HELLP SYNDROME AND ITS COMPLICATIONS: A CASE REPORT OF A GIANT SUBCAPSULAR HEPATIC HEMATOMA

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Abstract: Introduction: Subcapsular hepatic hematoma is a rare but life-threatening complication of hypertensive disorders of pregnancy, particularly severe preeclampsia and HELLP syndrome. (Saad et al., 2025) Although uncommon, it is associated with significant maternal and perinatal morbidity and mortality, especially in cases of delayed diagnosis or hepatic rupture. The underlying

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pathophysiology involves systemic endothelial dysfunction secondary to abnormal placentation, leading to an antiangiogenic state, hepatic microangiopathy, ischemia, and progressive subcapsular hemorrhage. (Kang et al., 2025) Case Presentation: We report the case of a 39-year-old woman with a pregnancy conceived via in vitro fertilization who developed preeclampsia with severe features, stage I fetal growth restriction, and incomplete HELLP syndrome. In the immediate postpartum period, she presented with severe right upper quadrant pain radiating to the shoulder and progressive elevation of liver enzymes. Abdominal computed tomography revealed a giant right-lobe subcapsular hepatic hematoma measuring approximately 20 cm in maximal diameter, without evidence of active arterial bleeding on CT angiography. Given hemodynamic stability, a conservative management strategy in the intensive care unit was chosen, including close monitoring and multidisciplinary support. Her course was complicated by transient hematological, metabolic, and respiratory dysfunctions, all managed medically. Serial imaging demonstrated gradual resolution, with complete radiological resolution at six weeks postpartum. Discussion: The lesion was consistent with a grade III hepatic injury according to the American Association for the Surgery of Trauma (AAST) classification, reflecting involvement of more than 50% of the hepatic surface. (Gupta et al., 2021) Clinical presentation is often nonspecific, making early recognition challenging. Computed tomography remains the diagnostic gold standard for assessing extent and ruling out active bleeding, while ultrasound may underestimate lesion severity. (Moore et al., 2018) Management depends primarily on hemodynamic status; in stable patients without rupture, conservative management in an intensive care setting with hemodynamic optimization, correction of coagulopathy, and serial imaging is considered safe and effective. (Chahine et al., 2025) Surgical or endovascular interventions, including hepatic artery embolization or abdominal packing, are reserved for cases of rupture or hemodynamic instability. (Augustin et al., 2022) Conclusion: Subcapsular hepatic hematoma associated with HELLP syndrome represents a rare but catastrophic obstetric emergency. Early diagnosis and individualized management are essential to improve outcomes. This case supports that even giant hepatic hematomas can be successfully managed conservatively in carefully selected hemodynamically stable patients, with favorable maternal outcomes.

Keywords: Subcapsular Hepatic Hematoma, HELLP Syndrome, Hepatic Rupture, and Preeclampsia with Severe Features

INTRODUCTION

Subcapsular hepatic hematoma is a rare and uncommon complication of severe preeclampsia, eclampsia, and HELLP syndrome, which typically occurs in the late second trimester and throughout the third trimester, including the immediate postpartum period within the first 24–48 hours. (Saad et al., 2025) However, cases have been reported up to six weeks after delivery. (Brito et al., 2021) It occurs in approximately 0.9–2% of pregnancies complicated by preeclampsia or HELLP syndrome, with maternal mortality rates of 10–15% and perinatal mortality rates of up to 41%, depending on the presence of rupture, timing of diagnosis, and availability of therapeutic interventions. (Kang et al., 2025) Reported risk factors include advanced maternal age, multiparity, obesity, and HELLP syndrome itself. (Saad et al., 2025)

The pathophysiology is explained as a consequence of abnormal placentation that triggers inflammatory changes in response to ischemia, resulting in generalized endotheliosis. (Brito et al., 2021) At the hepatic level, this process induces an imbalance between angiogenic factors such as vascular endothelial growth factor (VEGF) and anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1), leading to vasospasm and reduced hepatic blood flow. This results in initial intrahepatic vascular congestion, coagulation abnormalities, and increased fibrin deposition within hepatic capillaries, producing ischemia and hepatocellular injury with subsequent reduction in oxygen delivery. (Gupta et al., 2021) As the condition progresses, hepatic infarction and microvascular hemorrhages occur, leading to blood accumulation. Intrahepatic pressure increases, forming a subcapsular hematoma initially contained by Glisson's capsule; however, with ongoing bleeding and pressure elevation, hepatic rupture may occur, resulting in immediate hypovolemic shock and circulatory collapse. (Saad et al., 2025)

The right hepatic lobe is most commonly affected, and clinical manifestations are often vague and nonspecific, requiring a high index of suspicion for diagnosis. Symptoms may include epigastric or right upper quadrant pain radiating to the right shoulder or neck, dyspnea, pleuritic pain, nausea, and vomiting. It may be associated with pleural effusion, oliguria, blurred vision, and sudden changes in fetal heart rate patterns. (Kang et al., 2025) In cases of hepatic rupture, signs of hypovolemic shock, sudden onset of pain, and abdominal distension secondary to hemoperitoneum may be present. (Saad et al., 2025)

We present a case of a subcapsular hepatic hematoma diagnosed in the immediate postpartum period, associated with severe preeclampsia, stage I fetal growth restriction, and incomplete HELLP syndrome, which was successfully managed conservatively.

CASE PRESENTATION

A 39-year-old woman with a history of obesity, prior bilateral partial salpingectomy 10 years ago, and tubal recanalization 2 years ago, with obstetric history of gravida 3, para 2, with two living children, presented to the outpatient clinic at 29 weeks and 3 days of gestation according to her last menstrual period in her third pregnancy, conceived via in vitro fertilization.

At 28.1 weeks of gestation, she had been hospitalized for two days due to headache and elevated blood pressure values, during which she received fetal lung maturation with dexamethasone (4 doses of 6 mg intramuscularly every 12 hours). Preeclampsia was initially ruled out, and she was discharged. On the current presentation, she reported severe abdominal pain in the epigastrium and right upper quadrant, rated 9/10 on the visual analog scale, with radiation to the right shoulder, associated with blood pressure readings ranging from 139/95 to 150/88 mmHg, without clinical signs of vasospasm.

On physical examination, a gravid uterus was noted with a single live fetus in cephalic presentation, left occipito-transverse position, fundal height appropriate for gestational age, and no clinical uterine activity. Fetal heart rate was 142 bpm with preserved movements. Abdominal

examination revealed a soft abdomen with tenderness on deep palpation in the epigastrium and right upper quadrant. External genitalia were normal for a nulliparous appearance, vaginal examination was deferred, and lower limbs showed no edema. Deep tendon reflexes were 2/5. Electronic fetal monitoring was category I with no uterine contractions.

Obstetric ultrasound showed a single live fetus in cephalic presentation, left dorsal position, fetal heart rate of 159 bpm, male sex, biparietal diameter (BPD): 65 mm, head circumference (HC): 251 mm, abdominal circumference (AC): 227 mm, femur length (FL): 49 mm, and estimated fetal weight of 1003 g, corresponding to the 2nd percentile for gestational age. The placenta was anterior, homogeneous, with normal decidual-placental interface, and amniotic fluid measured by deepest vertical pocket was 3.97 cm.

Doppler studies showed umbilical artery pulsatility index (PI) of 1.07 (48th percentile, normal), middle cerebral artery PI of 1.20 (1st percentile, vasodilation), cerebroplacental ratio of 1.12 (2nd percentile, flow redistribution), ductus venosus PI of 0.54 (43rd percentile, normal), and mean uterine artery PI of 1.75 (>99th percentile, elevated). These findings were consistent with stage I fetal growth restriction (Figure I).

Laboratory tests showed: hemoglobin 13.1 g/dL (VR: 12.0–16.0 g/dL), hematocrit 38.8% (VR: 36–46%), platelets 228,000 K/µL (VR: 150,000–450,000 K/µL), prothrombin time 11.6 s (VR: 11–13.5 s), partial thromboplastin time 23.1 s (VR: 25–35 s), INR 1.06 (VR: 0.8–1.2), urea 22 mg/dL (VR: 15–40 mg/dL), creatinine 0.66 mg/dL (VR: 0.6–1.1 mg/dL), AST 54.7 U/L (VR: 0–40 U/L), ALT 56.4 U/L (VR: 0–41 U/L), LDH 224 U/L (VR: 140–280 U/L), indirect bilirubin 0.4 mg/dL (VR: 0.2–0.8 mg/dL), direct bilirubin 0.6 mg/dL (VR: 0.0–0.3 mg/dL), total bilirubin 1.0 mg/dL (VR: 0.3–1.2 mg/dL), tyrosine kinase 16,384.00 pg/mL (VR: assay-dependent, usually <20,000 pg/mL in late pregnancy), placental growth factor (PlGF) 15.50 pg/mL (VR: gestational-age dependent, typically decreased in preeclampsia), and sFlt-1/PlGF ratio 1057.03 (VR: <38 low risk; >85 high risk for preeclampsia).

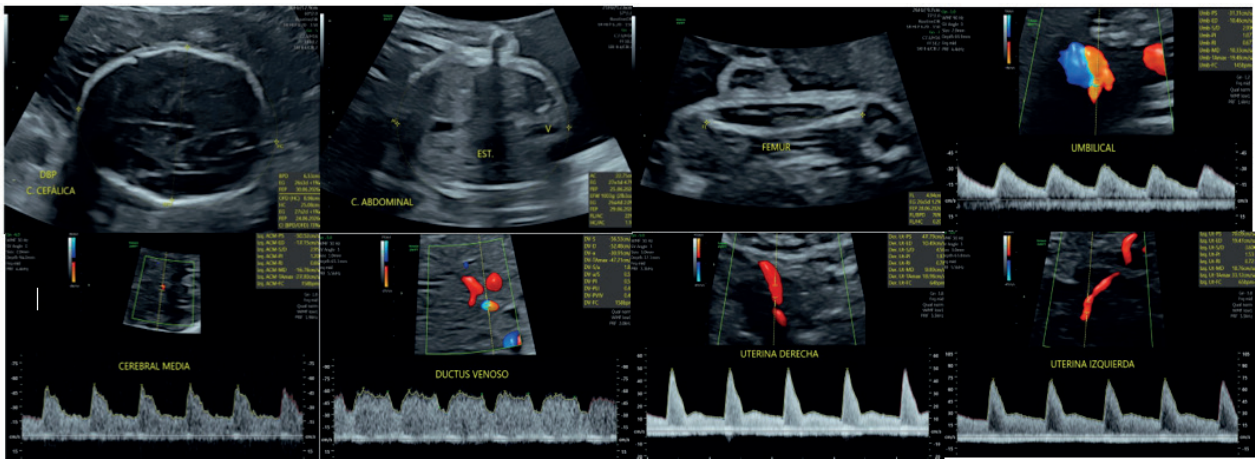

Figure 1. Obstetric ultrasound at admission A) Biparietal diameter (BPD): 65 mm. B) Abdominal circumference (AC): 227 mm. C) Femur length (FL): 49 mm. D) Umbilical artery pulsatility index (PI): 1.07 (48th percentile). E) Middle cerebral artery PI: 1.20 (1st percentile). F) Ductus venosus PI: 0.54 (43rd percentile). G) Right uterine artery PI: 1.97. H) Left uterine artery PI: 1.53. Mean uterine artery PI: 1.75 (>99th percentile).

TREATMENT

After correlating the patient's clinical presentation with laboratory findings, the diagnosis of preeclampsia with severe features associated with stage I fetal growth restriction was established. Therefore, the patient was admitted for delivery via cesarean section.

On admission, she presented persistently elevated blood pressure values >140/90 mmHg. Continuous electronic fetal monitoring demonstrated decreased variability with fetal bradycardia, consistent with category II tracing, which persisted despite intrauterine resuscitation maneuvers. Consequently, two intravenous lines were established, and intravenous crystalloids were initiated. Magnesium sulfate was administered with a loading dose of 4 g IV over 20 minutes, followed by a maintenance infusion of 1 g/hour. Nifedipine 10 mg was administered orally at that time. A Foley catheter was placed, prophylactic preoperative antibiotics were given, and intravenous gastric protection

was initiated. Two units of packed red blood cells were crossmatched and prepared, and an emergency cesarean section was indicated due to fetal compromise.

The procedure resulted in the delivery of a live male newborn in cephalic presentation, weighing 995 g, with a head circumference of 30 cm and length of 40 cm. Apgar scores were 7 and 8 at 1 and 5 minutes, respectively. Amniotic fluid was clear and of moderate volume. The placenta was anterior fundal, with approximately 20% abruption and abundant clots. The umbilical cord was noted to be small.

Intraoperatively, a Couvelaire uterus with uterine atony was identified despite uterotonic therapy; therefore, an obstetric hysterectomy was performed. The placenta, uterus, and adnexa were sent for histopathological analysis. Estimated blood loss was 500 mL.

Pathology findings

Uterus: No evidence of malignancy in the examined specimen.

Cervix: Decidual reaction.

Endometrium: Basal.

Myometrium: Hypertrophy secondary to pregnancy.

Placenta: Placenta weighing 200 g, measuring 13 × 9 cm. The maternal surface shows complete, lobulated cotyledons with deep sulci. The decidual (Nitabuch) membrane measures 0.1 cm. On serial sectioning, the parenchyma is wine-red with a spongy appearance. The fetal surface shows moderate to marked tessellation (++/+++), with an area of hemorrhagic material measuring 3.5 × 2 cm. The umbilical cord shows central insertion, measuring 21 × 1 cm. Membranes are slightly opaque, complete, grayish, with marginal insertion.

Histologically, there is delayed villous maturation, cytotrophoblastic hyperplasia and hypertrophy, segmental thickening of the villous basement membrane, and a reduction in vasculosyncytial membranes. The umbilical cord and membranes show no pathological alterations.

Results and Follow-up

At the end of surgery, the patient was transferred to the intensive care unit (ICU), where she developed hypotension requiring vasopressor support. She also presented with decreased breath sounds bilaterally, oxygen desaturation, and an increased oxygen requirement of 2 L/min via nasal cannula, along with oliguria.

Electrocardiography demonstrated peaked T waves, and arterial blood gas analysis was consistent with metabolic acidosis and moderate hyperkalemia. The patient additionally reported severe right upper quadrant abdominal pain.

Follow-up laboratory tests showed: hemoglobin 12 g/dL (VR: 12.0–16.0 g/dL), hematocrit 35% (VR: 36–46%), platelets 246,000 K/µL (VR: 150,000–450,000 K/µL), prothrombin time 11.9 s (VR: 11–13.5 s), partial thromboplastin time 23 s (VR: 25–35 s), INR 1.09 (VR: 0.8–1.2), urea 37.6 mg/dL (VR: 15–40 mg/dL), creatinine 1.28 mg/dL (VR: 0.6–1.1 mg/dL), AST 698 U/L (VR: 0–40 U/L), ALT 673 U/L (VR: 0–41 U/L), direct bilirubin 0.69 mg/dL (VR: 0.0–0.3 mg/dL), total bilirubin 1.09 mg/dL (VR: 0.3–1.2 mg/dL), fibrinogen 335.2 mg/dL (VR: 200–400 mg/dL), potassium (K^+) 7.57 mmol/L (VR: 3.5–5.1 mmol/L), and sodium (Na^+) 131.7 mmol/L (VR: 135–145 mmol/L).

Imaging studies were performed. Abdominal ultrasound revealed an elongated heterogeneous avascular lesion with poorly defined borders measuring 8.5 × 3.9 cm on color Doppler, associated with 460 cc of perihepatic fluid. This was further evaluated with non-contrast computed tomography of the abdomen and pelvis, which demonstrated a right hepatic lobe subcapsular hematoma measuring 20 cm with an estimated volume of 900 cc, as well as free fluid in the pelvic cavity and perisplenic region suggestive of hemoperitoneum.

CT angiography confirmed the diagnosis and excluded active arterial bleeding. Additional findings included bilateral pleural effusion. Chest X-ray revealed a right-sided pleural effusion (Figure 2).

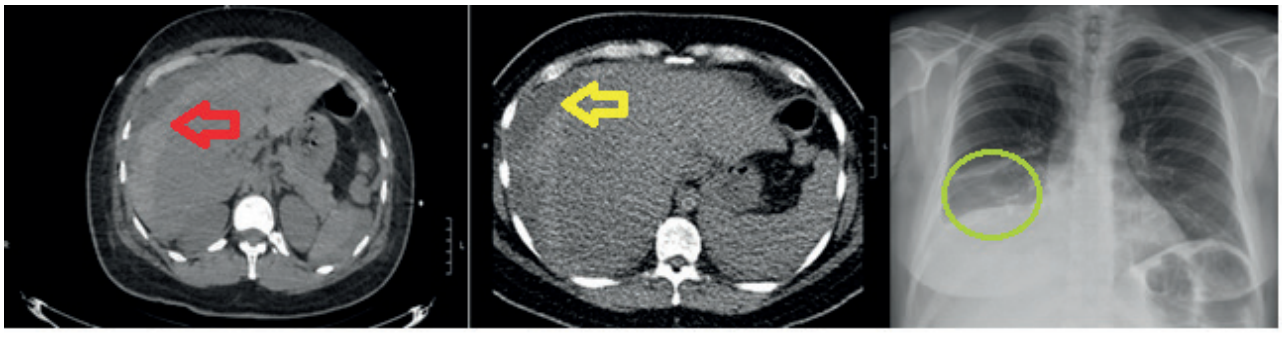

Figure 2. Postoperative imaging on postoperative day 1 A) Non-contrast abdominal and pelvic computed tomography demonstrating a right hepatic lobe subcapsular hematoma measuring 20 cm with an estimated volume of 900 cc, indicated by a red arrow. B) CT angiography showing a subcapsular hepatic hematoma without evidence of active arterial bleeding, indicated by a yellow arrow. C) Chest X-ray demonstrating a right-sided pleural effusion, indicated by a green circle.

Based on the findings from complementary studies, a multidisciplinary approach was initiated involving general surgery, nephrology, pulmonology, and internal medicine. The patient's working diagnoses included acute kidney injury (KDIGO stage II), moderate hyperkalemia, incomplete HELLP syndrome, subcapsular hepatic hematoma, acute hypoxemic respiratory failure (type I), and right-sided pleural effusion.

Conservative management of the hepatic hematoma was initially selected due to the absence of active bleeding on imaging and the patient's hemodynamic stability. The patient received 24 hours of magnesium sulfate infusion at 1 g/hour, low-dose intravenous fluids, and antihypertensive therapy according to protocol with scheduled labetalol and nifedipine administration. Hyperkalemia was managed with salbutamol and regular insulin therapy, along with loop diuretics and calcium gluconate. Analgesia was provided as needed.

Perioperative antibiotic prophylaxis with cefazolin was administered for three doses. Additional supportive measures included respiratory therapy and the use of compression stockings. The patient

also received one unit of packed red blood cells and one unit of fresh frozen plasma.

During postoperative days 2 and 3, the patient showed clinical improvement with reduction of right upper quadrant pain. However, follow-up laboratory tests revealed severe anemia and hematologic deterioration, with hemoglobin 7 g/dL (VR: 12.0–16.0 g/dL), hematocrit 21.4% (VR: 36–46%), platelets 63,000 K/µL (VR: 150,000–450,000 K/µL), prothrombin time 17.6 s (VR: 11–13.5 s), partial thromboplastin time 30.3 s (VR: 25–35 s), INR 1.64 (VR: 0.8–1.2), AST 3,639 U/L (VR: 0–40 U/L), ALT 4,800 U/L (VR: 0–41 U/L), urea 47.5 mg/dL (VR: 15–40 mg/dL), creatinine 0.78 mg/dL (VR: 0.6–1.1 mg/dL), potassium 4.09 mmol/L (VR: 3.5–5.1 mmol/L), and sodium 137.3 mmol/L (VR: 135–145 mmol/L). Based on these findings, transfusion of two units of packed red blood cells and two units of fresh frozen plasma was indicated, and hyperkalemia-targeted therapy was discontinued.

From postoperative day 4 onward, progressive clinical and laboratory improvement was observed, with increasing hemoglobin and platelet counts and a marked decrease in transaminase levels. The patient continued to report mild right upper quadrant pain, which remained controlled with analgesia. Breath sounds remained decreased, predominantly on the right side, and she continued on supplemental oxygen at 2 L/min. The abdomen remained soft with mild tenderness in the right upper quadrant. Vaginal bleeding was minimal, and urine output remained adequate. Serial ultrasound evaluations showed a stable subcapsular hepatic hematoma without evidence of progression.

On postoperative day 6, the patient was transferred from the intensive care unit to the inpatient ward after evaluation by the Infectious Diseases service, which recommended intravenous ampicillin-sulbactam 3 g every 6 hours for 7 days. In the general ward, she remained hospitalized for an additional 8 days, during which medical management was optimized, including scheduled antihypertensive therapy with labetalol and nifedipine, low-volume intravenous fluids, respiratory expansion exercises, abdominal care measures, loop diuretics, compression stockings, antibiotic therapy, analgesia, and digestive enzymes. Thromboprophylaxis with enoxaparin 40 mg was initiated during the last 4 days of hospitalization.

During this period, the patient showed progressive clinical improvement, with resolution

of abdominal pain and improved respiratory mechanics. Oxygen supplementation was successfully discontinued, and no hypertensive crises requiring intravenous labetalol were observed. Follow-up ultrasound studies demonstrated no increase in the size of the subcapsular hematoma, which remained stable until discharge.

The patient was ultimately discharged in stable condition with continuation of oral antibiotic therapy (amoxicillin 500 mg every 12 hours for 7 additional days) and antihypertensive treatment with enalapril 10 mg every 12 hours plus carvedilol 3.125 mg every 12 hours for 15 days until follow-up. At 6-week follow-up, abdominal computed tomography demonstrated complete resolution of the subcapsular hepatic hematoma, and the patient was discharged from surgical care.

DISCUSSION

In the present case, the patient developed a giant subcapsular hepatic hematoma classified as grade III according to the American Association for the Surgery of Trauma (AAST), as it involved more than 50% of the hepatic surface. (Moore et al., 2018) This entity is most frequently associated with hypertensive disorders of pregnancy, particularly preeclampsia with severe features and HELLP syndrome. (Saad et al., 2025)

For diagnosis, it is essential to correlate often nonspecific clinical manifestations with laboratory findings. Elevated levels of liver enzymes such as ALT, AST, LDH, and uric acid have been associated with a significantly increased risk of severe maternal morbidity, reported to exceed 75% in high-risk obstetric populations. (Kang et al., 2025) Thrombocytopenia may progress rapidly, with platelet counts decreasing up to 40% per day, while hematocrit levels may decline. Therefore, the American College of Obstetricians and Gynecologists (ACOG) recommends serial laboratory monitoring at least every 12 hours in the postpartum period, considering that initial laboratory results may be within normal ranges, as observed in this patient, and may worsen within the first 48 hours postpartum. Hepatic enzymes typically begin to normalize or decrease by the fourth postpartum day. (Chahine et al., 2025)

Imaging plays a fundamental role in diagnosis. While focused assessment with sonography for trauma (FAST) and ultrasound are useful initial tools, they may fail to detect posterior hepatic lesions. Computed tomography and magnetic resonance imaging provide higher sensitivity for detecting hepatic rupture and accurately assessing the size and extent of the hematoma. (Augustin et al., 2022)

Regarding management, open surgical approaches were historically considered the standard treatment for subcapsular hepatic hematomas. However, laparotomy in this context may increase intra-abdominal pressure and precipitate further bleeding, particularly in an inflamed and friable liver, making surgical access technically challenging. For this reason, alternative strategies have been proposed depending on whether the hematoma is ruptured or contained. (Raja et al., 2026)

In cases without rupture, conservative management is recommended, ideally initiated in an intensive care unit during the first 48 hours for hemodynamic stabilization. This includes fluid resuscitation, correction of coagulopathy with cryoprecipitate to maintain fibrinogen >2 g/L, administration of 2–4 units of fresh frozen plasma to maintain INR <1.5, platelet transfusion when counts are <20,000/μL, and red blood cell transfusion when hemoglobin is <7 g/dL. (Kang et al., 2025) Additional measures include antibiotic therapy, frequent laboratory monitoring every 6–12 hours, and serial ultrasound evaluation every 48 hours. (Saad et al., 2025) In addition, maneuvers that increase intra-abdominal pressure should be avoided to reduce the risk of rupture.

Surgical intervention is reserved for patients with disseminated intravascular coagulation refractory to transfusion, persistent hypovolemic shock despite vasopressor support, or failed resuscitation. In cases of ruptured hematoma, management may include minimally invasive procedures such as hepatic artery embolization in hemodynamically stable patients, followed by laparoscopic evacuation or drainage, abdominal packing, vascular ligation of portal or hepatic venous branches, omental patching, use of hemostatic materials, partial hepatectomy, and, in cases of acute liver failure or refractory coagulopathy, liver transplantation. (Sucan et al., 2025)

Postpartum follow-up is recommended with imaging at one week and at six weeks, although complete resolution may take several months. (Zhou et al., 2024)

In the present case, conservative management in the ICU was selected due to the absence of active bleeding on imaging and hemodynamic stability. The patient received cautious fluid therapy, blood product transfusion, analgesia, abdominal protective measures, and strict blood pressure control. Magnesium sulfate therapy was completed, and low-molecular-weight heparin was later initiated due to the postpartum thrombotic risk and immobilization, given the absence of significant bleeding risk. The patient evolved favorably, with complete radiological resolution of the hematoma at one-month follow-up and subsequent discharge from surgical care. (Albuquerque et al., 2025)

CONCLUSION

Subcapsular hepatic hematoma is a rare but potentially life-threatening complication associated with hypertensive disorders of pregnancy, particularly HELLP syndrome. Its clinical presentation is often nonspecific, which may delay diagnosis and increase the risk of hepatic rupture and hypovolemic shock.

In this context, imaging modalities such as computed tomography and magnetic resonance imaging are essential for definitive diagnosis. Management should be individualized, as conservative treatment represents a safe and effective option in hemodynamically stable patients, even in cases of large or giant hematomas.

However, the presence of complications such as hemoperitoneum, clinical deterioration, or hemodynamic instability necessitates surgical or interventional management. This case highlights the importance of a high index of clinical suspicion, early diagnosis, and a multidisciplinary approach to optimize maternal outcomes. Furthermore, it demonstrates that giant subcapsular hepatic hematomas can be successfully managed with conservative strategies in carefully selected patients, in agreement with current evidence.

Conflict of interest

The authors declare no conflicts of interest related to the publication of this article. All authors participated in the conception, writing, design, and analysis of this manuscript. The authors further declare that no financial or institutional support was received from any entity that could influence the validity or interpretation of the presented findings.

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