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# Post-partum eclampsia complicated by HELLP syndrome: a case report

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## Abstract

**Introduction:** HELLP syndrome is a variant of pre-eclampsia with severe features that presents with haemolysis, elevated liver enzymes and low platelets. It is associated with significant morbidity and mortality.
**Case presentation:** A 24 year old Para 3+0 Gravida 4 at term with prolonged labour and severe pre-eclampsia presented from a peripheral facility without a referral letter. She delivered before arrival and had one convulsion prior to admission. Magnesium sulphate and antihypertensives were initiated at admission. A few hours later, she convulsed two more times, her level of consciousness deteriorated; she developed a bleeding diathesis and went into renal shutdown. At this point, magnesium sulphate was stopped in favour of phenytoin. Steroids were administered and acute haemodialysis was performed. Her neurological status and renal function improved and the HELLP syndrome resolved over the next five days. She was observed for three more days and was discharged in good condition, with no adverse sequelae noted during follow up.
**Result:** Prompt diagnosis and timely conscientious treatment were the keys to the good outcome in this patient.
**Conclusion:** A high index of suspicion of HELLP syndrome is essential in all patients with hypertensive disorders of pregnancy in order to enable early diagnosis and prompt management.

**Key words:** HELLP syndrome, Post-partum eclampsia, High index of suspicion, Early diagnosis

## Introduction

HELLP syndrome is a variant of severe pre-eclampsia characterized by haemolysis, elevated liver enzymes and low platelets (1). It occurs in 10 – 20% of women with pre-eclampsia (2). Despite being intertwined with severe pre-eclampsia and eclampsia, its aetiology and pathogenesis remain unclear (1). An association has been made between pre-eclampsia and cardiovascular disease (3-5). HELLP syndrome is often misdiagnosed due to its variable presentation (1,6), often resulting in high morbidity and mortality (7). The highest levels of mortality occur in low-income countries (6), possibly due to difficulties in establishing the diagnosis and inadequacies in management approach. Diagnosis is made by objectively demonstrating haemolysis, hepatocellular damage and thrombocytopenia (1).

## Case presentation

A 24 year old Para 3+0 was sent to Kenyatta National Hospital (KNH) from a peripheral facility without a referral letter due to poor progress of labour and severe pre-eclampsia at term. While en route to the referral hospital, she delivered a live female infant who cried immediately after birth. She then suffered a generalized tonic-clonic seizure. After the seizure, she was reported to have developed malaise, headache and epigastric pain but denied difficulty in breathing and visual disturbances.

Her antenatal card revealed an uneventful antenatal period. Her haemoglobin level was 11.5g/dl, she tested negative for syphilis and Human Immunodeficiency Virus (HIV), and had been normotensive. There was no significant past medical or family history. Her history was corroborated by her sister. Examination revealed a young sick-looking, confused post-partum mother who was not pale, jaundiced, or in respiratory distress. Her blood pressure was 182/106 mmHg, with a pulse rate of 126 beats per minute, respiratory rate of 16 breaths per minute and a temperature of 36.9$^{o}$C. The uterus was well contracted, lochia loss minimal and she had moderate bipedal pitting oedema. She had no focal neurological deficits. Other systems were normal. An initial admission diagnosis of post- partum eclampsia was made. She was catheterized to facilitate strict input-output monitoring. Magnesium sulphate and antihypertensive therapy was initiated with oral methyldopa (500 mg thrice a day) and hydralazine (5mg slow IV bolus then 25mg orally thrice a day). Blood samples were taken for full blood counts, a peripheral blood film, renal and hepatic function testing.

Four hours later, she convulsed twice despite magnesium sulphate infusion, and her neurological status deteriorated from simple confusion to responding only to pain. She also developed a bleeding diasthesis,

with prolonged bleeding from her tongue and sites of intravenous catheter insertion. Bedside clotting time was 17 minutes. This raised the index of suspicion for HELLP syndrome. She became oliguric with frank haematuria. Magnesium sulphate was stopped, and phenytoin was administered intravenously at a maintenance dose for 24 hours after the last convulsion.

Table 1 shows the renal and hepatic function profile from admission to discharge. The renal and hepatic functions were deranged, and the initially elevated indirect bilirubin suggested haemolysis. There was gradual improvement and restoration of normal function during the course of therapy. The electrolytes remained within acceptable ranges.

**Table 1:** Renal and hepatic function profile from admission to discharge

| Parameter | Day 1 | Day 2 | Day 4 | Day 6 | Day 8 | Ref Range |
|---|---|---|---|---|---|---|
| Urea(mmol/L) | 26.2 | 28.7 | 20.6 | 15.7 | 7.8 | 1.5 – 6.0 |
| Na+(mmol/L) | 147.0 | 145.3 | 143.7 | 141.9 | 137.6 | 135.0 – 145.0 |
| K+(mmol/L) | 4.8 | 4.1 | 4.0 | 3.7 | 3.8 | 3.5 – 5.0 |
| Creatinine (umol/L) | 472.0 | 546.0 | 356.0 | 237.0 | 108.0 | 35.0 – 80.0 |
| ALT (u/L) | 42.0 | 78.0 | 50.0 | 35.0 | 28.0 | 0.0 – 34.0 |
| AST (u/L) | 76.0 | 84.0 | 67.0 | 48.0 | 30.0 | 0.0 – 31.0 |
| TBIL(umol/mL) | 141.7 | 220.0 | 150.7 | 100.9 | 58.1 | 0.0 – 17.0 |
| DBIL(umol/mL) | 83.7 | 96.8 | 72.7 | 54.3 | 25.7 | 0.0 – 7.0 |
| IBil (umol/mL) | 62.0 | 123.2 | 78.0 | 46.6 | 32.4 | 0.0 – 10.0 |
| TPROT (g/L) | 73.0 | 54.0 | 57.0 | 65.0 | 70.0 | 62.0 – 83.0 |
| ALB (g/L) | 34.0 | 22.0 | 26.0 | 29.0 | 33.0 | 35.0 – 54.0 |

*Key: ALT - Alanine Transaminase; AST – Aspartate Transaminase; TBIL – Total Bilirubin; DBil – Direct Bilirubin; IBil – Indirect Bilirubin; TPROT – Total protein; ALB - Albumin*

Table 2 shows the results of the full blood counts, which depicted severe thrombocytopenia, with subsequent haemogram assessment showing improvement in the haematological parameters with management.

**Table 2:** Full blood counts from admission to discharge

| Parameter | Day 1 | Day 2 | Day 5 | Day 7 | Ref. Range |
|---|---|---|---|---|---|
| Leukocytes | 11.6 | 13.3 | 10.7 | 10.5 | 4.0 – 11.0 x 109 |
| Neutrophils | 85.0 | 81.0 | 90.0 | 72.0 | 40 – 70% |
| Hemoglobin (g/dL) | 13.6 | 11.4 | 10.8 | 12.4 | 11 – 14g/dL |
| Mean corpuscular volume(fL) | 84.1 | 79.4 | 80.2 | 86.7 | 76 – 94 fL |
| Platelets | 95.0 | 53.0 | 153.0 | 175.0 | 150 – 450 x 1012 |

The peripheral blood film done at admission showed schistocytes, target cells and burr cells, providing more objective evidence of haemolysis. A repeat film on the fifth post-admission day was normal and showed adequate platelets. These investigations confirmed the diagnosis of post-partum eclampsia complicated with HELLP syndrome.

Following the establishment of the diagnosis, further management included acute haemodialysis, transfusion with three units of fresh frozen plasma, two units of whole blood, two 12 mg intramuscular doses of dexamethasone and maintenance doses of anti-hypertensive medications. By Day 5, after the third session of haemodialysis, she was fully conscious, the blood pressure had stabilized, haematological, hepatic and renal functions improved (Tables 1 and 2) and her fluid input and output normalized. These findings were indicative of resolution of HELLP syndrome. She was observed for another three days and then discharged in good condition. Subsequent clinic reviews revealed no adverse sequelae in the mother and child.

## Discussion

Since the aetiology and pathogenesis of HELLP syndrome remains uncertain, diagnosis depended on a high index of suspicion. The entry point into the suspicion of the problem was the haemorrhagic diasthesis, which dictated a need for full investigation to confirm the specific diagnosis. Being a multi-system disorder, outcomes are optimized through objective investigations and monitoring of functions of affected organs in order to prevent any possible permanent damage. In the presence of the often dramatic presentation of eclampsia, it is possible to miss the diagnosis of HELLP syndrome.

Targeted treatment in this patient focused on prevention of convulsions, restoration of renal function, correction of bleeding diasthesis, monitoring of hepatic function and optimization of blood pressure control. Since she had already delivered, her management was essentially supportive to prevent permanent systemic damage. Derangement of renal function dictated a need to replace magnesium sulphate with phenytoin (8). Although there are no significant differences reported in maternal and perinatal mortality following administration of steroids in HELLP syndrome, the repeated observation that patients with HELLP syndrome on steroids show faster rise in platelet counts than those not on steroids (9,10) may possibly be due to the anabolic effect of steroids, which might enhance thrombopoiesis.

## Conclusion

Awareness of the existence of HELLP syndrome, a high index of suspicion and prompt appropriate treatment were the keys to the good outcome of this patient. These tenets remain the focal points in preventing morbidity and mortality from HELLP syndrome.

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*Conflict of Interest:* The authors declare that this is an original article that has not been submitted elsewhere for consideration for publication, that there is no conflict of interest and that the authors contributed equally to the writing of this paper.

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