CASE REPORT

HELLP syndrome: a diagnostic conundrum with severe complications

Devika Rao,¹ Nikulkumar Kumar Chaudhari,² Robert Michael Moore,³ Belinda Jim¹¹Department of Medicine, Jacobi Medical Center, Bronx, New York, USA²Department of Medicine/ Nephrology, Jacobi Medical Center, Bronx, New York, USA³Jacobi Medical Center, Bronx, New York, USA

Correspondence to

Dr Belinda Jim,
belindajim286@gmail.com

Accepted 27 July 2016

SUMMARY

The HELLP (haemolysis, elevated liver enzymes, low platelets) syndrome is believed to be part of the spectrum of pre-eclampsia, which falls within the category of hypertensive disorders of pregnancy. Maternal and fetal complications are more severe in HELLP as opposed to pre-eclampsia alone. We describe a 26-year-old primigravida woman with no medical history who presents with signs of HELLP with marked transaminitis and mild disseminated intravascular coagulation at 35 weeks of gestation who required emergent delivery of the fetus; the patient also sustained acute kidney injury requiring continuous veno-venous hemodiafiltration and a prolonged intensive care unit admission. Remarkably, with supportive care, all laboratory derangements, including renal function, normalised after 4 weeks. We discuss the diagnostic conundrum when faced with the possible diagnosis of HELLP in discriminating from its many imitators in order to assume proper treatment.

BACKGROUND

Hypertensive disorders in pregnancy represent a large spectrum of disorders where the HELLP syndrome is on the severe end. HELLP is an acronym which refers to the triad of haemolysis with a microangiopathic blood smear, Elevated Liver enzymes and a Low Platelet count.¹ In this report, we present an unexpected case of HELLP syndrome with a complicated clinical course. We will then provide a detailed review of the most dangerous imitators of this condition and hope to illustrate the diagnostic dilemma that a clinician faces in diagnosing an atypical presentation of the HELLP syndrome.

CASE PRESENTATION

A 26-year-old primigravida with no significant medical history presented to obstetrics triage at 35 weeks of pregnancy reporting right upper quadrant abdominal pain for 4 hours prior. Her pregnancy thus far had been uneventful. The patient's initial vital signs at triage were unremarkable; however, the fetal tracing was concerning as it was read as 'Category III', which is abnormal and may be indicative of hypoxic risk to the fetus. Given the suspicion for placental abruption, the patient was consented for an emergent caesarean section (C-section) under general anaesthesia. The patient's intraoperative course resulted in the delivery in a live male neonate and was noted to have a gradual increase in blood pressure to 139/104 mm Hg immediately post C-section. After returning uterus to the abdomen,

pooling of blood in the pelvis was noted. Upon re-exploration of the surgical field, additional oozing of blood was noted from the subcutaneous tissue, though no source of bleeding was identified. An intraoperative haematocrit was found to be 16 g/dL. At this time, review of her laboratory findings from prior to C-section revealed a platelet count of 75 000 cells/mm³ and an ALT of 2002 U/L (table 1).

INVESTIGATIONS

Because of the persistent bleeding from the subcutaneous layer, there was concern for disseminated intravascular coagulation (DIC). A review of the coagulation panel obtained at triage was within normal limits (table 1). However, repeat blood work at the end of C-section demonstrated a significant decrease in the haemoglobin from 11.6 to 6.1 g/dL, a decrease in platelet count to 48 000 cells/mm³ and an increase in prothrombin time (PT)/international normalised ratio (INR) from 11.1/1.1 to 13.3/1.3. There was also a decrease in fibrinogen from 289 to 166 mg/dL. Her bilirubin had increased from 2.3 mg/dL at triage to 3.0 mg/dL immediately post C-section. Her peripheral smear demonstrated 1–2 schistocytes per high-power field with decreased number of giant platelets. Her ALT remained high at 1252 U/L. Postoperatively, the patient's urine appeared to be dark red with a decreased output rate of 10–20 cc/hour, though her blood urea nitrogen (BUN)/creatinine remained normal at 11/0.7 mg/dL. Her urine sediment revealed numerous muddy brown casts with monomorphic red blood cells. The patient remained intubated and sedated. By this time, blood clots were noted in the oro-gastric tube output, the abdominal dressing and the vagina. Given the significant decrease in haemoglobin and concern for haemolysis, the massive transfusion protocol was initiated. A diagnosis of severe HELLP syndrome with clinical DIC was made.

DIFFERENTIAL DIAGNOSIS

Pre-eclampsia/HELLP syndrome is a pregnancy-specific condition that needs to be distinguished from other common pregnancy-related complications with similar features such as acute fatty liver of pregnancy (AFLP), thrombotic thrombocytopenic purpura (TTP), atypical haemolytic-uraemic syndrome (aHUS), lupus flare and antiphospholipid syndrome (APS). All of these conditions are associated with high maternal mortality as well as various degrees of long-term sequelae. Clinically, the HELLP syndrome may be confused with AFLP as they can present with elevated levels of

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To cite: Rao D, Chaudhari NK, Moore RM, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2016-216802

Table 1 Vital signs and laboratory parameters

	Pregnancy—week 30 (baseline)	On triage (prior to C-section)	Immediately following C-section	Following massive transfusion	2 days postoperative
Temperature (°F)	98.6	98.6	95.9	98.8	98.4
Heart rate (bpm)	94	106	93	92	108
Blood pressure (mm Hg)	119/69	118/88	150/97	162/97	135/87
Respiratory rate (breaths/min)	12	17	16	18	19
SpO ₂ (%)	100 on room air	100 on room air	100 on mechanical ventilator, FiO ₂	100 on mechanical ventilator, FiO ₂	100 on mechanical ventilator, FiO ₂
WCC (cells/mm ³)	7700	9500	6900	5600	9100
Haemoglobin (g/dL)	12.2	11.6	6.1	10.6	9.9
Haematocrit (%)	36.7	37.2	18	31.8	30.3
Platelets (cells/mm ³)	215 000	75 000	48 000	56 000	135 000
Sodium (mEq/L)	136	134	138	141	145
Potassium (mEq/L)	4	Haemolysed	6.2	4.3	4.5
Chloride (mEq/L)	106	104	107	110	117
Bicarbonate (mEq/L)	22.4	22.2	17.2	22.3	19.1
BUN (mg/dL)	3	10	11	15	28
Creatinine (mg/dL)	0.4	0.8	0.7	1.7	5.2
PT/INR	10/1	11.1/1.1	13.3/1.3	11.9/1.1	11.3/1.1
aPTT	25.6	30.2	33.3	28.1	32.9
Fibrinogen (mg/dL)	NA	289	166	322	446
Albumin (g/dL)	4.2	3.7	2.8	2.6	2.4
AST (U/L)	13	Haemolysed	Haemolysed	1545	154
ALT (U/L)	12	2002	1252	486	33
Total bilirubin (mg/dL)	0.7	2.3	3.0	8.2	7.6
Direct bilirubin (mg/dL)				5.8	
Lactate			5		
Peripheral smear		Normochromic, few target cells appreciated. 1–2 schistocytes per HPF. Decreased platelets. Giant platelets noted. Neutrophils with toxic granulations. Few large granulocytic lymphocytes are also appreciated.			

ALT, alanine transaminase; aPTT, activated partial thromboplastin time; AST, aspartate aminotransferase; BUN, blood urea nitrogen; HPF, high-power field; INR, international normalised ratio; PT, prothrombin time; WCC, white cell count.

AST/ALT, alkaline phosphatase, bilirubin, white cell count and low platelets, and signs of DIC with possible renal failure. However, hypoglycaemia is a specific finding to AFLP which is not present in HELLP. Their symptoms differ in that pre-eclampsia/HELLP usually presents with right upper quadrant or epigastric pain or headaches, while AFLP demonstrates nausea, anorexia, jaundice, ascites and encephalopathy, though there may be significant overlap.² Given that haemolysis in HELLP is a micro-angiopathic process, it may also be confused with the renal thrombotic microangiopathies (TMAs) which include TTP and HUS characterised by disseminated occlusion of arterioles and capillaries with fibrin and/or agglutinated platelets. A deficiency in a disintegrin and metalloprotease with thrombospondin-1-like domains (ADAMTS13), a von Willebrand factor-cleaving protease, has explained a fraction of TMAs.³ On the other hand, patients with atypical HUS (aHUS) exhibit mutations in genes encoding the complement regulatory proteins that cause a heightened activation of the alternative complement pathway.⁴ Interestingly, pregnancy itself can be a trigger for ADAMTS-13 deficiency TTP⁵ and aHUS.⁶ The timing of TTP is usually in the second or third trimester and in the postpartum period for aHUS. Though abnormalities in liver enzymes may be abnormal in TTP/aHUS, they are usually not prominent and overshadowed by the microangiopathic anaemia and thrombocytopenia which would help to differentiate them from HELLP. Perhaps the most obvious diagnosis to exclude is a lupus flare, since liver pathology is generally not present in

lupus. However, the APS with or without lupus may present with liver dysfunction; despite this, the dominant features are thromboses, either of the venous system or of arterial system and recurrent pregnancy loss. Due to significant overlap and concurrent diagnoses, it is crucial to have the clinical expertise to make an accurate diagnosis given different treatment and long-term implications. For example, delivery is the only option for pre-eclampsia/HELLP and AFLP. However, plasma exchange is appropriate for ADAMTS-13-deficient TTP,⁷ while the new agent eculizumab, a targeted inhibitor of complement protein C5, has been found to be effective in aHUS.^{3, 8} For a lupus flare, administration of immunosuppressor is necessary,⁹ while anticoagulation is the treatment of choice in APS. A comprehensive guideline is depicted in [table 2](#).

TREATMENT

In the postanesthesia care unit, the patient continued to receive blood products as well as tranexamic acid, an antifibrinolytic agent to treat excessive blood loss. Due to the patient's continued decrease in haemoglobin, it was decided to re-explore the abdomen for source of bleeding in the operating room. At that time, she was noted to be normothermic with improved coagulation parameters ([table 1](#)). Upon exploration, the abdominal wound was found to have ~100 cc of clot which was irrigated out. Only minor bleeding was identified with no obvious signs of vessel injury. Approximately 800 cc of frank blood was suctioned from the abdominal cavity. There was again noted to be

Table 2 Differentiating among the imitators of pre-eclampsia^{7 10–12}

	Pre-eclampsia	HELLP	TTP-HUS	aHUS	AFLP	APS	Lupus flare
Timing in pregnancy	>20 weeks	>20 weeks	Higher incidence in second trimester	Higher in postpartum	Higher incidence in third trimester	All gestational ages	All gestational ages
Blood pressure	Elevated	Variable	Variable	Elevated	Variable	Variable	Variable
Neurological involvement	Variable	Unlikely	Common	Unlikely	Unlikely	Variable	Variable
Fever	Absent	Absent	Present	Variable	Absent	Present	Present
Blood smear	Normal	Moderate schistocytes	Many schistocytes	Schistocytes	Occasional schistocytes	Schistocytes	Pancytopenia
Low platelet count (cells/ μ L)	None	Moderate to severe	Moderate to severe	Severe	Mild to moderate	Moderate	Mild to moderate
Elevated liver enzymes	None	Moderate to severe	Normal to mild	Mild	Moderate to severe	Normal to mild	None
Proteinuria	Mild to moderate	Severe	Mild to moderate	Mild to moderate	mild	Normal to severe	Mild to moderate
ADAMTS13 activity	NA	Low	Very low	Low	NA	NA	NA
Treatment	Delivery of fetus	Delivery of fetus	Plasma exchange	Plasmapheresis/eculizumab	Delivery of fetus	Aspirin +anticoagulation	Immunosuppression

ADAMTS13, a disintegrin and metalloprotease with a thrombospondin type 1 motif, member 13; AFLP, acute fatty liver of pregnancy; APS, antiphospholipid syndrome; HELLP, haemolysis, elevated liver enzymes, low platelets; NA, not applicable; TTP-HUS, thrombotic thrombocytopenic purpura-haemolytic uraemic syndrome.

pooling of blood from the pelvis around the previous C section site. Upon opening the fascia of the Pfannenstiel incision, dense clots were found overlying the surface of the C-section scar. The areas were packed and cauterised followed by closure of the abdomen.

OUTCOME AND FOLLOW-UP

The patient was then transferred to the surgical intensive care unit where her haematocrit level had stabilised but her low platelet count required additional transfusions. She further developed acute kidney injury in setting of HELLP syndrome and was aggressively resuscitated with intravenous crystalloids and colloids and was administered a loading dose of intravenous magnesium sulfate to prevent seizures. However, her renal function continued to worsen and peaked at a BUN level of 41 mg/dL and a serum creatinine of 6.7 mg/dL with a poor urine output rate of 25–35 cc/hour. The aggressive fluid resuscitation thus led to peripheral oedema and pleural effusions. She subsequently required temporary renal replacement therapy with continuous veno-venous hemodiafiltration (CVVHDF) for 48 hours. The patient was able to be transitioned off CVVHDF to intravenous furosemide therapy and extubated on post-operative day 7. She slowly recovered her renal function and was eventually discharged home on postoperative day 16 with a serum creatinine of 2.0 mg/dL. Her follow-up in the outpatient renal clinic four weeks later showed complete resolution of her renal failure with a serum creatinine of 0.7 mg/dL. The patient was also counselled to have appropriate pre-natal care with a maternal fetal specialist for future pregnancies.

DISCUSSION

While the acronym HELLP itself was not coined until 1982,¹³ descriptions of the syndrome can be found in literature dating back to the 1950s.¹¹ HELLP syndrome is estimated to complicate 0.1% to 0.8% of pregnancies, while 10% to 20% of HELLP occur with severe pre-eclampsia.¹⁴ AKI is more commonly associated with HELLP, complicating 7–15% of cases, as opposed to pre-eclampsia alone without HELLP at 1%. HELLP is widely considered to be a severe form of pre-eclampsia yet up to 15–20% patients with HELLP do not have antecedent signs

or symptoms of pre-eclampsia.¹⁵ Thus there is considerable debate whether HELLP is a completely separate entity or part of the spectrum of pre-eclampsia. Part of the confusion is derived from the lack of standardised criteria for diagnosis, with variations from time of onset, to different types and degrees of laboratory abnormalities. HELLP has been considered anything from “an early form of severe preeclampsia”¹⁶ to a “unique variant”¹⁷ to “misdiagnosed preeclampsia”¹⁸ to “mild disseminated intravascular coagulation”.¹⁸ The pathophysiology of HELLP is not entirely clear. There is no known precipitating cause but appears to be associated with pre-eclampsia, the latter of which is known to have pathological vascular lesions in multiple organs, such as the liver, the kidney, and the placenta. The HELLP syndrome is a constellation of clinical and pathological manifestations of endothelial damage resulting from platelet activation, the release of thromboxane A₂, and reduced prostacyclin production all leading to worsened platelet agglutination and aggregation.¹⁹ HELLP may also be associated with DIC, and in fact some investigators believe that DIC is the primary process.^{20 21} Although there may be similarities in the histology of microthrombi seen in HELLP and DIC, most patients with HELLP do not exhibit these findings. Typically, signs and symptoms of HELLP develop between 28 and 36 weeks of gestation (70%) or within 48 hours postpartum (30%).¹⁹ Besides immediate delivery of the fetus, there is no specific treatment for pre-eclampsia/HELLP. The use of corticosteroids has been studied with variable results. A Cochrane review showed that there was essentially no difference in the risk of maternal death, severe maternal morbidity or perinatal/infant death between corticosteroids and placebo or no treatment.²² The additional use of magnesium sulfate as a preventive measure to halt the progression to eclampsia is recommended,²³ as is blood pressure control with pregnancy-safe antihypertensive medications to avoid a cerebrovascular event.²⁴ Recently, there was a case report of treating a woman with severe pre-eclampsia/HELLP at 26 weeks of gestation with eculizumab that resulted in the complete normalisation of liver enzymes and haematological parameters by treatment day 6 and prolongation of her pregnancy by 17 days.²⁵ Though the mechanism of improvement is unclear, it is believed that mitigating the ongoing inflammatory

response via complement proteins is a factor. Ongoing research in mechanism of disease will help to create a more targeted treatment approach. Whether HELLP will recur in subsequent pregnancies is often a subject of great concern to affected mothers. Despite limited data, a recent meta-analysis shows a general recurrence rate of 20.7% of any type of hypertensive disorders of pregnancy (pre-eclampsia, gestational hypertension, HELLP, small for gestational age), but the rate is magnified to 36.3% of any type of hypertensive disorder if the index pregnancy was affected by HELLP. The actual recurrence of an HELLP pregnancy, however, is low at 7.2%.²⁶ There is now considerable evidence of long-term cardiovascular outcomes in patients afflicted with pre-eclampsia, and if we believe, the HELLP is part of the spectrum of pre-eclampsia, these effects would apply as well.^{27–28} Chronic hypertension developed in 22–33% of HELLP patients after ~5 years.^{29–30} Further metabolic derangements such as diabetes, obesity, thyroid disease, renal disease have been described as long-term maternal complications; however, these reports are retrospective without a comparative group.^{29–30} Although not commonly reported, the psychological effects of pre-eclampsia/HELLP including anxiety and depression are increased, especially in those who are anticipating additional pregnancies.^{29–30} Thus, the postpartum care of these women for physical and mental health become paramount. Our case describes how catastrophically HELLP may occur, from an uneventful pregnancy with no risk factors to multiorgan failure within hours despite delivery of the fetus, with full recovery within 1 month of delivery.

Learning points

- ▶ Signs and symptoms of HELLP may be confused with acute fatty liver of pregnancy, TTP/HUS/aHUS, lupus flare, antiphospholipid syndrome.
- ▶ Clinical onset is unpredictable.
- ▶ Delivery is the treatment of choice for HELLP.
- ▶ Complete resolution of end-organ involvement is possible with aggressive supportive care.
- ▶ Significant cardiovascular outcomes after an episode of HELLP may ensue for decades afterwards.

Contributors DR, NKC and BJ have cared for the patient and written the manuscript. RMM has cared for the patient and edited the manuscript.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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