A Case Report

Hypertensive Chorioretinopathy in Preeclampsia

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ABSTRACT

Background: Hypertensive disorders of pregnancy are one of the most important and controversial unresolved issues in obstetrics. These disorders are one of the leading causes of maternal morbidity and mortality. Clinical manifestations of severe preeclampsia include severe headache, blurred vision, epigastric pain, hypertension and severe edema. The key to treating preeclampsia and vision disorders is controlling blood pressure, preventing seizures and labor induction. We herein report a case of preeclampsia with hypertensive chorioretinopathy.

Case description: A 23-year-old, G2P1L1, 35-week pregnant woman was presented with complaints of headache, dizziness, blurred vision in the left eye, lower extremity edema and less severe generalized edema. The patient was hospitalized with diagnosis of severe preeclampsia after further investigations. Given that the patient was not in the delivery stage, labor induction via cesarean section was carried out. The patient was then hospitalized in the coronary care unit for 24 hours and was later transferred to the gynecology ward. At day four of hospitalization, the patient fully recovered and was discharged.

Conclusion: Headaches and vision problems are common complaints of most women with severe preeclampsia and eclampsia, which require immediate attention of obstetricians and midwives. It is recommended to immediately refer mothers with severe symptoms to a center equipped with intensive care for proper management of the maternal and fetal complications of preeclampsia.

Keywords: Preeclampsia; Hypertension; Chorioretinopathy; Delivery

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INTRODUCTION

Hypertensive disorders of pregnancy are one of the most important and controversial unresolved issues in the field of midwifery that affects 5-10% of all pregnancies. Hypertensive disorders along with bleeding and infection comprise the lethal triad that plays a major role in maternal mortality. Preeclampsia alone or along with hypertension is the most life-threatening hypertensive syndrome of pregnancy (1). According to the World Health Organization reports, 16% of maternal deaths in developed countries are due to hypertensive disorders (2). Importantly, half of these deaths are associated with preventable hypertension (3).

The underlying cause of preeclampsia remains unclear. The imbalance between placental growth factors and anti-angiogenic protein could be a possible contributing factor to occurrence of preeclampsia (4). These changes cause endothelial dysfunction and affect important organs, such as the eye (5). In case of eye involvement, the most common sign is blurred vision (6). There is no specific treatment for ocular manifestations, and treatment primarily involves the treatment of systemic conditions because choriotretoninopathy usually resolves quickly in the postpartum stage. However, this condition can lead to permanent vision loss due to ischemia-induced cerebral infarction (7). In fact, the occurrence of neurological manifestations such as visual impairment, blurred vision and severe headache indicates the worsening of the disease and is an imminent symptom of eclampsia and seizures (8). In this case, generalized cerebral edema may develop, which usually manifests as mental disorders ranging from confusion to coma. This condition is life threatening because it can cause fatal transtentorial herniation (8). Therefore, it is necessary to be cautious when dealing with these patients and consider appropriate treatment measures.

CASE PRESENTATION

A 23-year-old, G2P1L1, 35-week pregnant woman was presented with complaints of headache, dizziness, blurred vision in the left eye, edema of the lower extremities and less severe generalized edema. According to the patient, edema started a day after she received 30,000 units of vitamin D injection due to weakness in the limbs a week ago. The edema was progressive and accompanied with lower limb pain, reduced vision and blackout of vision in the left eye. It should be noted that edema onset following vitamin D injection is only important for determining the time of edema onset and has nothing to do with edema. The patient's blood pressure at the time of admission was 125/85 mmHg. Nonstress test was performed which was satisfactory. Due to headache, sudden edema and suspicion of preeclampsia, an emergency urinalysis test was requested. After consulting with a gyneecologist, the patient was hospitalized due to severe proteinuria (+++), sudden edema and decreased vision with a diagnosis of severe preeclampsia. Blood samples were taken for the following tests: complete blood count, aspartate transaminase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALK-P), lactate dehydrogenase (LDH), blood urea nitrogen (BUN), creatinine, sodium, potassium, total bilirubin (Bill T), direct bilirubin (Bill D), prothrombin time (PT) and partial thromboplastin time (PTT). Because the patient was not in the delivery stage, labor induction via cesarean section was carried out. A preterm infant was born at 34 weeks with Apgar score of 8 at one minute and 10 at five minutes. The amniotic fluid was small in volume but clear.

Before transfer to the operating room, 4g of MgSO₄ was immediately injected intravenously. The patient’s blood pressure was 130/80 mmHg during the operation and 170/90 mmHg in the recovery room. The patient was then transferred to coronary care unit (CCU) after receiving maintenance dose
of MgSO$_4$ (1 g/h). After consultation with a cardiologist, hydralazine (5 mg) was immediately injected and metoprolol, captopril and hydralazine were administered orally to control blood pressure following start of feeding. Table 1 shows the results of laboratory tests at time of admission.

### Table 1. Laboratory findings at time of admission

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>10.9</td>
<td>mg/dl</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>31.7</td>
<td>%</td>
</tr>
<tr>
<td>Platelets count</td>
<td>248</td>
<td>×10$^3$/µL</td>
</tr>
<tr>
<td>Partial thromboplastin time (PTT)</td>
<td>24</td>
<td>Sec</td>
</tr>
<tr>
<td>Prothrombin time (PT)</td>
<td>10.1</td>
<td>Sec</td>
</tr>
<tr>
<td>International normalised ratio (INR)</td>
<td>0.9</td>
<td>--</td>
</tr>
<tr>
<td>Urea</td>
<td>27</td>
<td>mg/dl</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1</td>
<td>mg/dl</td>
</tr>
<tr>
<td>Uric acid</td>
<td>5.3</td>
<td>mg/dl</td>
</tr>
<tr>
<td>Total bilirubin (Bill T)</td>
<td>0.9</td>
<td>mg/dl</td>
</tr>
<tr>
<td>Direct bilirubin (Bill D)</td>
<td>0.3</td>
<td>mg/dl</td>
</tr>
<tr>
<td>Aspartate transaminase (AST)</td>
<td>25</td>
<td>U/L</td>
</tr>
<tr>
<td>Alanine aminotransferase (ALT)</td>
<td>13</td>
<td>U/L</td>
</tr>
<tr>
<td>Lactate dehydrogenase (LDH)</td>
<td>799</td>
<td>U/L</td>
</tr>
<tr>
<td>Alkaline phosphatase (ALK-P)</td>
<td>483</td>
<td>U/L</td>
</tr>
</tbody>
</table>

Treatment with the maintenance dose of MgSO$_4$ continued until 24 hours after the delivery. Moreover, due to decreased vision and neurological symptoms (headache, dizziness and blurred vision), neurological consultation was requested, which was followed by ophthalmology consultation. It was recommended that the patient be referred to a referral hospital for further examination, which was temporarily postponed by the gynecologist due to the patient’s general condition.

During hospitalization in the CCU, the volume of urine was less than 50 cc/hour; therefore, the level of BUN, creatinine, sodium and potassium were checked, which were 33 mg/dl, 1.1 mg/dl, 139 mEq/l and 3.9 mEq/l, respectively. Next, a 20 mg furosemide ampoule and a half a liter 5% dextrose solution were administered, which resolved oliguria and improved urine volume. On the second day of hospitalization, the patient weighed 77 kg and dizziness and headache were improved, but she still complained of decreased vision in the left eye. Eventually, the patient was transferred to the gynecology ward with a blood pressure of 130/80 mmHg. In the morning after discontinuation of MgSO$_4$, BUN and creatinine levels were 30 mg/dl and 0.98 mg/dl, respectively (Table 2).

On the third day of hospitalization, blurred vision improved and blood pressure was 120/87 mmHg. Captopril and metoprolol were discontinued, but hydralazine therapy continued. Finally, after four days, the patient was discharged with prescription of 25 mg/day hydralazine. In the postpartum follow up (ten days after the discharge), the patient weighed 58 kg (19 kg weight loss compared to the second postpartum day) and there was no evidence of blurred vision, headache or dizziness. Edema was resolved completely and blood pressure was normal (115/70 mmHg).
Table 2. Changes in maternal serum creatinine level of the mother

<table>
<thead>
<tr>
<th>Test time</th>
<th>Serum creatinine level</th>
</tr>
</thead>
<tbody>
<tr>
<td>At admission</td>
<td>1 mg/dl</td>
</tr>
<tr>
<td>Hospitalization day 1</td>
<td>1.1 mg/dl</td>
</tr>
<tr>
<td>Hospitalization day 2</td>
<td>0.98 mg/dl</td>
</tr>
</tbody>
</table>

DISCUSSION

Preeclampsia complicates 2-8% of all pregnancies. Preeclampsia and eclampsia increase the risk of maternal morbidity and mortality as well as pregnancy outcomes such as preterm delivery, fetal growth restriction, placental abruption and perinatal mortality (9). In our case, the severity of preeclampsia led to premature delivery. Preeclampsia is more prevalent in nulliparous women and multiparous women aged 35 or older (10). However, our case was a 23-year-old, G2P1 woman with no history of background disease including cardiovascular disease, diabetes, hypertension and metabolic syndrome. She also had no history of gestational hypertension and preeclampsia in the previous pregnancy. Laboratory abnormalities of preeclampsia include proteinuria, elevated serum creatinine, thrombocytopenia and elevated liver enzymes (8), which were all present in our case. Clinical manifestations of severe preeclampsia include severe headache, blurred vision, epigastric pain, hypertension and severe edema (11). Our case also had blurred vision, vision impairment, sudden and severe edema and high blood pressure. Preeclampsia have various neurological manifestations, which require immediate attention. Headache and scotoma occur because of cerebrovascular hyperfusion and do not respond to conventional analgesics. As expected, postpartum headache was resolved after MgSO₄ administration. The other neurological manifestations are seizures caused by excessive release of excitatory neurotransmitters and blindness, which is rare in preeclampsia but often occurs in association with eclamptic seizures (8). Eventually, generalized cerebral edema may develop, usually manifesting as consciousness changes, ranging from confusion to coma. In fact, blurred vision, scotoma and diplopia are common in severe preeclampsia and eclampsia. These symptoms usually resolve with MgSO₄ therapy or lowering blood pressure. Blindness is less common and usually reversible (12). In our case, blurred vision and visual impairment resolved completely after MgSO₄ therapy. The key to treating preeclampsia and vision disorders is controlling blood pressure, preventing seizures and labor induction. The preferred antihypertensive drugs are hydralazine, labetalol and nifedipine. The preferred drug for the prevention of seizures is MgSO₄, which reduces vascular spasm (13). In our case, the medications prescribed to control blood pressure in the pregnant mother were 5 mg intravenous hydralazine, followed by oral hydralazine, metoprolol and captopril. In fact, good medical management of hypertension in this patient prevented occurrence of more severe complications and eclampsia.

CONCLUSION

Headaches and vision problems are common complains of most women with severe preeclampsia and eclampsia, which require immediate attention of obstetricians and midwives. In severe cases, cesarean section is recommended when normal delivery is not possible. Therefore, it is recommended to immediately refer mothers with severe symptoms to a center equipped with intensive care for proper management of the maternal and fetal complications of preeclampsia.
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REFERENCES


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