RESEARCH PAPER

HELLP Syndrome with Postpartum Eclampsia, Disseminated Intravascular Coagulation, and Acute Kidney Injury Requiring Renal Replacement Therapy: A Case Report

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ABSTRACT

Background:
HELLP syndrome is a severe hypertensive disorder of pregnancy that may present antepartum or postpartum and can rapidly progress to multiorgan failure. Its coexistence with postpartum eclampsia, disseminated intravascular coagulation (DIC), haemorrhagic shock, and acute kidney injury (AKI) is uncommon but life-threatening.

Case summary:
A 23-year-old primigravida delivered vaginally at term outside our centre and developed a generalized tonic-clonic seizure with altered sensorium about 10 minutes after delivery. She was intubated at the referring facility, received loading dose of magnesium sulphate and blood products, and was transferred to our tertiary care centre in shock, oligo-anuria, severe thrombocytopenia, jaundice, and biochemical evidence of HELLP syndrome. She required vasopressor support, mechanical ventilation, repeated correction of coagulopathy with packed red cells, fresh frozen plasma, platelets, and cryoprecipitate, treatment for secondary postpartum haemorrhage, and multiple heparin-free Sustained Low-Efficiency Dialysis (SLED) sessions for AKI. Neurology, nephrology, critical care, respiratory medicine, cardiology and gastroenterology teams were involved. Her haemodynamics, coagulation profile, liver function, and urine output improved gradually, and she was discharged after a prolonged but successful recovery.

Conclusion:
This case highlights that HELLP syndrome with postpartum eclampsia, DIC, haemorrhagic shock, and AKI can be survivable when managed early and aggressively in a multidisciplinary tertiary-care setting. Serial laboratory monitoring, timely blood product replacement, and renal replacement therapy were central to the favourable outcome.

Keywords: HELLP syndrome; postpartum eclampsia; disseminated intravascular coagulation; haemorrhagic shock; acute kidney injury; postpartum haemorrhage

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Introduction

HELLP syndrome, an acronym for Haemolysis, Elevated Liver enzymes, and Low Platelet count, is a severe variant of pre-eclampsia with superimposed hepatic and haematological dysfunction.[1-3] It complicates approximately 0.5–0.9% of all pregnancies and carries a maternal mortality of 1–3% in developed settings, with significantly higher rates in resource-limited environments.[1,3,4] The syndrome may manifest antepartum, intrapartum, or postpartum, with nearly one-third of cases presenting after delivery.[1,2,3]

When HELLP syndrome coexists with eclampsia, DIC, and haemorrhagic shock, the case complexity escalates dramatically.[4] The underlying endothelial dysfunction of pre-eclampsia precipitates a cascade of platelet activation, microangiopathic haemolytic anaemia, hepatocellular injury, and consumption coagulopathy.[1,2] The resulting DIC propagates haemorrhage, which in

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turn worsens tissue hypoperfusion, leading to multi-organ dysfunction including acute kidney injury (AKI).[6] AKI requiring renal replacement therapy (RRT) in this context further burdens an already critically ill obstetric patient and is associated with significant short- and long-term morbidity.[8,9]

Successful management requires early recognition, prompt correction of the coagulopathy with blood products, vasopressor and mechanical ventilatory support, and seamless coordination across obstetrics, critical care, nephrology, neurology, and other specialties.[6]

Case presentation

Patient information and obstetric history

A 23-year-old woman, primigravida, underwent a term vaginal delivery at an outside hospital. She had been diagnosed with gestational hypertension 4 days before delivery and had been started on antihypertensive treatment. About 10 minutes after delivery, she developed altered sensorium and a single generalized convulsion with tongue bite, consistent with postpartum eclampsia. Loading dose of magnesium sulphate was administered, she was intubated for airway protection, and blood products were transfused at the referring centre. She was transferred to our tertiary care centre because of persistent haemodynamic instability, oliguria, jaundice, and suspected DIC and AKI.

Examination on arrival

On arrival she was intubated, drowsy, tachycardic, and vasopressor-dependent. She had pallor, icterus, and generalized oedema. Chest examination revealed bilateral basal crepitations. The abdomen was distended but soft, and the uterus was well contracted. Initial assessment suggested severe postpartum illness with multiorgan dysfunction, and the clinical working diagnosis was HELLP syndrome with postpartum eclampsia, DIC, haemorrhagic shock, and AKI.

Key investigations

Parameter	Value(s)	Reference	Interpretation
Haemoglobin	6.9 g/dL; nadir 5.5 g/dL	12 - 16 g/dL	Severe anaemia requiring transfusion
Platelets	24 x 10^3/µL; later 59-97 x10^3/µL	150 - 400 x10^3/µL	Marked thrombocytopenia consistent with HELLP/DIC
AST/ALT	AST 1677-2494 IU/L; ALT up to 1325 IU/L	<40 IU/L	Severe hepatocellular injury
Total bilirubin	8.46 - 9.75 mg/dL	0.3 - 1.2 mg/dL	Significant jaundice/haemolysis
Creatinine	2.13 mg/dL rising to 3.17 mg/Dl	0.5 - 1.1 mg/dL	Oligo-anuric AKI
INR	1.51 - 1.72	<1.2	Consumptive coagulopathy
D-dimer	3783 - 5098 ng/mL	<500 ng/mL	Marked activation of coagulation/fibrinolysis
Fibrinogen	232 - 261 mg/dL	200 - 400 mg/dL	Low-normal, supported by replacement

Relevant imaging and procedural findings. Echocardiography showed preserved cardiac function (ejection fraction about 60%). Chest radiography demonstrated minimal bilateral pleural effusions. Ultrasound of the abdomen showed intrauterine clots with postpartum uterine changes and minimal ascites. Brain CT/MRI showed no acute intracranial lesion. Because renal function remained impaired despite supportive care, a renal biopsy was performed during the later stage which was suggestive of acute tubular necrosis (ATN).

Management and clinical course

The patient was managed in the intensive care unit with mechanical ventilation, noradrenaline infusion, magnesium sulphate already started at the referring centre, antiepileptic therapy, broad-spectrum antibiotics, strict input-output monitoring, and serial laboratory assessment. A femoral haemodialysis catheter was inserted and heparin-free SLED was initiated because of oligo-anuric AKI and haemodynamic instability.

The patient required massive transfusion support during her ICU stay, including 9 units of packed red blood cells, 6 units of fresh frozen plasma, 4 units of cryoprecipitate, 9 units of random donor platelets and 4 units of single donor platelet for correction of severe anemia, hemorrhagic shock, and disseminated intravascular coagulation, refractory thrombocytopenia, and consumptive coagulopathy.

On the second hospital day at our centre, she developed secondary postpartum bleeding. Examination showed bleeding from the uterine cavity without cervical or vaginal tear. She was treated conservatively with uterotonics and tranexamic acid, and interventional radiology opinion was sought. Her DIC parameters gradually improved with continued transfusion support. Vasopressor requirement decreased over the next few days, and ventilatory support was gradually weaned to non-invasive support and then room air.Renal

dysfunction remained the most persistent organ failure. 9 SLED sessions were required initially, followed by 4 intermittent haemodialysis sessions. A right internal jugular tunneled dialysis catheter was placed after initial stabilization. Urine output eventually improved from oligo-anuria to more than 2 L/day. She underwent renal biopsy for definitive assessment of the kidney injury which was suggestive of ATN. The dialysis catheter was later removed uneventfully, and she was discharged in stable condition after a prolonged recovery.

A multidisciplinary team approach was central to the patient's management. The Obstetrics and Gynaecology team coordinated overall care, managed postpartum hemorrhage, and initiated uterotonic and interventional measures. Critical Care specialists supervised ventilatory support, vasopressor therapy, and intensive care management. The Nephrology team managed acute kidney injury with vascular access placement, repeated SLED/hemodialysis sessions, and renal biopsy. Neurology contributed to seizure evaluation and antiepileptic management. General Medicine assisted in managing shock, DIC, and antimicrobial optimization. Gastroenterology evaluated hepatic dysfunction associated with HELLP syndrome, while Cardiology performed hemodynamic and cardiac assessment. Respiratory Medicine guided ventilator weaning and non-invasive ventilation support. Interventional Radiology was consulted for management of secondary postpartum hemorrhage.

Discussion

A. Pathophysiology and Diagnostic Assessment

This case illustrates the complex interaction of HELLP syndrome, postpartum eclampsia, DIC, secondary PPH, and AKI, all contributing to progressive multiorgan dysfunction.[1,2] HELLP syndrome is characterized by hemolysis, elevated liver enzymes, and thrombocytopenia.[1-3] Our patient fulfilled Tennessee Class I HELLP criteria with severe thrombocytopenia ($24 \times 10^3/\mu L$), markedly elevated transaminases (AST 1677–2494 IU/L; ALT 1325 IU/L), hyperbilirubinemia, and hemolytic features.

The coagulopathy was likely multifactorial. HELLP syndrome itself can precipitate DIC through endothelial injury and platelet activation, while secondary PPH further aggravates consumptive coagulopathy.[6] Elevated INR and D-dimer with relatively preserved fibrinogen suggested evolving compensated-to-decompensated DIC.

Postpartum eclampsia occurring shortly after delivery reflected persistent cerebral endothelial dysfunction associated with severe pre-eclampsia and HELLP syndrome.[1,4] Up to 40% of eclamptic seizures occur postpartum.[4,5] Normal neuroimaging excluded structural intracranial pathology.

AKI was probably secondary to a combination of renal hypoperfusion, thrombotic microangiopathy, and acute tubular necrosis.[8,9] The patient fulfilled KDIGO Stage 3 AKI criteria with oligo-anuria and rising creatinine despite supportive care.[9] Gradual recovery of urine output following dialysis suggested reversible intrinsic renal injury.

B. Management Highlights

Management required aggressive multidisciplinary critical care. Massive transfusion with packed red blood cells, plasma, platelets, and cryoprecipitate was guided by serial DIC monitoring and principles of balanced component therapy used in severe obstetric haemorrhage.[6,7]

Heparin-free SLED was chosen because of active DIC and bleeding risk. Renal replacement therapy is recommended in haemodynamically unstable obstetric AKI patients with severe metabolic derangement or persistent oligo-anuria.[8,9]

Secondary PPH was successfully managed conservatively using uterotonics, tranexamic acid, and close monitoring, consistent with WHO recommendations.[7] Vasopressor support and mechanical ventilation were gradually weaned as haemodynamic and respiratory status improved.

Magnesium sulphate remained the primary therapy for eclampsia, while levetiracetam and lacosamide were added because of severe neurological presentation and critical illness.[5]

C. HELLP Syndrome versus PPH as the Primary Driver of DIC

Although both HELLP syndrome and PPH contributed to DIC, the clinical sequence favored HELLP syndrome as the initiating pathology. Severe thrombocytopenia, transaminitis, and coagulopathy were already present before significant postpartum bleeding occurred.[6] Secondary PPH likely worsened the pre-existing consumptive coagulopathy. Clinically, however, both conditions acted synergistically and required simultaneous management.

D. Outcome and Learning Points

The patient recovered after prolonged intensive care with resolution of DIC, improvement in renal function, and no residual neurological deficit. This case highlights that severe HELLP syndrome complicated by postpartum eclampsia, DIC, haemorrhagic shock, and AKI can be successfully managed with early referral, multidisciplinary care, serial laboratory monitoring, timely blood product replacement, and renal replacement therapy.[6,8]

Heparin-free SLED may be particularly useful in haemodynamically unstable obstetric patients with active coagulopathy.[8,9] The case also emphasizes the importance of vigilance for postpartum eclampsia and HELLP syndrome even after delivery.

Conclusion

We present a rare and complex case of a 23-year-old primigravida who, following an outside full-term vaginal delivery, developed postpartum eclampsia, HELLP syndrome, DIC, haemorrhagic shock, and oligo-anuric AKI within hours of delivery. Through rapid transfer to a tertiary care centre and coordinated multidisciplinary management including massive transfusion, heparin-free SLED, vasopressor therapy, mechanical ventilation, and treatment of secondary

PPH, the patient made a remarkable recovery, being discharged neurologically intact with improving renal function on day 25.

This case reinforces the importance of early tertiary referral and multidisciplinary critical care in severe HELLP syndrome complicated by DIC, postpartum eclampsia, and AKI. Serial coagulation monitoring and evidence-based blood product replacement remain essential in managing concurrent HELLP syndrome, DIC, and postpartum haemorrhage.[6,7]

Patient consent

Written informed consent for publication of the case and accompanying clinical details should be confirmed before submission. Identifying information has been removed from this draft.

Declarations:

Conflicts of interest: None declared.

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Data availability: All case details are contained in the manuscript and source records.

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