Spontaneous Hepatic Rupture in Pregnancy

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ABSTRACT

Spontaneous hepatic rupture (SHR) is a rare and potentially fatal condition associated with both benign and malignant liver disease. Though rare, pregnant patients with HELLP (Hemolysis, Elevated Liver enzymes and Low Platelets) syndrome and preeclampsia are at elevated risk of SHR. Early identification and a high clinical index of suspicion for SHR in patients with preeclampsia and HELLP syndrome can reduce both maternal and fetal mortality. We review the existing literature and present the case of a 35-year-old woman with SHR. The patient was originally admitted for abdominal pain at 38 weeks' gestation and found to have preeclampsia with severe features. Cross-sectional imaging demonstrated a subcapsular hepatic hematoma on imaging. Following an uncomplicated Cesarean delivery, she became hemodynamically unstable. Imaging demonstrated bilateral hepatic rupture. She was successfully treated with angioembolization and operative control of the hepatic hemorrhage.

KEYWORDS: Spontaneous hepatic rupture; Preeclampsia; HELLP; Pregnancy

INTRODUCTION

Spontaneous hepatic rupture (SHR) in pregnant patients is a rare condition with high rates of morbidity and mortality. In a retrospective study of 391 patients with SHR, the maternal mortality rate was 22.1%.[1,2] Overall, the most common cause of SHR is hepatocellular carcinoma. In pregnant patients, SHR has been linked with trauma, but it is most commonly associated with preeclampsia with severe features (70.4%) and HELLP syndrome (Hemolysis, Elevated Liver enzymes, and Low Platelets, 83.3%).[2,3] SHR can also be caused by neoplasms or peliosis hepatis.[3,4] Disseminated intravascular coagulation (DIC), placental abruption, and acute renal failure are some of the more frequent complications associated with this condition. Treatment options include angioembolization, hepatic packing, arterial ligation, formal hepatectomy, and, less commonly, liver transplantation.[5]

In pregnancy, SHR can be fatal to both the pregnant patient and fetus. It commonly presents in patients with no history of hypertension or coagulopathy. SHR may be diagnosed intraoperatively during a cesarean section or in the postpartum period, with a median gestational age of 35 weeks at the time of diagnosis.[2] Presenting symptoms include severe abdominal pain, nausea, emesis, anemia, and sudden hemodynamic instability. Understanding the treatment pathways after diagnosis of hepatic rupture is crucial for safely managing patients. We present the case of a pregnant patient at full-term who had SHR requiring multiple surgical interventions and a protracted stay in the intensive care unit. This case emphasizes the importance of clinical awareness of SHR and highlights the advantage of multidisciplinary care in treating this condition.

CASE REPORT

A 35-year-old G3P0020 woman with a history of two spontaneous abortions presented at 38- weeks six-days gestation with severe right upper quadrant abdominal pain and emesis. Her prenatal course was complicated by urinary tract infection, resolved placenta previa, and large-for-gestational-age fetus on antenatal ultrasound. She was found to have a blood pressure of 152/100 on admission and elevated liver transaminases (ALT 548, AST 1480), leading to a diagnosis of preeclampsia with severe features. She was treated with labetalol and magnesium sulfate. She had no prior history of hypertension. Abdominal computed tomography (CT) demonstrated a 2.3 x 0.9 x 2.5 cm right hepatic lobe peripheral hypodensity suspicious for subcapsular liver hematoma without active extravasation [**Figure 1**]. She underwent a cesarean section for preeclampsia during which the estimated blood loss was 1 L, which is considered as the upper limit of normal expected blood loss in this operation. A surgical consult was obtained at the time of the cesarean section to evaluate the liver. The hepatic lesion was palpated, and the liver capsule was found to be intact. Her hemoglobin decreased from 13.1 to 9.0 within a few hours postoperatively.

Five hours later, the patient became somnolent, pale, and hypotensive to 68/41. On physical exam, her abdomen was distended and rigid to palpation with rebound tenderness. Extremities were cool and peripheral pulses were thready. An ultrasound revealed free fluid in the hepatorenal recess concerning for intraperitoneal hematoma. The massive transfusion protocol was initiated, and the patient received 8

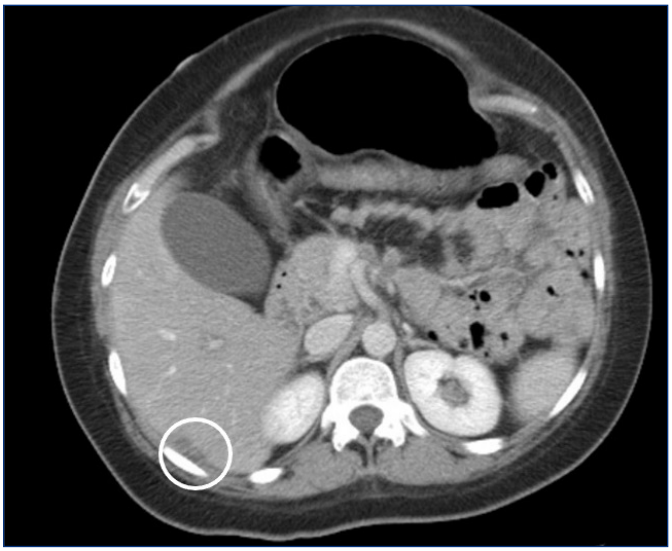

Figure 1. Initial CT on admission demonstrating a 2.3 x 0.9 x 2.5 cm right subcapsular hepatic hematoma (circle).

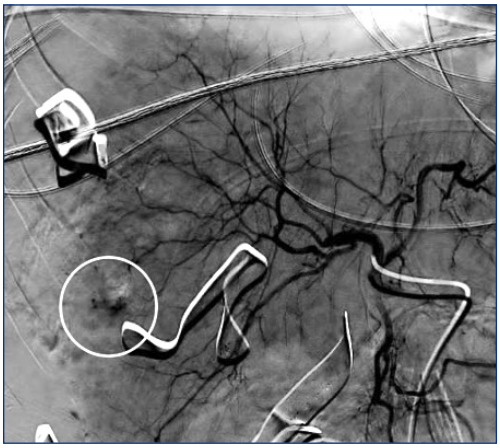

Figure 2A. Mesenteric angiography demonstrates diffuse vessel irregularity throughout the liver with some focal areas of contrast blush distally in the right hepatic lobe (circle).

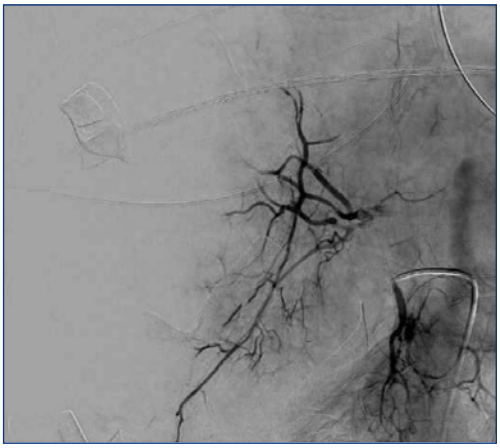

Figure 2B. Angiogram after Gel foam embolization of hepatic segments 6-8.

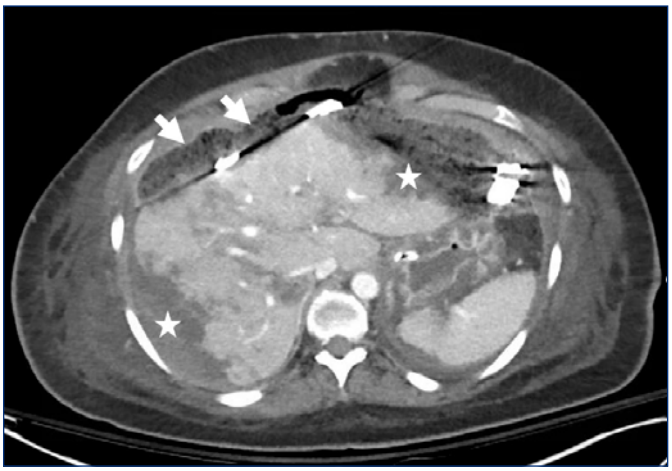

Figure 3. CT angiogram on post-operative day 5 demonstrating diffuse subscapular hematomas (stars) and packing material (arrows).

units (u) of packed red blood cells, 2 u of cryoprecipitate, 5 u of fresh frozen plasma, and 1 u of platelets. Arterial blood gas showed a pH of 7.1, lactate of 6.0 mmol/L, and low fibrinogen, concerning for DIC. An emergent exploratory laparotomy was performed during which the patient was found to have active hemorrhage from hepatic segments V and VI. The abdomen was packed and the patient was taken for catheter-directed embolization of hepatic segments VI-VIII [**Figures 2a,b**]. The Pfannenstiel and upper- midline incisions were left open, and a temporary abdominal closure device was placed.

The patient returned to the operating room on postoperative day 1. Although the initial right-lobe hemorrhage was hemostatic, the left liver capsule was found to be ruptured with considerably disrupted parenchyma. Bipolar cautery, topical hemostatic agents, and packing were used to control bleeding. In conjunction with the maternal fetal medicine service, the uterus was examined and was found to be unremarkable. On post-operative day 4, the patient was taken for operative washout of the abdomen and closure of the Pfannenstiel incision. A CT angiogram performed at that time demonstrated extensive hepatic damage, with liver heterogeneity and peripheral low-density hepatic/foci hemorrhage [**Figure 3**].

Her postoperative course was complicated by persistent respiratory failure and pneumonia requiring tracheostomy and thoracentesis. She also developed purpuric skin lesions consistent with a microvascular thrombotic process as seen in DIC on biopsy. She was eventually decannulated and discharged home after a 32-day hospital stay. After a few days in the neonatal intensive care unit for respiratory support, the newborn was deemed medically ready for discharge home.

DISCUSSION

Spontaneous hepatic rupture in pregnancy is a life-threatening emergency that can be successfully treated with prompt recognition and treatment. Multidisciplinary collaboration of obstetricians, surgeons, and radiologists is critical. The maternal and fetal mortality rates are 22.1% and 37.2%, respectively.[2] Most patients present in the late-second or third trimester. In a study of 391 patients with SHR, 250 (63.9%) were diagnosed during pregnancy (63.9%) and 141 (36.1%) were diagnosed in the postpartum period.[2] SHR is

strongly associated with hypertensive disorders of pregnancy, including preeclampsia or HELLP syndrome. In a prospective cohort study of 442 patients with HELLP syndrome, 0.9% developed SHR.[6] As a result of this association, prior literature has proposed that patients with preeclampsia or HELLP syndrome who have right upper-quadrant pain and hemodynamic instability warrant exclusion of SHR as the initial diagnosis.[2,7] However, not all hepatic rupture cases stem from HELLP and preeclampsia.[8,9] Augustin et al found that 81.4% of women with SHR had HELLP syndrome, 70.4% had preeclampsia, and 9.1% had eclampsia.[2] This association is often predicated on the assumption of universal prenatal care and early diagnosis of hypertensive disorders of pregnancy. Thus, clinical suspicion for SHR should exist for any third-trimester pregnant or postpartum patient with abdominal pain and unstable hemodynamics.

SHR is not evenly distributed in the liver. In the majority of cases, the right lobe is the origin of the hemorrhage (70.9%).[2,10] Rupture occurs bilaterally in (22.1%), and uncommonly in the left lobe alone (6.9%).[2,10] Management depends on patient stability, individual presentation, hospital resources, and physician expertise. While observation may be reasonable in a hemodynamically stable patient, active intervention is the best initial management option for acutely ill patients.[5,7] Interventions can include angioembolization, surgical packing, topical hemostatic agents, electrocautery, hepatic artery ligation, hepatectomy, and liver transplantation.[11] Surgical packing of the liver bed is the most common surgical intervention (56.4%).[2]

Some studies demonstrate a decrease in maternal mortality with liver transplantation and embolization; however, surgical packing is less morbid and should be considered initially with or without adjunct interventions like embolization.[2,5,12] Liver transplantation for HELLP is rare and reserved for severe hepatic failure. There were eight deceased donor liver transplants between 1987 and 2003 for HELLP syndrome of which six had long-term post-transplant survival.[13] As with the case presented above, damage-control laparotomy followed by additional operations for liver-directed therapy are appropriate in hemodynamically unstable patients. Hepatic embolization may also be suitable when both hepatic lobes are involved. Augustin et al found that while surgical packing was the most common treatment, it did not seem to influence survival.[2] In contrast, embolization was performed less frequently (n= 33 in comparison to n= 213 for liver packing) and was shown to have a significant survival benefit.[2] However, these therapies may be difficult to compare when using a retrospective approach. Persistent hemorrhage requires an individualized approach, so interval inspection and repacking can aid in the guidance of the treatment plan.

Interdisciplinary management has been shown to reduce the rates of maternal and fetal mortality and improves patient outcomes.[15] Furthermore, this multidisciplinary approach should continue after discharge from the hospital for continued optimization of both maternal and newborn outcomes. While there is a paucity of data regarding SHR recurrence in future pregnancies, the consequences of the resolved hepatic pathology, such as liver function, and interventions must be considered.[14] Close communication and continued interdisciplinary team follow-up can help identify potential physical and psychological complications after discharge.[15]

Special considerations are necessary in post-partum patients. Early consultation with lactation specialists and allowing women an opportunity to bond with their newborns is essential in optimizing the patient experience.

CONCLUSION

This case emphasizes the importance of clinical awareness in the rare but potentially fatal diagnosis of SHR in pregnant and post-partum patients. Individualized interventions, including endovascular and operative approaches, can be used to control the acute rupture. While the mortality rate in SHR is high, early identification and interdisciplinary management have the potential to optimize both maternal and fetal outcomes.

References

1. Brito M, Gamito M, Neves AR, Caeiro F, Martins A, Dias E, Veríssimo C. Conservative management of a pregnancy complicated by preeclampsia and postpartum spontaneous hepatic rupture: A case report and review of the literature. Eur J Obstet Gynecol Reprod Biol. 2021 Dec;267:79-89.
2. Augustin G, Hadzic M, Juras J, Oreskovic S. Hypertensive disorders in pregnancy complicated by liver rupture or hematoma: a systematic review of 391 reported cases. World J Emerg Surg. 2022;17(1):40.
3. Nam IC, Won JH, Kim S, Bae K, Jeon KN, Moon JI, Cho E, Park JE, Jang JY, Park SE. Transcatheter Arterial Embolization for Spontaneous Hepatic Rupture Associated with HELLP Syndrome: A Case Report. Medicina (Kaunas). 2021 Oct 2;57(10):1055.
4. Choi SK, Jin JS, Cho SG, Choi SJ, Kim CS, Choe YM, Lee KY. Spontaneous liver rupture in a patient with peliosis hepatis: a case report. World J Gastroenterol. 2009 Nov 21;15(43):5493-7.
5. Mascarenhas R, Mathias J, Varadarajan R, Geoghegan J, Traynor O. Spontaneous hepatic rupture: a report of five cases. HPB (Oxford). 2002;4(4):167-70.
6. Sibai BM, Ramadan MK, Usta I, Salama M, Mercer BM, Friedman SA. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). Am J Obstet Gynecol. 1993;169(4):1000-1006.
7. Pavlis T, Aloizos S, Aravosita P, Mystakelli C, Petrochilou D, Dimopoulos N, Gourgiotis S. Diagnosis and surgical management of spontaneous hepatic rupture associated with HELLP syndrome. J Surg Educ. 2009 May-Jun;66(3):163-7.
8. Han GH, Kim MA. Recurrent spontaneous hepatic rupture in pregnancy: A case report. Medicine (Baltimore). 2018 Jul;97(29):e11458.
9. Sutton BC, Dunn ST, Landrum J, Mielke G. Fatal postpartum spontaneous liver rupture: case report and literature review. J Forensic Sci. 2008 Mar;53(2):472-5.

10. Henny CP, Lim AE, Brummelkamp WH, Buller HR, Ten Cate JW. A review of the importance of acute multidisciplinary treatment following spontaneous rupture of the liver capsule during pregnancy. Surg Gynecol Obstet. 1982;156:593-8.
11. Hunter SK, Martin M, Benda JA, Zlantik FJ. Liver transplant after massive spontaneous hepatic rupture in pregnancy complicated by preeclampsia. Obstet Gynecol. 1995;85:819–22.
12. Wilson SG, White AD, Young AL, Davies MH, Pollard SG. The management of the surgical complications of HELLP syndrome. Ann R Coll Surg Engl. 2014 Oct;96(7):512-6.
13. Shames BD, Fernandez LA, Sollinger HW, Chin LT, D'Alessandro AM, Knechtle SJ, Lucey MR, Hafez R, Musat AI, Kalayoglu M. Liver transplantation for HELLP syndrome. Liver Transpl. 2005 Feb;11(2):224-8.
14. Stevenson JT, Graham DJ. Hepatic haemorrhage and HELLP syndrome: a surgeon's perspective. Am Surg. 1995;61:756–60.
15. Bottom-Tanzer SF, Poyant JO, Louzada MT, Abela D, Boudouvas A, Poon E, Power L, Kim WC, Hojman HM, Bugaev N, Johnson BP, Bawazeer MA, Mahoney EJ. Longitudinal Study Evaluating Post-ICU Syndrome Differences between Acute Care Surgery and Trauma SICU Survivors. J Trauma Acute Care Surg. 2023 Jun 14.

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