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Case report

A complicated case of antepartum eclamptic fit with HELLP syndrome, acute renal failure and multiple intracranial hemorrhages: A mortality report

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KEYWORDS

Cerebral hemorrhage;
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Abstract HELLP is an acronym for hemolysis, elevated liver enzymes and low platelets count, affecting 0.2–12% of all pregnancies or 4–12% of those with preeclampsia. The maternal mortality reported from the literature is up 4% due to disseminated intravascular coagulation, placental abruption, acute renal failure, eclampsia, and cerebral hemorrhage. A 20 year old, G2P1, at 36 weeks of gestation, was referred to our hospital because of postictal coma state with bilateral mydriasis and epistaxis due to repeated antepartum eclamptic fits. Elevated blood pressure level 170/110 mmHg was accompanied with massive proteinuria. Cesarean section was performed and female newborn were delivered. Laboratory findings were characteristic of preeclampsia, HELLP syndrome and renal failure. The patient developed an intraventricular hematoma and an intracerebral hemorrhage with subarachnoid one, which were not suitable to neurosurgical treatment. The patient died from refractory hemolytic anemia, spontaneous bleeding of multiple organs, renal failure and intracranial hemorrhage. Preeclampsia, HELLP syndrome, and acute fatty liver of pregnancy might overlap and be associated with potentially fatal complications, including intracranial hemorrhage, as in the present case. Early detection and diagnosis are crucial to ensure appropriate management and treatment success.

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1. Introduction

Pregnancy induces physiological, hormonal, and physical changes that may cause hypertensive, hematological, and liver complications. Less than 1% of women require admission to intensive care unit (ICU) during pregnancy and the peripartum period, which is associated with maternal and fetal mortality [1].

Preeclampsia, HELLP (hemolysis, elevated liver enzymes, and low-platelet count) syndrome, and acute fatty liver of pregnancy (AFLP) are the main causes of thrombotic microangiopathy [2] and severe liver disease during pregnancy [3].

The present article describes a rare case of acute intracranial hemorrhage, acute renal hemorrhage and DIC occurring

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as fatal complications of severe thrombotic microangiopathy during pregnancy.

2. Case report

A 20 year old female, G2P1, with history of normal vaginal delivery was admitted to our hospital at a gestational age of 36 weeks due to postictal coma state after repeated antepartum eclamptic fits since 4 h before admission and bleeding from mouth and nose. She was irregular in her antenatal checkups and last visit was since months.

Examination revealed a coma state with poor response to reflexes and bilateral mydriasis. The patient's blood pressure was 170/110 mmHg, heart rate nearly 100 beat/min, and proteinuria +++. Bleeding from mouth and nose was noted. Foley's urinary catheter revealed liquorice colored urine. Fundal level was nearly 36 weeks, and cervix closed with no vaginal bleeding. Ultrasound revealed a nearly 36 week living female with normal amount of liquor and slow fetal motion. The patient was transferred to the ICU with endotracheal intubation and mechanical ventilation. Investigations revealed Hb 12 gm%, platelet count around 49,000 per cubic milliliter, so blood, plasma and platelet were transfused.

An emergency cesarean section was done under general anesthesia and nitrate infusion ending in delivery of a single female baby with good APGAR score (Fig. 1). Continuous oozing from sutures characteristic of DIC was noted. Good hemostasis was done with intraperitoneal and subsheath drains inserted.

The patient was immediately transferred to the ICU on mechanical ventilation (Fig. 1). The patient continued nitrate and magnesium sulfate infusion.

The laboratory tests indicated anemia with a hemoglobin concentration of 8 g/dL; fragmented red blood cells; a platelet count of 40,000/mm^3, aspartate transaminase (AST), 200 U/L; alanine transaminase (ALT), 500 U/L; total bilirubin, 5 mg/dL; direct bilirubin, 4 mg/dL; lactic dehydrogenase (LDH) 700 U/L; creatinine, 2.2 mg/dL; urea, 27 mg/dL; normal prothrombin time, significant proteinuria, uric acid, 10 mg/dL, and serum albumin 2 gm%. Serologic tests for hepatitis and the human immunodeficiency virus (HIV) were performed but were negative.

The patient exhibited spontaneous bleeding at other sites, such as the mouth, nose, and vagina (although the coagulation tests had been normal). So nasal packs were inserted (Fig. 1).

Brain CT was ordered and showed massive intracranial hemorrhage intracerebral, intraventricular and subarachnoid hemorrhage with generalized brain edema (Fig. 2).

Notably, the original neurosurgical plan had been to perform a decompressive craniotomy and to treat the acute subdural hematoma; however, the anesthetist had to interrupt the procedure because of progressive and refractory hemodynamic deterioration despite aggressive management.

The patient general condition deteriorated over time with continuing convulsions and no regain in consciousness. The patient exhibited oliguria and necessitated hemodialysis.

The laboratory tests indicated hemoglobin concentration of 5 g/dL; fragmented red blood cells; a platelet count of 30,000/mm^3, aspartate transaminase (AST), 90 U/L; alanine transaminase (ALT), 100 U/L; total bilirubin, 5 mg/dL; and kidney failure (creatinine, 5 mg/dL; urea, 70 mg/dL).

The patient exhibited hemodynamic instability that was difficult to manage despite fluid replacement, blood component transfusion, and the use of vasoactive drugs. The patient died five days after admission.

3. Discussion

Preeclampsia is characterized by hypertension, proteinuria, and edema. It affects approximately 7% of pregnant women, and 65% might progress to HELLP syndrome. For these reasons, preeclampsia remains a main cause of maternal morbidity and mortality. Preeclampsia occurs during the second or third trimester of pregnancy, but occasionally, the disease has been detected before 20 weeks. Its consequences include hypertensive crises, kidney failure, liver rupture, neurological complications such as convulsions and stroke, and increased perinatal morbidity and mortality. The pathogenesis of preeclampsia is believed to be associated with placental ischemia, endothelial dysfunction, cytotoxic and genetic factors. Severe preeclampsia is defined by systolic arterial pressure levels that are persistently $\geqslant$160 mmHg or diastolic arterial pressure $\geqslant$110 mmHg, massive proteinuria (4+ in a dipstick, or $>$2.0 g/24 h), or the presence of clinical (epigastric pain, nausea, and vomiting) and laboratory (platelet count $<$50,000/mm^3, creatine kinase $>$200 U/L, LDH $>$1400 U/L, AST $>$150 U/L, ALT $>$100 U/l, uric acid $>$7.8 mg/dL, and serum creatinine $>$1.2 mg/dL) manifestations [4,5].

The present case exhibited features compatible with HELLP syndrome, and severe preeclampsia. HELLP

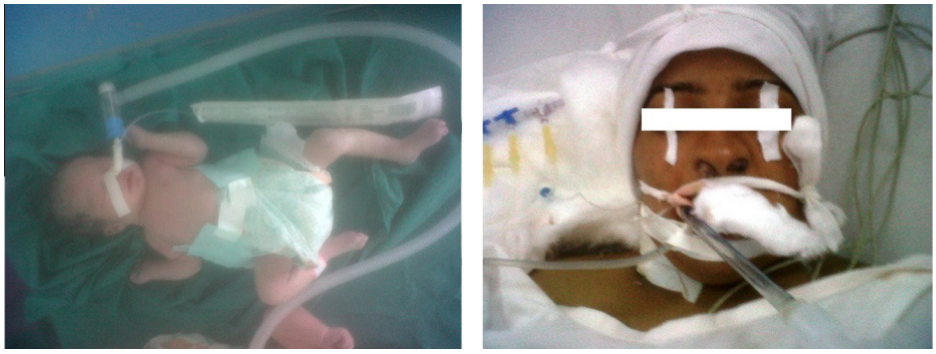

Figure 1 The living baby on the left side and the mother on mechanical ventilation with nasal pack on the right side.

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