

CASE REPORT

Acute renal failure due to HELLP syndrome and acute renal failure in mid gestation

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SUMMARY. A 34-year-old grand multipara (para 7, 4 alive) was managed at the National Hospital, Abuja, Nigeria for acute renal failure due to HELLP syndrome following referral from a peripheral hospital. She presented with a history of vomiting, headache, epigastric pain, loss of consciousness and tonic/clonic seizures. Though she was unsure of her exact dates, clinically the gestational age was estimated at 22 weeks. She was managed in the intensive care unit, following delivery of a macerated fetus within 15 h of hospital admission. The patient received mechanical ventilation and three sessions of haemodialysis as part of her successful management while in the intensive care unit. The uncommon presentation of eclampsia and HELLP syndrome before obvious preeclampsia is discussed, as well as the other signs and symptoms and patient management. The case also highlights the resource-poor environment of peripheral and tertiary hospitals in Nigeria.

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INTRODUCTION

The syndrome of haemolysis, elevated liver enzymes and low platelets (HELLP) is a life-threatening ailment, often seen as a severe variant of preeclampsia.¹ The disorder is reported to complicate between two and 12% of gestations² and is associated with a high perinatal and maternal morbidity.¹ Since 1982, it has been reported that HELLP syndrome is associated with maternal mortality rates between 1% and 24%,³ with an average mortality rate of 5%.⁴ Hypertension and proteinuria occur in 80% of patients with HELLP syndrome.⁴

Acute renal failure (ARF) is a major complication of HELLP syndrome,^{5–7} but is thought to be rare before the third trimester.⁶ Though our patient was unsure of her exact dates, she appeared clinically to be in the mid-trimester. While an earlier presentation of acute renal failure due to the HELLP syndrome has been reported in Europe,⁶ we feel that this report will contribute to the literature on this topic from our part of the world.

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CASE REPORT

A 34-year-old grand multipara (para 7, four alive) presented at the accident and emergency department of the National Hospital, Abuja, Nigeria, unconscious (the Glasgow coma scale score was about 9/15), jaundiced and pale but afebrile, following referral from a peripheral hospital in Abuja. She presented with a week's history of headache, vomiting and epigastric pain. Following these signs and symptoms, she visited a private hospital in her home town where she was managed as an out-patient. When these symptoms recurred after about five days following the initial management, she visited the referring hospital where she spent a day on admission before presentation at the National Hospital. She had had tonic-clonic convulsions for the first time two hours after admission in the referring hospital followed by a further four convulsions before referral. She was reportedly managed on hydralazine (10 mg i.v. initially given when the blood pressure was 200/110 mmHg) and diazepam before referral. There was nothing in the referral notes to show that she received oxygen or arterial oxygen saturation monitoring in the referring centre.

She had had a caesarean section for pregnancy-induced hypertension complicated by intrauterine death two years before presentation. She was unsure of her

dates, but thought her last menstrual period was about 20–23 weeks previously. There was no record of a previous ultrasound scan to date the index pregnancy.

On presentation at our centre the blood pressure was 180/100 mmHg, the pulse rate 104 beats/min and temperature 36.6 °C. Abdominal examination revealed a gravid uterus corresponding to 22 weeks' gestational age and an old Pfannenstiel incision. The uterus was soft on palpation. The fetal heart was not heard. The cervix was 2 cm long, posterior and uneffaced and the os was closed. A urethral catheter that had apparently drained about 80 mL of dark coloured urine was in place. A naso-gastric tube was also in-situ. A diagnosis of antepartum eclampsia in an unbooked patient with intrauterine death was made, and the patient was admitted to the labour ward for further management. She was given a Ringer's lactate infusion, diazepam, hydralazine (20 mg i.v. if the diastolic blood pressure was >110 mmHg), ampicillin plus cloxacillin, an oro-pharyngeal airway, oral suction as needed, oxygen by facemask and fluid input/output monitoring.

Labour was induced with oxytocin 5 units in 500 mL of 5% dextrose in water. A macerated fetus was delivered within 15 h of admission. Labour pains were not apparent, as the patient, already unconscious, was given diazepam 20 mg i.v. slowly plus 40 mg in 500 mL of 5% dextrose. No analgesia was given, but the blood pressure was closely monitored and she required a single 20-mg i.v. bolus of hydralazine.

Results of laboratory investigations done on admission were as follows: urea 13.8 mmol/L, creatinine 327 mmol/L, aspartate aminotransferase (AST) 1869 units/L, alanine aminotransferase (ALT) 466 units/L, bilirubin 18.0 mg/dL and random blood sugar 3.8 mmol/L. The packed cell volume was 30% on admission and the platelet count 78×10^9 /L. Urinalysis showed bilirubin (++) and glucose (+).

The diagnosis was now changed to eclampsia with HELLP syndrome and acute renal failure. The patient was admitted to the intensive care unit (ICU) for further management after a review by one of us (UVO). There was medical consultation following ICU admission.

On admission to the ICU, she was deeply unconscious (Glasgow coma scale 7/15), she had epistaxis but was breathing spontaneously. The blood pressure was 185/117 mmHg and the pulse rate 118 beats/min. Hydralazine 20 mg was given i.v. to lower the blood pressure. Following i.v. sodium thiopentone 500 mg, to obtund the pressor response, and suxamethonium 100 mg, she was intubated with a cuffed endotracheal tube (internal diameter 8.5 mm) and ventilated with a Newport ventilator. The ventilatory settings were as follows: tidal volume 10–15 mL/kg body weight, respiratory rate 12–16 breaths/min, inspiratory/expiratory ratio 1:2, inspired oxygen 0.6–1.0. Prolonged neuromus-

cular blockade was achieved with intermittent bolus doses of atracurium. The new serum urea/electrolytes/creatinine profile revealed a hyponatraemia and increasing levels of urea (26.4 mmol/L) and creatinine (583 μ mol/L). The patient's serum was negative for antibodies to hepatitis B. The white blood cell count was 11.4×10^9 /L, with neutrophils 76%, lymphocytes 22%, eosinophils 2%, monocytes 0% and basophils 0%. However, the platelet count increased within 48 h after delivery to 104×10^9 /L. Coagulation tests were ordered but apparently were not done. The patient had two violent convulsions in the ICU, which were controlled with i.v. sodium thiopentone 500 mg on each occasion. Magnesium sulphate was unfortunately not available for use in the management of this patient.

Following further medical consultation, ranitidine, frusemide, ciproflaxacin, metronidazole, diazepam, anti-hypertensive agents and i.v. fluids (5% dextrose/saline restricted to 2 L daily), were administered without evident improvement. In view of the patient's worsening condition and persistent oliguria during the three days of ICU admission (87 mL, 133 mL and 225 mL daily respectively), she had three sessions of single-needle dialysis, with four units of blood transfused during the sessions. Single needle dialysis involves using a single catheter lumen to catheterise the femoral artery, while a fistula cannula is used to catheterise a peripheral vein in forearm or upper arm, as in this case.

Subsequently, her condition improved and the urine output increased from 576 mL over 24 h on the day of the third and final session of dialysis to 3920 mL over 24 h six days later. Laboratory investigations showed improvements in AST (56 units/L) and ALT (74 units/L). The patient was extubated after four days of mechanical ventilation, while semi-conscious, and regained full consciousness after eight days in the ICU. The blood pressure was labile, but there was otherwise steady improvement in her general condition with the SpO₂ at 96–100%, initially with oxygen through an intranasal cannula and later on air. There were varying degrees of confusion as she regained full consciousness.

She was transferred to the gynaecological ward after ten days of ICU admission with a blood pressure of 180/80 mmHg and a pulse rate of 104 beats/min. She was discharged after 30 days of hospital admission with a blood pressure of 130/80 mmHg and in satisfactory general condition.

DISCUSSION

HELLP syndrome was described in 1982 by Weinstein,⁸ and is thought to be caused by endothelial cell injury, with subsequent vasospasm, platelet activation, an

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