, but anonymity cannot be guaranteed

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## Conflicts of Interest

There are no conflicts of interest.

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