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CASE REPORT

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Expectant management of severe early-onset preeclampsia in a low-resource setting: A case report

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Abstract

Background: Severe early-onset preeclampsia is associated with increased likelihood of maternal complications and poor perinatal outcomes, regardless of expectant management.
Case presentation: A 17-year-old primigravida at 30 weeks' gestation presented to the obstetric ward with complaints of headaches and painless per vaginal bleeding for one day. Her blood pressure was 174/106 mmHg. She had proteinuria of 3+. The ultrasound revealed a 30-week single intrauterine pregnancy with a partial placenta previa. A diagnosis of severe early-onset preeclampsia was made. She was started on expectant management with antihypertensives, antenatal corticosteroids, and magnesium sulfate with close monitoring. She developed hemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome after seven days and was delivered by cesarean section. Her clinical condition improved markedly after delivery.
Conclusion: Expectant management of severe early-onset preeclampsia can safely prolong pregnancy in select cases, with potential neonatal benefits.

Keywords: early-onset preeclampsia, expectant management, corticosteroids

Introduction

Severe early-onset preeclampsia (EO-PE) complicates about 0.3% of pregnancies globally (1). In Kenya, previous studies have reported an incidence rate of 0.38 percent (2). Early-onset preeclampsia is often associated with complications arising from progressive worsening of the maternal and fetal condition (2,3,4). Consequently, expedited delivery was recommended as per clinical practice guidelines. Subsequent studies have, however, shown that expectant management can safely prolong pregnancy in well-selected patients, allowing for further fetal growth and maturation, resulting in improved neonatal outcomes without compromising maternal safety (1). This is a case of severe EO-PE successfully managed expectantly.

Case presentation

A 17-year-old primigravida presented to the obstetric ward at the Kenyatta national hospital (KNH), as a referral. She gave a history of headaches and painless vaginal bleeding for one day, the first bleeding episode in her pregnancy. The bleeding was was not associated with trauma. She perceived regular fetal activity. At antenatal clinic (ANC) booking, she weighed 52 kilograms, had a hemoglobin of 10.2 g/dL. Her human immunodeficiency virus (HIV) and Venereal Disease Research Laboratory (VDRL) serology were negative, and urinalysis was normal. She was put

on hematinics and had three ANC visits after that, during which she was normotensive. On examination at the referring facility, her blood pressure (BP) was 174/106 mmHg, and a dipstick urinalysis revealed proteinuria of 3+. An obstetric ultrasound scan revealed partial placenta previa with a 30-week pregnancy. She received a STAT dose of nifedipine 20mg orally and a single intramuscular dose of dexamethasone 6mg before referral.

On admission at the KNH, the bleeding had resolved. Her BP was 134/82 mmHg. Cardiac and pulmonary examinations were normal, and there was no tenderness on abdominal palpation. The uterine fundal height corresponded to 32 weeks gestation. There was no edema, and a non-stress test was reassuring. An obstetric scan showed a 30 week, 1 day fetus with a biophysical profile score (BPPS) of 8/8 and an estimated weight of 1 500 grams. The umbilical artery (UA) Doppler indices were normal, and amniotic fluid volume was adequate. Partial placenta previa was noted without abruption. Her creatinine and total bilirubin levels were 90 and 17.1 µmol/L, respectively; alanine transaminase (ALT) and aspartate transaminase (AST) levels were 15 and 35 IU/L, respectively. Her white blood cell (WBC) count was 12.6×10^9/L, hemoglobin (Hb) of 13.2 g/dL, and platelet count 154×10^9/L.

The patient was managed conservatively, aiming for delivery at 34 weeks gestation. The management protocol included oral nifedipine and methyldopa to maintain blood pressure between 140-150 mmHg systolic and 90-100 mmHg diastolic, intravenous labetalol in case of severe hypertension, dexamethasone for fetal lung maturation, magnesium sulfate for neuroprotection, daily monitoring of maternal perception of fetal kicks, alternate-day non-stress tests, weekly obstetric scan, twice-weekly laboratory evaluation and daily monitoring for vaginal bleeding. During the first six days of admission, the patient remained asymptomatic. On day six, an ultrasound scan showed a 31-week fetus with an estimated weight of 1 675 grams, BPPS of 8/8, and amniotic fluid index (AFI) of 11.5cm. The UA Doppler indices were normal, and there was no evidence of placental abruption. Seven days post-admission, the patient complained of dyspnoea. She had a respiratory rate of 20 beats per minute (BPM) and oxygen saturation of 96% on room air. Pulmonary edema was considered. Her BP was 132/87 mmHg, and there were apparent facial puffiness and bilateral lower limb edema on general examination. Cardiac examination was normal, and both lung fields were clear on auscultation. The non-stress test was reassuring.

Laboratory evaluation revealed thrombocytopenia of 38×10^9/L, creatinine of 116 µmol/L, AST and ALT of 45 and 54 IU/L, respectively, and LDH 851 U/L (Figure 1). A diagnosis of hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome was made. She was transfused with one unit of platelet concentrate and a unit of fresh whole blood. The patient was delivered by emergency cesarean section. This was done under spinal anesthesia by a consultant anesthesiologist after conducting a risk versus benefit analysis. Significant ascites was found intraoperatively. A 1440-gram newborn female with 1 and 5 minute APGAR scores of 8 and 9, respectively, was delivered. The placenta was located posteriorly and was delivered without difficulty by controlled cord traction. There was no evidence of placental abruption, and she did not develop postpartum hemorrhage. Her clinical condition improved remarkably post-delivery. On the fourth postoperative day, edema had resolved, and her laboratory parameters were normal. Blood pressure was still elevated, and she was continued with oral nifedipine. The baby developed neonatal jaundice. She was discharged from the nursery unit after three weeks, at a weight of 1800 grams. During the postnatal follow-up, the patient remained asymptomatic. Her BP, measured twice weekly, ranged between 111-132 mmHg systolic and 64-86 mmHg diastolic. Nifedipine was discontinued on the first postnatal visit. She was discharged from postnatal follow-up at 12 weeks postpartum.

Discussion

Severe EO-PE compared to late-onset PE is associated with more frequent and more severe maternal and perinatal complications (2). The associated complications include eclampsia, HELLP syndrome, acute kidney injury, pulmonary edema, placental abruption, fetal growth restriction, and perinatal mortality. The conventional prevailing practice was to deliver women with severe PE without delay, regardless of the gestational age. However, this was associated with high perinatal morbidity and mortality due to prematurity in gestations that were remote from term (4). Maternal multi-organ dysfunction, fetal compromise, and mid-trimester gestation (less than 28 weeks gestation) are widely accepted contraindications to expectant management of severe EO-PE (5). However, another approach for women with severe EO-PE without these features has been advocated. This was informed by observations that some patients' clinical condition stabilized or even improved during initial observation (1,6). The expectant approach aims to improve neonatal outcomes by prolonging pregnancy without compromising maternal safety. Careful in-patient maternal and fetal monitoring is

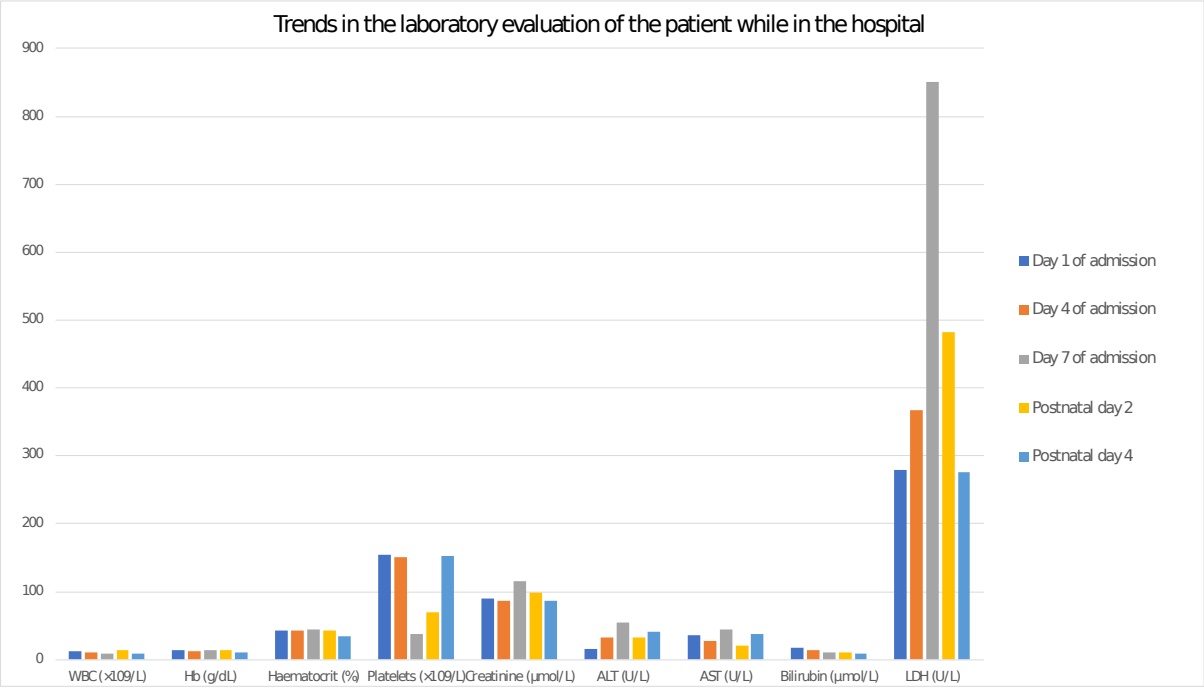

Figure 1: Trends in the laboratory evaluation of the patient

the cornerstone of this approach.

Expectant management of severe EO-PE includes antihypertensives, corticosteroids for fetal lung maturation, magnesium sulfate for neuroprotection, frequent evaluation of maternal vital signs and urine output, serial laboratory evaluation for HELLP syndrome and renal dysfunction, serial sonographic fetal evaluation for for growth and amniotic fluid. Delivery is conducted if any contraindications of expectant management develop or at 34 weeks gestation. Few randomized trials that compared delivery versus expectant management of severe EO-PE reported conflicting results. Odendaal H. et al. and another by Sibai B. et al. reported an association between expectant management with reduced neonatal complications, higher birth weight, less frequent neonatal intensive care unit (NICU) admissions, less frequent respiratory distress syndrome (RDS) and necrotizing enterocolitis (NEC) but more frequent small for gestational age newborns (7,8). However, Virgil de Garcia P. et al. found that perinatal mortality and morbidity were not improved with expectant management. Fetal growth restriction and placental abruption were more frequent in the group managed expectantly (9). The HELLP syndrome was a rare complication in all three studies. The most recent Cochrane systematic review concludes that expectant management of severe EO-PE is associated with decreased neonatal morbidity but recognizes the need for more trials (10). This case exemplifies the maternal risks associated with expectant management of severe EO-PE. However, the good neonatal outcome, early detection, and timely intervention for maternal complications were achieved.

Conclusion

In centers with prerequisite monitoring and specialized management facilities, expectant management can safely prolong pregnancy in select patients with severe early-onset preeclampsia. Therefore, it is possible to manage patients with severe EO-PE in a low-resource setting with adequate monitoring adapting existing evidence on a case-by-case basis.

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Declarations

Conflict of interests

The authors declare no conflicts of interest.

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