EMBJ.ORG FOR YOUNG DOCTORS

EUROMEDITERRANEAN BIOMEDICAL JOURNAL
2021,16 (27) 114-117
(*FORMERLY: CAPSULA EBURNEA*)

Case report

SUBCAPSULAR HEPATIC HEMATOMA: A COMPLICATION OF HELLP SYNDROME. CONSERVATIVE MANAGEMENT AND COMPARISON WITH LITERATURE

Giulia Tralli, Zairo Ferrante, Monica Graziano, Elisabetta Salviato, Roberto Galeotti

Department of Radiology, Sant'Anna University Hospital of Ferrara, Ferrara, Italy

ARTICLE INFO

Article history:
Accepted 19 Jan 2021
Revised 28 Apr 2021
Published 08 Aug 2021

Keywords:
Subcapsular liver hematoma, hepatic hematoma, transcatheter arterial embolization, preeclampsia, pregnancy, liver complication, HELLP syndrome.

ABSTRACT

Microangiopathic hemolytic anemia, elevated liver enzymes and low platelet count, also known as HELLP syndrome, is a rare complication of pregnancy with high maternal and fetal mortality. One of the most serious complications is a subcapsular liver hematoma. The diagnostic techniques used for diagnosis are ultrasound, CT and MRI. Hemodynamically stable patients should be treated conservatively and in cases of active bleeding with percutaneous embolization of the hepatic arteries. We present a case of a 35-year-old woman with subcapsular liver hematoma with active arterial bleeding who underwent an emergency cesarean section and was treated conservatively with artery embolization with gelatin sponge.

1. Introduction

HELLP syndrome is a rare, potentially life-threatening, complication of pregnancy characterized by the association of microangiopathic hemolytic anemia, elevated liver enzymes and low platelet count. It is considered a severe form of preeclampsia[1]. The incidence is about 0.2–0.6% of all pregnancies but it occurs in up to 10–20% of women with severe preeclampsia[1]. It frequently occurs before delivery, mostly between the 27th and 37th week of gestation with a higher risk of complications in cases that develop after delivery [2].

The exact etiology of HELLP syndrome is still unknown. It is probably due to placental factors and maternal genetic predisposition which lead to an acute inflammatory process, liver damage and activation of the coagulation system[1].

The syndrome shows a familial tendency. The major maternal complications include disseminated intravascular coagulation, abruptio placentae, acute renal failure, pulmonary edema, subcapsular liver hematoma and hepatic rupture[2]. In most cases, postpartum HELLP syndrome resolves spontaneously within 48 hours. One of the most serious complications of HELLP syndrome is subcapsular liver hematoma.

It has been reported in less than 2% of pregnancies complicated by HELLP syndrome and the incidence of rupture in pregnancies varies from 1/40,000 to 1/250,000 deliveries[3]. Rupture of hepatic hematoma associated with HELLP syndrome is an unusual fatal complication of pregnancy; maternal and fetal mortality rates are still high despite advances in diagnosis and treatment[4]. HELLP syndrome may present with nonspecific symptoms, such as right upper quadrant or epigastric pain, nausea or vomiting, headache and a history of malaise. Abdominal pain is the most important symptom, caused by the stretching of Glisson's capsule[2]. Microangiopathic hemolytic anemia is characterized by abnormal peripheral smear, elevated serum bilirubin, low serum haptoglobin levels, elevated lactate dehydrogenase levels, and low hemoglobin levels[5]. At present, the two major guidelines used for HELLP syndrome diagnosis are the Tennessee and Mississippi classification, that are based on biochemical laboratory data such as hemolysis, elevated liver enzymes and low platelet count. The Mississippi classification emphasized thrombocytopenia as the most important factor to asses the severity of HELLP syndrome[6]. Ultrasound, CT and MRI are the techniques used for diagnosis[5,7]. The prognosis can be changed by timely diagnosis and treatment.

* *Corresponding author*: Giulia Tralli, *giuliatrl86@libero.it*
DOI: 10.3269/1970-5492.2021.16.27
 ISSN: 2279-7165 - Available on-line at www.embj.org

In this case, we report a subcapsular liver hematoma in HELLP syndrome managed conservatively.

2. Case report

A 35-year-old woman, gravida 1, was admitted to the emergency department at 35 weeks of gestation with acute epigastric pain initially treated with ranitidine without benefit. She denied nausea and vomitting. The patient was normotensive. Laboratory tests showed high serum alanine aminotransferase (ALT 329 U/L), high lactic dehydrogenase (LDH 521 U/L) and low platelet count (PLT 139,000/µL). Clinical examination on admission demonstrated abdominal pain without tenderness or rigidity. In obstetrical ultrasonography, fetal heart rate was regular and fetal measurement was compatible with 35 weeks of gestation. An abdominal ultrasound revealed intrahepatic hematoma presenting as a hypoechogenic area with diffuse heterogeneity (anteroposterior diameter of 3cm) without free fluid in the abdominal cavity (Figure 1). The patient was hospitalized with a diagnosis of elevated liver enzymes and low platelets with suspected HELLP syndrome complicated with subcapsular hepatic hematoma. The patient underwent an emergency cesarian section and a live-born infant was delivered.

In the immediate post-operative period, the patient was transferred to the intensive care unit and started antihypertensive and heparin therapy. Computed tomography abdominal scan confirmed the large subcapsular hematoma of the right liver lobe without capsular rupture and after intravenous administration of iodinated contrast material multiple focal high-attenuation areas were revealed that represented a collection of extravasated contrast material secondary to arterial active bleeding (Figure 2a-c; Figure 3).

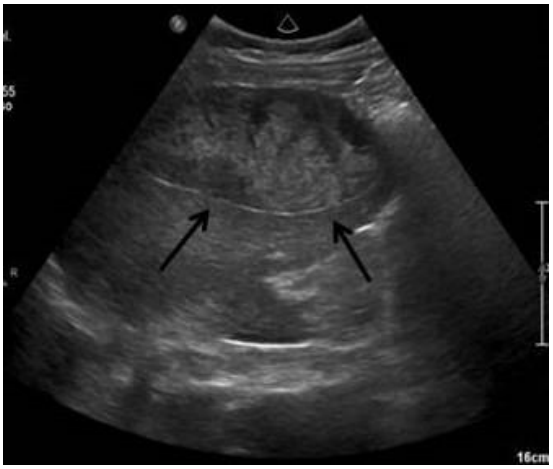
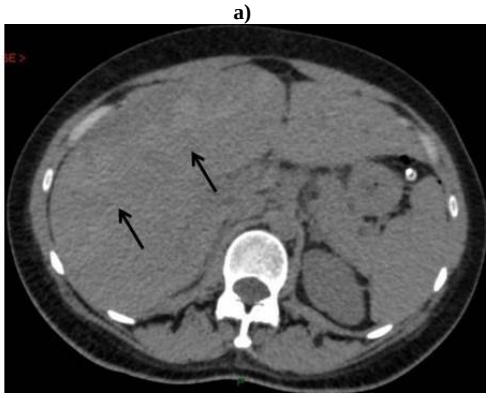
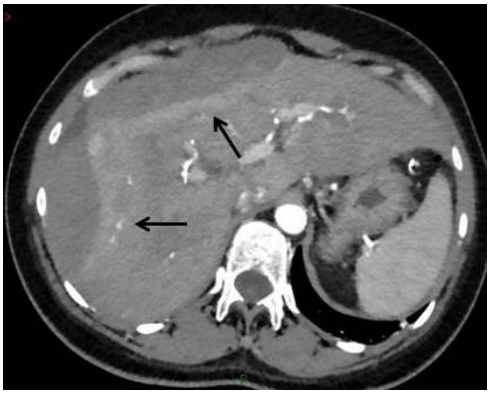


Figure 1. Transabdominal ultrasound shows a heterogenous collection with multiple areas of different echogenicity consistent with subcapsular liver hematoma. There were no signs of rupture and no free fluid in the abdomen (arrows).

a)



b)



c)

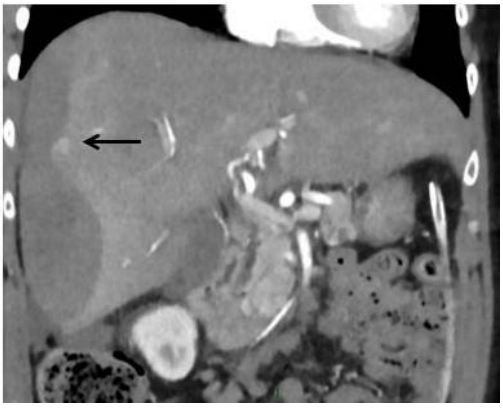


Figure 2. a) MDCT of the abdomen in the axial plane obtained before intravenous administration of iodinated contrast material shows ill-defined, intrahepatic, peripheral hypoattenuating area (arrows) consistent with intrahepatic hematoma. b-c) MDCT of the abdomen in the axial (Fig.2b) and coronal plane (Fig.2c) obtained after intravenous administration of iodinated contrast material during the arterial phase shows a large subcapsular liver hematoma with multiple hyperdense areas due to arterial active bleeding (arrows).

The patient underwent angiography that confirmed multiple foci of contrast medium extravasation in the peripheral branches of the right hepatic artery (Figure 4).
The portal vein was widely patent. The proximal right hepatic artery was selectively catheterized with a 5-F catheter and the embolization was performed with gelatin sponge mixed with contrast material and antibiotic (Gentamicin). Post-embolization arteriography demonstrated cessation of bleeding of all peripheral right hepatic artery branches (Figure 5).
During follow up, the patient repeated ultrasound examinations every 2-3days which demonstrated a stable hematoma with signs of organization. Her blood data improved with a decrease in inflammatory indexes and hepatic enzymes. At 6 days a computed tomography abdominal scan showed a dimensional reduction of the hematoma that appeared more hypodense due to the organizing process (Figure 6). The patient remained stable throughout the hospitalization period and she was discharged at 30 days.

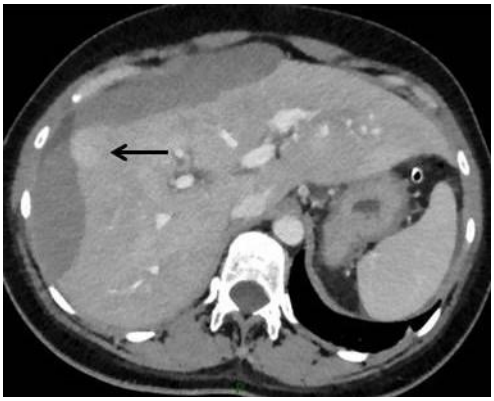


Figure 3. MDCT of the abdomen in the axial plane obtained during the portal phase of intravenous administration of iodinated contrast material shows a well defined hypodense subcapsular hematoma with compression of hepatic parenchyma and a larger hyperattenuating area consistent with arterial active bleeding (arrow).

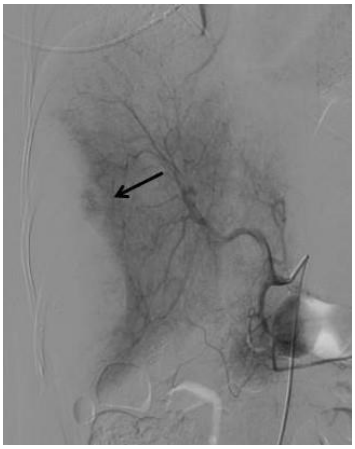


Figure 4. Selective hepatic artery angiogram demonstrated extravasation of iodinated contrast material (arrow). Transcatheter arterial embolization was performed resulting in immediate resolution of arterial bleeding.

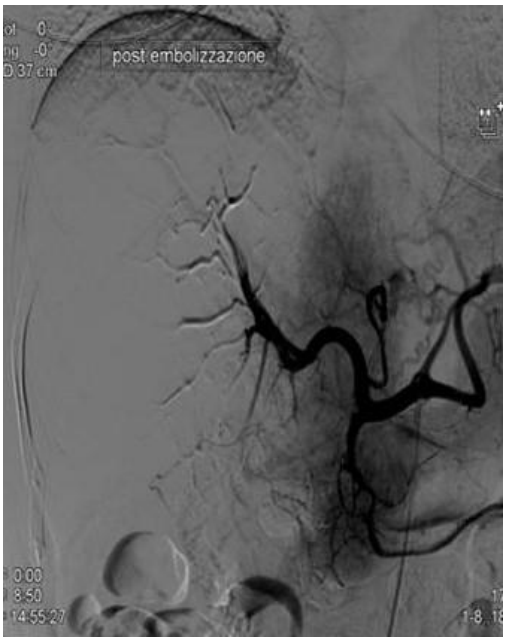


Figure 5. Post-embolization angiogram demonstrates the cessation of bleeding of all peripheral right hepatic artery branches.

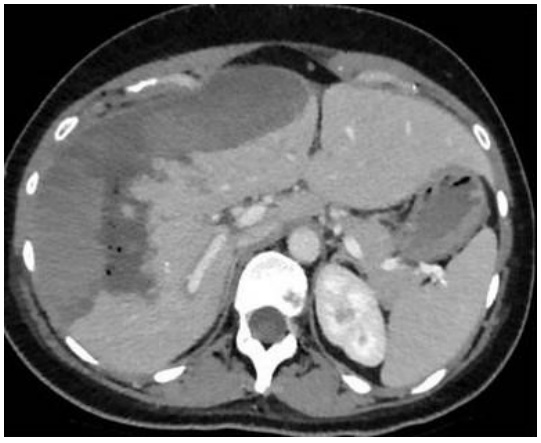


Figure 6. Computed tomography abdominal scan showed a dimensional reduction of the hematoma that appeared more hypodense due to the organizing process.

3. Discussion

Subcapsular hepatic hematoma has an incidence of approximately 1 per 45.000 live births[9] and is most frequently observed in advanced maternal age and multiparous patients. The etiopathogenesis is not well established. It has been reported that preeclamptic syndrome induces deposition of fibrinoid thrombi in the hepatic sinusoids which causes platelet activation leading to hepatic ischemia, necrosis and subcapsular hepatic hematoma[8].
Hepatic hematoma occurs more frequently in the right liver. The diagnosis can be established with an abdominal ultrasound examination.

The subcapsular hematoma appears as a hypoechogenic area with diffuse heterogeneity; if intraperitoneal fluid is present a hepatic rupture must be considered.

At unenhanced CT acute hematomas are typically hyperattenuating (40–60 HU) relative to normal liver parenchyma. At contrast-enhanced CT they appear as an elliptic collection of low-attenuation blood between the liver capsule and enhancing liver parenchyma.

In our case, the ultrasound was performed before delivery and the patient underwent emergency cesarian section. CT scan confirmed the sonographic findings and showed subcapsular liver hematoma with active bleeding; furthermore, it showed a small amount of free fluid in the abdominal cavity but no signs of hepatic rupture. In these cases, the conservative approach is accepted worldwide, as surgical intervention is associated with a worse maternal prognosis.

Haemodynamically stable patients should be followed up conservatively by intensive medical support with the replacement of blood products and fluids and treatment of HELLP syndrome or/and preeclampsia and even with percutaneous embolization of the hepatic arteries in cases of active bleeding[8], such as in our case. Hepatic rupture occurs when the hepatic hematoma over distends the liver capsule resulting in a life-threatening condition.

If the patient is hemodynamically unstable, surgery can be necessary. Liver transplantation has been reported when the hemorrhage cannot be controlled and acute liver failure occurs[8].

Post-partum follow-up should include serial assessment with ultrasound, CT or MRI and several laboratory tests to evaluate hemodynamic and coagulation parameters.

In our case, the patient was hemodynamically stable and underwent conservative management with antihypertensive therapy for the treatment of HELLP syndrome and arterial embolization with gelatin sponge for the subcapsular hematoma. She was followed up daily with an abdominal ultrasound and with abdominal CT one week later, to asses the regression of hematoma. Our case had a favorable evolution under conservative management and no surgical procedures were needed. Close monitoring of these patients in the pre and the postpartum period is mandatory. In hemodynamically stable patients conservative management should be the first choice of treatment.

References

1. Smith L, Perronne L, Dohan A, Bazeries P, Guerrache Y, Fohlen A, Rousset P, Aube´ C , Laurent V, Morel O, Boudiaf M, Hoeffel C, Soyeret P. Hepatic involvement in HELLP syndrome: an update with emphasis on imaging features. Abdom Imaging (2015) 40:2839–2849 DOI: 10.1007/s00261-015-0481-1
2. Troja A, Abdou A, Rapp C, Wienand S, Malik E, Raab H-R Management of Spontaneous Hepatic Rupture on Top of HELLP Syndrome: Case Report and Review of the Literature. Viszeralmedizin 2015;31:205–208 DOI: 10.1159/000376601
3. Fujiyama F, Yamashita Y, Fujita D, Tabuki K, Kawabe S, Kato S, Soen H, Kamegai H, Ohmichi M, Yamamoto Z. An effective hepatic embolization for subcapsular hematoma in a case of postpartum HELLP syndrome a case report. Bullettin of the Osaka Medical College 59 (1) 31-35 2013.
4. Grand'Maison S, Sauve N, Weber F; Dagenais M; Durand M; Mahone M. Hepatic Rupture in Hemolysis, Elevated Liver Enzymes, Low Platelets Syndrome (Obstet Gynecol 2012;119:617–25) DOI: 10.1097/AOG.0b013e318245c283
5. Ditisheim A. and Sibai B.M. Diagnosis and Management of HELLP Syndrome Complicated by Liver Hematoma Clinical obstetrics and gynecology Volume 60, Number 1, 190–197
6. Rimaitis K, Grauslyte L, Zavackiene A, Baliuliene V, Nadisauskiene R, Macas A. Diagnosis of HELLP Syndrome: A 10-Year Survey in a Perinatology Centre. Int. J. Environ. Res. Public Health 2019, 16, 109; Doi:10.3390/ijerph16010109
7. Wicke C, Pereira P.L, Neeser E, Flesch I, Rodegerdts E. A., Becker H. D. Subcapsular liver hematoma in HELLP syndrome: Evaluation of diagnostic and therapeutic options: A unicenter study. American Journal of Obstetrics and Gynecology (2004) 190, 106e112
8. Karateke A, Silfeler D, Karateke F, Kurt R, Guler A, Kartal I. HELLP Syndrome Complicated by Subcapsular Hematoma of Liver: A Case Report and Review of the Literature. Case Reports in Obstetrics and Gynecology Volume 2014, Article ID 585672, 3 pages http://dx.doi.org/10.1155/2014/585672
9. Marinas M,C, Mogos G,F,R, Dragușin R,C, Tudorache S, Iliescu D, G. Postpartum Spontaneous Subcapsular Hepatic Hematoma (SSHH)-Conservative Management. Case Report and Review of Literature Current Health Sciences Journal. Vol. 44, No. 4, 2018 October-December.