Preeclampsia and HELLP syndrome complicated by subcapsular liver hematoma and rupture

S. Ainslie McBride MD MSc, Rebecca Small MD MSc, Kelle Hurd MD, Sunette Claassens MD

Cite as: *CMAJ* 2023 November 20;195:E1543-5. doi: 10.1503/cmaj.230851

A previously healthy 30-year-old primiparous woman at 39 weeks' gestation presented to her family physician with decreased fetal movement and was referred to triage at the labour and delivery unit. She had a 4-day history of right upper quadrant pain, cramping, nausea, vomiting and anorexia. She had no headache, visual changes, chest pain, dyspnea or edema to suggest preeclampsia. She was normotensive and tachycardic (heart rate 115 beats/min), and her cardiorespiratory examinations were unremarkable. Her abdomen was tender to palpation. Investigations revealed anemia with a hemoglobin level of 83 (normal range 120–160) g/L, thrombocytopenia, a normal creatinine level and an elevated protein-to-creatinine ratio (Table 1). Her hemoglobin level had been normal 8 weeks previously. Fetal heart rate abnormalities were seen with a sudden, severe and prolonged deceleration, and the patient was moved to the operating room for an emergency cesarian delivery.

In the operating room, the patient had a hemoperitoneum of about 700 mL with unclear origin. We consulted the general surgery team emergently for intraoperative exploration, but the source of the hemoperitoneum was not identified. The team delivered a live female infant with Apgar scores of 9 at 1 and 5 minutes. The patient's estimated blood loss was 1795 mL and she was resuscitated with packed red blood cells, platelets, tranexamic acid and activated fibrinogen. She also received a total of 9 g of intravenous magnesium sulfate (bolus and subsequent infusion), given the concern for evolving preeclampsia. Postoperative computed tomography (CT) of her abdomen and pelvis using a multiphasic abdomen protocol (unenhanced, arterial and portal venous

Key points

- Hypertensive disorders of pregnancy are increasingly common, including severe manifestations such as preeclampsia complicated by hemolysis, elevated liver enzymes and low platelets (HELLP) syndrome.
- Presenting features of hypertensive disorders of pregnancy can be nonspecific, including headache, visual disturbances, dyspnea, epigastric pain, nausea, vomiting and peripheral edema.
- Subcapsular liver hematoma is an uncommon complication of HELLP syndrome and may lead to liver rupture or infarct.
- Management of subcapsular liver hematoma should focus on timing and modality of delivery, blood pressure control and identifying and managing complications such as acute kidney injury, hemoperitoneum and thrombosis.

Table 1: Laboratory results of a 39-year-old woman (39 wk gestation) from initial presentation to 7 days postpartum

Investigation	Reference range	Initial presentation	Postpartum day		
			1	2	6–7
Hemoglobin, g/L	120–160	83 (127 baseline 8 wk previously)	93	65	94
Platelets, × 10^9/L	150–400	102	83	35	299
Leukocyte count, × 10^9/L	4.0–11.0	17.6	24.2	13	14
Creatinine, µmol/L	40–100	95	82	73	63
Estimated glomerular filtration rate, mL/min/1.73 m^2	≥ 60	69	83	96	114
Alanine transaminase, U/L	≤ 49	213	229	765	149
Lactate dehydrogenase, U/L	100–235	189	765	1364	351
Haptoglobin, g/L	0.30–2.00			0.18	
Total bilirubin, µmol/L	0–24	14	20	40	16
Urate, µmol/L	140–350		425	449	268
Urine protein-to-creatinine ratio, g/mmol	< 0.013	0.023			

phase) showed a large subcapsular liver hematoma (5.8 × 16.4 × 19.3 cm) with rupture through the hepatic capsule and extension along the right paracolic gutter into the pelvis (Figure 1) and a partially occlusive left gonadal (ovarian) vein thrombosis with extension into the left proximal renal vein. Three sites of active extravasation were identified and confirmed with digital subtraction angiography to originate from branches of the right hepatic artery. These sites were embolized successfully with a slurry of hemostatic absorbable gelatin powder.

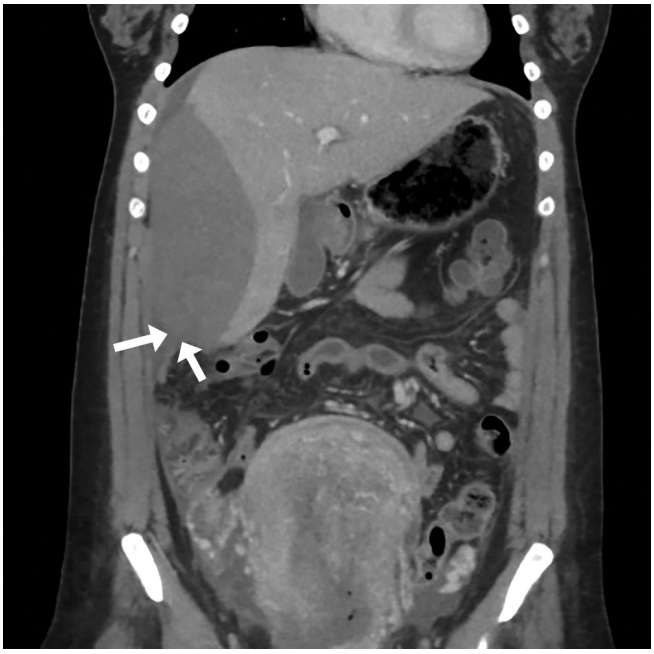

Figure 1: Coronal portal venous phase computed tomography scan of a 39-year-old woman after emergent cesarean delivery at 39 weeks' gestation, showing subcapsular liver hematoma breaking through the liver capsule (arrows) and extension along the right paracolic gutter.

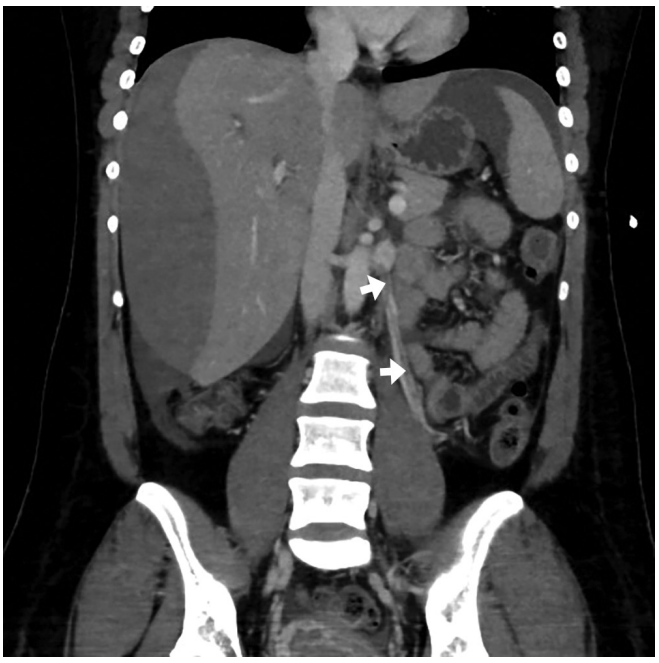

Figure 2: Coronal portal venous phase computed tomography scan of a 39-year-old woman on postpartum day 2 showing gonadal vein thrombus (arrows) and stable appearance of hepatic hematoma.

Postoperatively, the patient reported gradually resolving abdominal pain and distension. On postpartum day 2, she developed hypertension (peak blood pressure 151/100 mm Hg) and we started her on oral nifedipine XL (30 mg every 12 h). Based on clinical and laboratory abnormalities, we diagnosed preeclampsia complicated by hemolysis, elevated liver enzymes and low platelets (HELLP) syndrome (Table 1). We also diagnosed acute kidney injury after comparing her presenting creatinine level to her improved creatinine level postpartum in the absence of a pre-pregnancy baseline.

A CT scan on postpartum day 2 showed hypoattenuation consistent with hepatic infarction, likely as a complication of hepatic artery embolization, and stable appearance of both the hematoma and ovarian vein thrombosis (Figure 2). Her blood pressure was well controlled on oral nifedipine XL, and she was discharged on postpartum day 5 in stable condition.

Three weeks postpartum, the patient was seen in the obstetric internal medicine clinic. Her blood pressure ranged from 110 to 120/60 to 80 mm Hg and nifedipine was stopped. Eight weeks postpartum, she remained normotensive.

Discussion

Hypertensive disorders of pregnancy, including severe manifestations such as preeclampsia, are increasingly common, with a reported doubling in prevalence between 2000 and 2018 in the United States.[1] These disorders exist on a spectrum, including chronic or pre-existing hypertension, gestational hypertension and preeclampsia, with further characterization based on associated complications such as HELLP syndrome. According to the most recent guideline from the Society of Obstetricians and Gynecologists of Canada (SOGC), preeclampsia is diagnosed based on when hypertension is present with any one or more of new proteinuria; an adverse condition affecting the central nervous system, cardiorespiratory, hematologic, renal, hepatic or fetoplacental systems; or severe end-organ complications (SOGC Guideline No. 426 includes full diagnostic criteria).[2] The syndrome of HELLP is an uncommon complication in pregnancy, affecting 1% of pregnancies but occurring in 10%–20% of patients who develop preeclampsia.[3] It is typically associated with hypertension; however, 10%–20% of patients are normotensive.[4] Our patient met criteria for both preeclampsia (based on her elevated blood pressure and multiple adverse conditions and complications) and HELLP (based on evidence of hemolysis, elevated liver enzymes and low platelets), complicated by subcapsular liver hematoma. The initial biochemical abnormalities may have been explained by hemorrhage and prerenal acute kidney injury with intravascular depletion. However, the evolution of worsened anemia, thrombocytopenia, elevated liver enzymes and evidence of hemolysis, in parallel with development of hypertension, was most consistent with a unifying diagnosis of severe preeclampsia and HELLP syndrome, with associated complications of each including acute kidney injury, subcapsular liver hematoma and thrombosis.

Subcapsular liver hematoma is an uncommon complication of HELLP syndrome, reported in less than 2% of pregnancies with HELLP syndrome.[5] It can progress to liver rupture or infarct, which

can have devastating consequences for both the patient and baby if not promptly recognized and managed.[5] Patients can present with dyspnea; shoulder, back or epigastric pain; anorexia; or nausea and vomiting, as was the case for our patient.[5] Our patient was managed collaboratively by specialists in obstetrics and gynecology, general surgery, interventional radiology and obstetric internal medicine from an operative and medical perspective, given the atypical presentation, severe manifestations requiring emergent intervention and incidental finding of partially occlusive gonadal vein thrombosis.

The management options for subcapsular liver hematoma range from a conservative approach (i.e., hemodynamic support, transfusion and reversal of coagulopathy), to minimally invasive transcatheter hepatic artery embolization, to surgical intervention with packing, parenchymal suturing, resection or hepatic artery interruption.[6] Some patients require liver transplantation.[6] A recent systematic review reported maternal and fetal survival rates of 85% and 59% respectively, suggesting that these rates have remained largely unchanged over the last decade.[7] However, this is an improvement when compared with data collected before 2003, which estimated a maternal survival rate of 61%.[7] In a 2021 review of 73 patients with subcapsular liver hematoma, the best maternal and fetal survival rates were observed among those treated with hepatic artery embolization.[7] A minimally invasive approach with embolization should therefore be strongly considered as the first intervention if clinical stability allows, highlighting the importance of performing CT angiography in patients with HELLP syndrome with abdominal pain or elevated liver enzymes to facilitate timely diagnosis and management.[7] Computed tomography angiography provides both diagnostic information (particularly among patients who present with abdominal pain and abnormal liver enzymes, but who do not have indication for urgent cesarian delivery) and opportunity for minimally invasive therapy, and can be used to identify other pathology that may explain or be associated with the patient's presentation. Open surgical intervention has been suggested as a rescue option for patients who are clinically unstable or when CT angiography is unavailable.[7]

Gonadal vein thrombosis is rare, with an incidence of 0.05%–0.16% in all pregnancies, most commonly in the postpartum period. Of these thromboses, 90% occur in the right gonadal vein because of its length and dextrorotation of the gravid uterus, resulting in compression.[8] Risk factors include infection and cesarian delivery, particularly with the birth of multiples.[9] No consensus exists in the literature to guide treatment, and a recent review found no statistically significant correlation between use of anticoagulation and resolution of thrombosis.[8] The authors of the review suggested treatment only in the presence of symptoms, such as septic thrombophlebitis and concurrent deep vein thrombosis or pulmonary embolism. Current SOGC recommendations presume an infectious trigger for gonadal vein thrombosis and support the use of broad-spectrum antibiotics, as well as consideration of therapeutic anticoagulation.[9] The decision to treat with anticoagulation must be made on an individual basis, depending on the provoking factor, location and extent of thrombosis. For our patient, the left-sided location could suggest that the inflammatory and hypercoagulable states of preeclampsia and HELLP syndrome were more influential predisposing factors than anatomic changes associated with pregnancy. Based on the risk of recurrent bleeding in the setting of subcapsular liver hematoma and rupture, multifactorial anemia with evidence of hemolysis, the partially occlusive clot burden, the lack of extension into the inferior vena cava and the lack of evidence for an infectious trigger, we decided not to treat with anticoagulation or antibiotics.

We present a severe case of a patient with preeclampsia and HELLP — complicated by acute kidney injury, subcapsular hematoma and thrombosis — who initially presented with normal blood pressure, tachycardia and nausea and vomiting, and was found to have a hemoperitoneum intraoperatively. This case highlights the importance of having a high index of suspicion for preeclampsia or HELLP syndrome for patients with red-flag symptoms such as upper abdominal pain, headache, visual changes, altered mental status or dyspnea, and early biochemical signals, even in the absence of hypertension at presentation. Clinicians should consider prompt imaging and minimally invasive intervention if patient stability, resources and time permit.

References

1. Wen T, Schmidt CN, Sobhani NC, et al. Trends and outcomes for deliveries with hypertensive disorders of pregnancy from 2000 to 2018: a repeated cross-sectional study. *BJOG* 2022;129:1050-60.
2. Magee LA, Smith GN, Bloch C, et al. Guideline No. 426: Hypertensive disorders of pregnancy — diagnosis, prediction, prevention, and management. *J Obstet Gynaecol Can* 2022;44:547-71.e1.
3. Ditisheim A, Sibai BM. Diagnosis and management of HELLP syndrome complicated by liver hematoma. *Clin Obstet Gynecol* 2017;60:190-7.
4. Sibai BM. Diagnosis, controversies, and management of the syndrome of hemolysis, elevated liver enzymes, and low platelet count. *Obstet Gynecol* 2004;103:981-91.
5. Singh P, Warren K, Collier V. Ruptured subcapsular liver hematoma: a rare complication of HELLP syndrome. *Case Reports Hepatol* 2020;2020:8836329. doi:10.1155/2020/8836329.
6. Chandrasekaran S, Simon R. Hepatic complications in preeclampsia. *Clin Obstet Gynecol* 2020;63:165-74.
7. Gupta A, Joseph SR, Jeffries B. Managing a rare complication of HELLP syndrome in Australia: spontaneous liver haematoma in pregnancy. *Aust N Z J Obstet Gynaecol* 2021;61:188-94.
8. Plastini T, Henry D, Dunleavy K. Ovarian vein thrombus: To treat or not to treat? *Blood Adv* 2017;1:1120-3.
9. Chan WS, Rey E, Kent NE, et al.; Society of Obstetricians and Gynecologists of Canada. Venous thromboembolism and antithrombotic therapy in pregnancy. *J Obstet Gynaecol Can* 2014;36:527-53.

Competing interests: None declared.

This article has been peer reviewed.

The authors have obtained patient consent.

Affiliations: Cumming School of Medicine (McBride, Small, Hurd, Claassens), and Department of Medicine and Obstetrics and Gynaecology (Hurd), University of Calgary, Calgary, Alta.

Contributors: All of the authors contributed to the conception and design of the work, drafted the manuscript, revised it critically for important intellectual content, gave final approval of the version to be published and agreed to be accountable for all aspects of the work.

Correspondence to: S. McBride, susan.mcbride@albertahealthservices.ca