Case Report

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Severe EPH Gestosis Accompanied by HELLP Syndrome and Acute Renal Failure

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Key Words

EPH gestosis
HELLP syndrome
Renal failure
Preeclamptic toxemia of pregnancy

Abstract

A case of severe EPH gestosis accompanied by HELLP syndrome and acute renal failure which required vigorous treatment in order to prevent maternal death.

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Introduction

Severe EPH gestosis is diagnosed when one or more of the following symptoms are present: (1) blood pressure of at least 160 mm Hg systolic or 110 mm Hg diastolic on two readings 6 h apart; (2) proteinuria of 5 g/24 h or more; (3) oliguria of less than 400 ml in 24 h; (4) cerebral or visual disturbances; (5) pulmonary edema or cyanosis.

Hematological defects such as hemolysis, thrombocytopenia are well known to accompany severe EPH gestosis [1, 2].

Weinstein [3] suggested that a 6th criterion called the 'HELLP' syndrome, defined as hemolysis, elevated liver function tests and low platelet count, should be added to the 5 formally described criteria. The following report presents a patient with severe EPH gestosis that required aggressive treatment.

Case Report

A 25-year-old woman gravida 0 para 0 was admitted at her 39th gestational week of her first pregnancy, complaining of severe epigastric pains. Her past and family history were noncontributory. On admission, blood pressure was 160/110 mm Hg, the pulse rate was 88/min, 3+ proteinuria and patellar hyperreflexia. On gynecological examination the uterus size was of 35 weeks of gestation, vertex presentation of a single fetus, the cervix was posteriorly positioned 70% effaced and 2 cm dilated. Further examination was without pathological findings. Laboratory tests revealed hemoglobin of 14.1 g/dl; platelet count was 90,000 fibrinogen 440 mg/dl; pro-thrombin time 32 %; PTT 24 s; FSP over 40; glucose, urea and electrolytes were in the normal range; there was no hemoglobin in the urine.

The patient was treated with magnesium sulfate and apresoline (hydrazaline) but, despite this treatment, the blood pressure rose to 180/140 within a few minutes. The patient received diazoxide 300 mg and the blood pressure dropped to 150/100.

Fetal bradycardia of 60 beats/min appeared without remission and the pregnancy was terminated by

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a cesarean section. A normal male infant weighing 2,280 g was delivered with an Apgar score of 9 and 10. The operation was without complication except for a greater blood oozing than usual. Laboratory tests, 2 h following operation, showed Hb of 10.4 g/dl; hematocrit 30; platelets 30,000; fibrinogen 200 mg/dl; prothrombin time 24%; PTT 23 s; FSP over 40. The patient was treated with fresh frozen plasma thrombocytes.

Four hours after the cesarean section the patient became oliguric (less than 20 ml/h) and then anuric despite the administration of diuretics. Blood urea rose to 13 and the liver enzymes were abnormally high.

In order to control the hemodynamic situation, a central venous pressure (CVP) catheter was inserted and revealed a value of less than 1 cm of water. The patient received fluid albumin and electrolytes that raised the CVP to 18 and despite the continuation of diuretic drugs no diuresis was achieved. Hemodialysis was initiated. After 2 days, diuresis began with 182 ml in 24 h and on the 3rd day 330 ml in 24 h. On the 7th day diuresis was normal and all the laboratory tests returned to normal including the blood count, coagulation parameters, and liver enzymes. The patient was dismissed on the 10th day, the blood pressure on discharge was 130/85.

The damage to the kidneys (tubular necrosis) appeared to be caused by hemoglobinuria, or hypovolemia or both and we are quite sure that if a CVP catheter had been inserted earlier we could have avoided the acute renal failure.

Therefore, we would advise insertion of a CVP catheter in every patient with severe preeclamptic toxemia in order to achieve good results.

References

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Discussion

The main problem arising in the above case was acute renal failure. It is well established that severe preeclamptic toxemia can be accompanied by a coagulation defect [3]. The very low CVP (less than 1) was due to relative hypovolemia (in the intravascular space) and the use of diuretics was of no avail.