International Journal of Reproduction, Contraception, Obstetrics and Gynecology
*Borges AC et al. Int J Reprod Contracept Obstet Gynecol. 2023 May;12(5):1459-1464*
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20231242

**Case Report**

# Postpartum subcapsular liver hematoma: a rare complication successfully managed with conservative treatment

**Ana Catarina Borges*, Rita Almendra, Cristina Nogueira-Silva, Cátia Correia**

Department of Obstetrics and Gynecology, Hospital de Braga, Braga, Portugal

**Received:** 14 March 2023
**Accepted:** 06 April 2023

***Correspondence:**
Dr. Ana Catarina Borges,
E-mail: anacatarinasborges@gmail.com



## ABSTRACT

Subcapsular liver hematoma is a spontaneous bleeding between the Glisson's capsule and the liver parenchyma. It is a rare but serious complication of severe preeclampsia and/or HELLP (hemolysis, elevated liver enzyme and low platelets) syndrome that can have devastating consequences, the worst being hepatic rupture with maternal death. A young nulliparous woman, at 34 weeks 'gestation, was admitted for evaluation of new onset hypertension. The pregnancy was uneventful, yet at 32 weeks' gestation a severe fetal growth restriction was diagnosed. She presented a high urine protein to creatinine ratio, then a pre-eclampsia without severity criteria was diagnosed. At day 3 of hospitalization, she presented with intense epigastric pain and vaginal blood loss, so an emergency c-section was performed with the suspicion of placental abruption. Eighteen hours postpartum she complained of intense diffuse abdominal pain radiating to the right shoulder blade. Arterial hypotension and obtundation was observed. A computerized tomography was performed and revealed the presence of a bulky perihepatic hematoma measuring 12×9×21 cm. The patient went into hemorrhagic shock and so massive hemorrhage protocol was activated. After a multidisciplinary discussion a conservative management was decided. Clinical and analytical improvement was observed, and she was discharged home on postpartum day-27. The diagnosis of subcapsular liver hematoma is challenging, but it is essential to be done in a timely manner. Thus, a high index of suspicion, a prompt diagnosis and a multidisciplinary approach are the key factors for a successful outcome.

**Keywords:** Pregnancy, Pre-eclampsia; Liver disease, Hematoma, Conservative treatment

## INTRODUCTION

Subcapsular liver hematoma (SLH) is a spontaneous bleeding between the Glisson's capsule and the liver parenchyma. It is a rare but serious complication of severe preeclampsia and/or HELLP (hemolysis, elevated liver enzyme and low platelets) syndrome that can have devastating consequences, the worst being hepatic rupture with maternal death.[1] It is estimated that this entity complicates 1/40.000 to 1/250.000 pregnancies and 0.9% to 1.8% of HELLP syndrome cases.[1-3]

SLH occurs preferentially in the late second or third trimester of pregnancy although diagnosis can be delayed until the immediate postpartum period (15 to 30% of cases), generally within the first 48 hours.[3-5] The clinical presentation is usually nonspecific, and the most common presenting features are right upper quadrant or epigastric pain, shoulder pain, nausea, and vomiting.[2] Due to the lack of specificity regarding these symptoms, the variable clinical presentation and the low incidence of this pathology, diagnosis is often delayed. Even though mortality associated has decreased in recent years, it remains high for both the mother (17-86%), depending on whether the hematoma is ruptured, timely diagnosis and the availability of therapeutic options; and for the fetus (38-62%), usually related to prematurity and hypoxia.[1-3,6]

Therefore, given acute and critical nature of SLH, even with aggressive medical and/ surgical interventions,

outcome can be devastating, so it is crucial to establish diagnosis quickly.

We will report a case of a patient with preeclampsia that developed a SLH in the first 24 hours postpartum that was successfully managed with conservative treatment.

The report complies with the declaration of Helsinki and it was approved by the ethics committee for health research of Braga's Hospital. We were given informed consent from the patient to use the medical history and images.

## CASE REPORT

A woman in her late twenties, smoker and nulliparous, at 34 weeks´ gestation presented to our emergency department for evaluation of new onset hypertension in pregnancy with a blood pressure (BP) of 140/92 mmHg. Until 32 weeks, the pregnancy was uneventful. At that time a fetal growth restriction (FGR) was diagnosed with a fetus below 1st percentile. Her personal and family histories were unremarkable.

On admission the patient was without complaints, denied any occurrence of headache, visual changes, epigastric or right upper quadrant pain. Analytical study revealed a positive urine protein to creatinine ratio (P/C ratio) of 4.2 (Normal Range (NR)<0.3), aspartate transaminase (AST) 50 U/L (NR 12-40U/L), alanine transaminase (ALT) 43 U/L (NR 7-40U/L), without other relevant alterations (Table 1).

**Table 1: Evolution of analytical parameters.**

| Variables | Admission | Immediately after CS | CS + 2h | CS + 12h | CS + 18h | PP day 1 | PP day 3 | Discharge day (27) |
|---|---|---|---|---|---|---|---|---|
| **Hemoglobin (11.9-15.6 g/dL)** | 12.2 | 12.2 | 9.7 | 6.0 | 5.6 | 7.9 | 6.6 | 8.7 |
| **Hematocrit (36.6-45.0%)** | 35.2 | 35.0 | 28.2 | 17.9 | 17.2 | 23.1 | 19.0 | 27 |
| **Platelet (150-400 10×3/uL)** | 250 | 291 | 159 | 120 | 80 | 121 | 129 | 495 |
| **Leucocytes (4.0-11.0 10×3/uL)** | 14.3 | 16.5 | 32.4 | 17.2 | 18.1 | 19.4 | 21.4 | 8.4 |
| **PT (8.0-14.0 seg)** | 11.0 | 10.6 | 12.3 | 13.7 | 13.9 | 14.4 | 13.3 | - |
| **PTT (25.0-37.0 seg)** | 26.1 | 23.8 | 31.2 | 29.7 | 30.2 | 34.2 | 34.2 | - |
| **Fibrinogen (170-420 mg/dL)** | - | - | 136 | 119 | 141 | 171 | 373 | - |
| **BUN (19-49 mg/dL)** | 32 | 22 | 26 | - | 29 | - | 56 | - |
| **Creatinine (0.60-1.20) mg/dL** | 0.5 | 0.5 | 0.6 | - | 0.6 | - | 9.7 | 0.5 |
| **Ratio Pr/Cr urine** | 4.2 | | - | - | - | - | - | - |
| **ALT (7-40 U/L)** | 50 | 89 | 242 | 334 | 400 | 447 | 1848 | 63 |
| **AST (12-40 U/L)** | 43 | 92 | 332 | 330 | 437 | 511 | 1679 | 33 |
| **LDH (120-246 U/L)** | 366 | | - | 653 | 698 | 735 | 1841 | |
| **Magnesium (1.6-2.6 mg/dL)** | - | - | 8.1 | 11.9 | 8.2 | 6.3 | - | - |

CS-C-section; PP-postpartum; PT-Prothrombin time; PTT-Partial Thromboplastin Time; BUN-Blood urea nitrogen; Pr/Cr ratio-Protein/creatinine ratio; ALT-Alanine aminotransferase; AST-Aspartate aminotransferase; LDH-Lactate dehydrogenase.

Consequently, the diagnosis of pre-eclampsia without severity criteria was made. Modified biophysical profile assessment at admission was 10/10 and estimated fetal weight was 1810 gr (at 0.7 percentile). The pulsatility index in the umbilical artery doppler was above 95th percentile and the cerebroplacental doppler ratio was below the 5th percentile. The patient was admitted and the protocol of antenatal corticosteroids administration for fetal lung maturation was initiated. The patient remained asymptomatic with controlled blood pressure until day-3 of hospitalization, when she developed intense epigastric pain that did not give in with analgesics and a pre-eclampsia with severity criteria was assumed, so magnesium sulfate infusion was initiated. As a significant vaginal blood loss was observed an emergency c-section was performed based on the suspicion of placental abruption. A male infant was born with Apgar scores of 9 and 10 after 1 and 5 min, respectively that was admitted in

the intensive neonatal care unit due to prematurity. The c-section was uncomplicated and there were no findings of ascites or hemoperitoneum. At the end of c-section, BP was 144/96 mmHg and laboratory findings revealed increased liver enzymes (AST 89 U/L, ALT 92 U/L). After twelve hours postpartum, the patient developed arterial hypotension and obtundation associated with paresthesia. On physical examination she showed abolished patellar reflex, BP 69/49 of mmHg, respiratory rate (RR) of 12 cpm and urine output of 40 mL/h. Laboratory findings revealed significant anemia (Hemoglobin 6.0 g/dL), thrombocytopenia (120×103/mcL), increased liver enzymes (AST 330 U/L, ALT 334 U/L), hypofibrinogenemia (119 mg/dL), and hypermagnesemia (11.9 mg/dL), Table1. Based on the laboratory findings and the physical exam, it was decided to suspend the magnesium sulfate and to administer 2 units of red blood cells. She remained stable until six hours later when started complaining about intense diffuse abdominal pain that did not give in to analgesia, radiating to the right shoulder blade. A new analytical study revealed worsen anemia (Hg 5.6 g/dL), thrombocytopenia (80×103/mcL) and rising levels of liver enzymes (AST 437U/l; ALT 400U/L). On physical examination there were no signs of postpartum hemorrhage, and the uterus was well contracted. On abdominal inspection a distended, rigid abdomen was noted, and she presented signs of peritoneal irritation with defense more pronounced in the right hypochondrium. An urgent CT was performed which revealed the presence of voluminous subcapsular hematoma, with active hemorrhage, reaching practically the entire contour of the right hepatic lobe measuring 12×9×21 cm and a moderate hemoperitoneum associated (Figure 1).

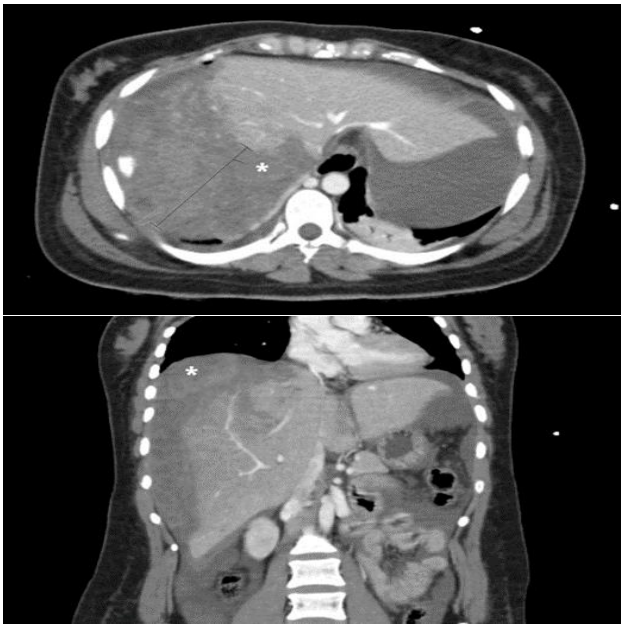

**Figure 1: CT scan at time of diagnosis (18 h postpartum)-voluminous subcapsular hematoma, with active hemorrhage, reaching practically entire contour of right hepatic lobe measuring 12×9×21 cm (*).**

As the patient was in hemorrhagic shock, a massive hemorrhage protocol was activated. She was admitted in the intensive care unit (ICU) and received 2 units of red blood cells, 6 units of fresh frozen plasma, 6 units of platelets, 1 gr of tranexamic acid and 10 mg of phytomenadione. Once clinical and analytical stabilization of the patient was achieved, a conservative approach was decided after a multidisciplinary discussion with general surgery, anesthesia, intensive care medicine and obstetrics.

A new CT scan 54 hours after diagnosis showed a stable hematoma and no evidence of active bleeding. The patient improved clinically and analytically (Table 1) and was transferred to the postpartum unit on day 5. A repeat CT scan on postpartum day 7 showed that the hematoma had decreased in size (Figure 2). As marked improvement continued the patient was discharged home on postpartum day 27.

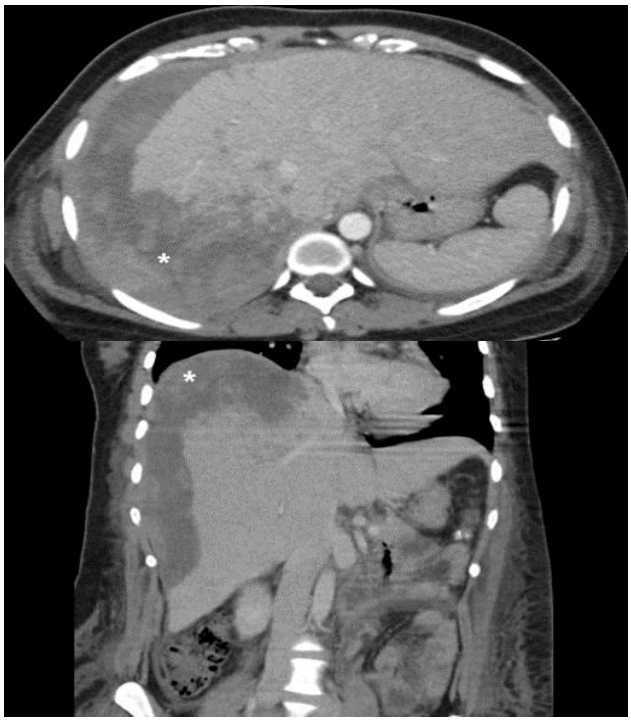

**Figure 2: CT scan at day 7 of postpartum-heterogeneous collection in hepatic subcapsular topography next to the right lobe, compatible with bulky subcapsular hematoma, measuring 9.9×8×16.5 cm. There is an accentuated molding of the hepatic parenchyma of the right lobe, particularly in segment VIII, without manifestations of active bleeding (*).**

The patient then proceeded to a full recovery with resolved transaminitis. A CT scan 3 months after the event showed organization of the hematoma measuring 9×5 cm. One year later, on the follow-up appointment the patient had no complaints, the analytical study was normal, and, on the CT, it remained an intraparenchymal subcapsular collection in hepatic segment VII, lenticular, with 7×4 cm, suggestive of intraparenchymal hematoma in resolution (Figure 3).

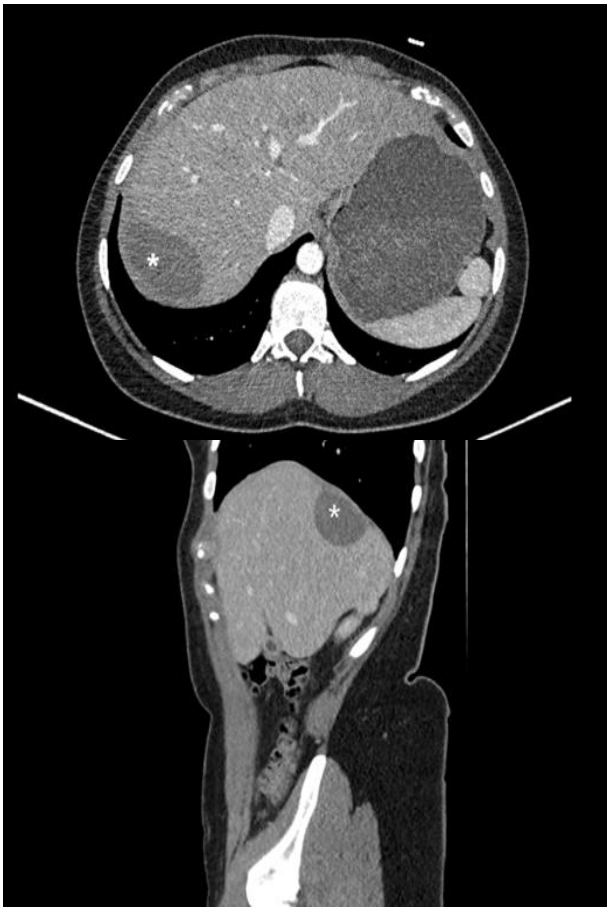

**Figure 3: CT scan 1 year after the episode - intraparenchymal subcapsular collection in hepatic segment VII, lenticular, with 7×4 cm, suggestive of intraparenchymal hematoma in resolution (*).**

## DISCUSSION

The pathogenesis of SLH is not well understood. The common proposed mechanism includes endothelial injury, fibrin deposition leading to platelet activation, thrombus formation and occlusion of hepatic sinusoidal which ultimately progresses to hepatic necrosis and hemorrhage. Neovascularization occurs in the affected parenchyma, and the newly formed vessels are more prone to bleeding in the event of hypertensives episodes.[1,7,8] Intrahepatic hemorrhage further forms into a subcapsular hematoma that can rupture into the peritoneal cavity as the hematoma expands, or after a rise in blood pressure or minor trauma.[6] While this phenomenon can complicate a preexisting lesion of the liver like hemangioma, tumors or adenoma, it most often occurs in association with preeclampsia or HELLP syndrome. HELLP syndrome, characterized by microangiopathic blood smear, elevated liver enzymes and low platelets, occurs in pregnant and postpartum women.[5,9] It complicates about 0.1-1% of all pregnancies and 10% to 20% of those complicated by preeclampsia.[1,4,10] The most common maternal complications of HELLP syndrome are disseminated intravascular coagulation (20%), acute renal failure (7%) and acute pulmonary edema (6%). Less frequently, hepatic hematoma and/or hepatic rupture can occur (1.8%) being this last one the most often fatal complication.[11]

Although HELLP syndrome figures the most frequent cause of SLH, in the presented case, the analytical changes suggestive of HELLP syndrome became pronounced in the postpartum period, so it is unclear whether the analytical changes are a consequence of the SLH associated to a preeclampsia or if they masked a HELLP syndrome too. In most cases described in literature, SLH was diagnosed as a complication of HELLP syndrome before delivery.[5,12] However, a recent retrospective study covering a period of 17 years, showed that the SLH was diagnosed with more frequently at the postpartum.[5] In this case, only 12 hours postpartum the deterioration of analytical parameters and clinical state were notorious.

SLH may present with multiple signs and symptoms, none of which are specific or diagnostic.[8,13,14] The most consistent clinical sign is persistent pain in the epigastrium or right hypochondrium with or not associated scapular irradiation. The pain is due to the distension of the hepatic parenchyma and Glisson's capsule following stasis of blood flow in the hepatic sinusoids. If hepatic rupture occurs, abdominal distension from hemoperitoneum and hypovolemic shock are seen. Our patient had, parallel to other analytical and clinical changes, increased serum levels of magnesium and an abolished patellar reflex that primarily made us suppose that it was a magnesium intoxication.

Very often symptoms appear before the laboratory tests show hepatic abnormal results and it seems that there is no correlation between laboratory abnormalities with intensity of clinical symptoms and the extent and severity of SLH.[1,13] In the presented case, when the patient complained of severe epigastric pain, the analytical study revealed anemia, thrombocytopenia, and moderate rising levels of liver enzymes (AST 437 U/l; ALT 400 U/L). The suspicion of SLH is difficult to achieve based on the initial values of hepatic enzymes, because they are usually altered in severe preeclampsia and HELLP syndrome. In our case, the maximum values of hepatic enzymes were observed only on postpartum day 3.

Rapid and accurate diagnosis is essential. Abdominal ultrasonography and CT are usually used for diagnosis. In the emergency setting, ultrasound is readily available, without moving the patient and can be used to directly identify the hematoma, that most often begins in the right liver lobe as a biconvex subcapsular lens.[15,16] The CT or magnetic resonance imaging (MRI), have very good sensitivity for detecting liver rupture and evaluate the extent of the hematoma, however the access to these imaging tools may be limited for hemodynamically unstable patients.[1] Thus, CT appears to be the modality of choice, as it is more easily available and takes shorter time to complete.

In order to classify injuries and help guide management, the American association for the surgery of trauma (AAST) created an injury scoring scale, reviewed in 2018, that grades the extent of injury from grade I to grade VI.[17] There is no adapted scale for pregnancy because of the rarity of these events. However, this classification system has been used to guide critical care management in these cases. The AAST also predicts the success of a nonoperative approach, which is higher for grade I-III injuries as opposed to grade IV-V injuries. Our patient had a hematoma categorized as grade III in the AAST score system, since it had a maximum dimension above 10 cm (maximum dimensions were 12×9×21 cm) and was intraparenchymal. According AAST, our patient's grade III hematoma was a good candidate to conservative management.[17]

The management of SLH can be conservative or surgical. While no guidelines exist, the AAST scoring system may be a very useful tool helping to decide the best management option between surgical or non-surgical interventions in hemodynamically stable patients. Generally conservative management is advocated in non-bleeding patients, and multiple previous studies reported SLH cases successfully managed conservatively.[7,14,16,18,19] Patient condition should be closely monitored, and any analytical change should be corrected. It's very important to avoid any kind of trauma to the liver, such as abdominal palpation, emesis, convulsion, or abdominal impact during patient transportation, due to the risk of hematoma rupture.[2] Repeated imaging studies, with ultrasound or CT, should be performed to monitor the size of the hematoma and if it increases or the maternal condition deteriorates, surgical evaluation must be sought.

Although surgery has long been considered the standard treatment for SLH, the range of its indications has decreased. Therefore, if rupture is suspected or in hemodynamically unstable patients' surgical management should be considered, and it may include drainage, abdominal packing, ligation of the appropriate branch of either the portal vein or hepatic artery or hepatic resection.[8,20]

Generally abdominal packing is the first treatment step when laparotomy reveals a hepatic rupture. Embolization of the hepatic arteries has also been proposed in the management of a rupture SLH in a stable patient, which is extremely rare, or in patients with SLH with no spontaneous regression.[21] Rinehart et al reported a maternal survival rate after selective hepatic embolization of 90%, and a mortality rate above 30% when other techniques like surgical ligation of the hepatic arteries or resection of hepatic necrosis plaques were considered.[6] Liver transplant, generally a last resource, should be considered in the presence of refractory liver hemorrhage or rapidly progressing fulminant liver failure.[20] Successful liver transplantation has been reported in pregnancy associated with SLH rupture at least in 2 cases.[22,23]

There is scarce data regarding recurrence rate of SLH. There are some cases reports of uneventful pregnancies after an SLH, and one case of recurrent SLH. The first event was an unruptured liver hematoma treated conservatively. In the second pregnancy the patient had a rupture of the hematoma and was treated with arterial embolization followed by surgery. Both events occurred in the postpartum period.[24]

Following improvements in imaging, diagnosis, resuscitation techniques and in the field of liver traumatology, the mortality of this rare complication has decreased in recent years. This case highlights the importance of urgent liver imaging for every woman with preeclampsia or HELLP syndrome with unusual epigastric symptoms even in the postpartum. Conservative management with a multidisciplinary team that includes obstetrics, critical care, surgery, hematology, and gastroenterology should be considered in hemodynamically stable patients with AAST grade I, II and III hematomas, such as the patient presented in this report. Thus, a high index of suspicion, a prompt diagnosis and a multidisciplinary approach with intensive hemodynamic support are the key factors for a successful outcome.

## ACKNOWLEDGEMENTS

Authors would like to thanks to clinicians from departments of obstetrics, general surgery, radiology, gastroenterology, and intensive care unit of hospital de Braga for their contribution.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

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**Cite this article as:** Borges AC, Almendra R, Nogueira-Silva C, Correia C. Postpartum subcapsular liver hematoma: a rare complication successfully managed with conservative treatment. Int J Reprod Contracept Obstet Gynecol 2023;12:1459-64.