Successful Outcome of HELLP Syndrome in a 23-Year-Old Pregnant Woman: A Case Report

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Abstract

Introduction: HELLP syndrome is a life-threatening complication of preeclampsia. We report a young pregnant woman with HELLP syndrome who was diagnosed, managed, and delivered in a timely manner.

Case Presentation: A 23-year-old second gravida twin pregnant woman was referred to our clinic due to high blood pressure. After delivery, she experienced a hemolytic condition with elevated liver enzymes and thrombocytopenia, defined as HELLP syndrome. After confirmation of HELLP syndrome by laboratory tests, the patient underwent hemodialysis and plasmapheresis. 10 days later, she was discharged under good general condition.

Conclusions: Women with a history of HELLP syndrome are considered to have an increased risk of death. Therefore, this life-threatening condition should be closely monitored and treated in a timely manner.

Keywords: HELLP Syndrome, Pregnancy, Preeclampsia

1. Introduction

HELPP syndrome is an acronym for hemolysis, elevated liver enzymes, and low platelets' count. The condition is an uncommon complication of preeclamptic toxemia of pregnancy. Its occurrence is rare, around 0.5% - 0.9% of all pregnancies (1, 2).

About 70% of such cases develop before delivery, the majority between 27th and 37th gestational weeks, and the rest within 48 hours after delivery (3).

Herein, we report a young pregnant woman with HELLP syndrome who was diagnosed, managed, and delivered in a timely manner.

2. Case Presentation

A 23-year-old twin pregnant woman, second gravid, was referred to the Department of Obstetrics and Gynecology due to increased blood pressure (BP = 170/110 mmHg) and the possible need for delivery. On admission, she was in a stable condition and as for vital signs, the heart rate had a normal sinus rhythm along with tachycardia (102 beats per minute) and the respiratory rate was 25 breath/min and she was afebrile. Her O2 saturation at rest and under room conditions with a pulse oximeter placed on her index finger was 95% - 96%.

In primary physical examination, we found evidence of 2+ edema in her lower limbs. Obstetric ultrasound study showed that the bi-parietal diameter [BPD] was 37 weeks and 4 days whereas according to femur length [FL], she was 36 weeks pregnant; the amniotic fluid index [AFI] was normal. Moreover, a uterus with twin live fetuses in longitudinal lie in cephalic/cephalic presentation was also revealed. Placenta location was in the fundal position with anterior extension. No evidence of fetal growth restriction was observed and cervical dilatation was 60% (6 cm) at initial examination.

Laboratory findings showed that hemoglobin (Hgb = 15.7 g/dL) and hematocrit (Hct = 40%) levels were in the normal range. Other laboratory tests including liver function tests revealed an aspartate amino transferase (AST) and the alanine amino transferase (ALT) of 350 IU/L and 200 IU/L, respectively. Coagulation tests such as Prothrombin Time (13 seconds), Partial Prothrombin Time (18 seconds), and INR (1) were in the normal range. Creatinine level was 0.5 mg/dL.

Magnesium sulfate therapy was immediately initiated according to the standard protocol; it was combined with
The patient underwent normal vaginal delivery in which one of the twins was born with a normal Apgar score. However, due to frequent failure in fetal heart rate (FHR) and a compound presentation, cesarean section under general anesthesia was performed for the other fetus. With respect to an atonic uterus and excessive bleeding, B-Lynch suture and uterus massage was performed. Misoprostol and Methylene blue were not prescribed because the liver function tests were elevated and the platelet count was critically low. This action continued until the consistency of the uterus was achieved and after a few minutes, bleeding was controlled.

Following cesarean section, some laboratory tests increased in nature including liver function tests (AST = 4332 IU/L and ALT = 914 IU/L) and Lactate dehydrogenase, which was 16684 unit per liter in our patient. Nevertheless, the platelet count severely dropped to 32,000 and hematocrit decreased to 21%. Loss of consciousness was diagnosed and her urinary output revealed oliguria. All laboratory findings are presented in Table 1.

Regarding the above-mentioned findings, two units of Fresh Frozen Plasma (FFP) and packed cells were transfused to the patient and after 2 days, plasmapheresis was initiated.

Based on nephrology consultation, hemodialysis was performed and ADAMTS13 (a disintegrin and metalloproteinase with thrombospondin type 1 motif, member 13) was requested. In parallel, according to neurologic consultation, CT scan and MRI were requested. The CT scan showed signs of ischemia in the occipital area, but MRI findings were normal and there was no need for further studies in this respect.

After 10 consecutive days of hemodialysis and plasmapheresis, the level of laboratory findings went into remission and finally due to a normal ADAMTS13, plasmapheresis was interrupted.

Before discharge, the patient was consulted regarding the need for further follow-up and blood pressure screening, especially in case of another pregnancy. The patient was then discharged under good general condition.

3. Discussion

HELLP syndrome is a life-threatening complication of preeclampsia. The severity of HELLP syndrome is measured and divided into three categories according to the blood platelets’ count of the mother (4). According to this classification, our patient was in the 1st class of HELLP syndrome which describes the severe condition of this disease. Mississippi classification of HELLP syndrome is displayed in Table 2 (5).

Hemolysis was early recognized in the urine and as mentioned in earlier observations, the increase in the AST and ALT levels preceded the decrease in platelets’ count (1). In our patient based on laboratory findings, the pathogenesis of this condition was confirmed as microangiopathic hemolytic anemia. Elevation of liver enzymes reflects the hemolytic process as well as liver involvement. Hemolysis contributes substantially to the elevated level of LDH, whereas enhanced AST and ALT levels are mostly due to liver injury (6, 7).

Thrombocytopenia (platelets < 150,000/µL) in pregnancy may be caused by gestational thrombocytopenia (59%), immune thrombocytopenic purpura (ITP) (11%), preeclampsia (10%), and the HELLP syndrome (12%) (6). Decreased platelet count in HELLP syndrome is due to their increased consumption. Platelets are activated and adhered to damaged vascular endothelial cells, resulting in increased platelet turnover with a shorter lifespan (8-11).

The main aspect of our case was the immediate management of the patient with hemodialysis and plasmapheresis, resulting in rapid return of the laboratory tests to their normal range. This approach can lead to better outcomes. Cho et al. reported a similar case resulting in successful management of HELLP syndrome in a pregnant woman, treated by the same protocol (12).

Life-threatening neurological complications of HELLP syndrome are rare, but incorporate large cerebral or brain stem hemorrhage, thrombosis, and infarctions or cerebral edema complicated by brain herniation (13, 14). There are several case reports on cerebral bleeding associated with HELLP syndrome (15-47). In another report (18) on the outcome of pregnancies complicated by HELLP syndrome, cerebral bleeding was not mentioned as a complication. In contrast, Osmanagaoglu et al. showed that in a highly selected group of 37 women with HELLP syndrome transferred to an obstetric intensive care unit in Turkey, 15 women (40%) had cerebral haemorrhage (19). In our patient, CT scan and MRI studies were also performed after caesarean section, which revealed normal results.

In conclusion, there is a lack of screening test(s) for predicting HELLP syndrome before its occurrence while the current tests only confirm its diagnosis. On the other hand, women with a history of HELLP syndrome are considered to have an increased risk of complications in future pregnancies, highlighting the need for standard antenatal follow-up protocols.

Acknowledgments

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Table 1. Main Laboratory Findings of Our Patient Confirming the HELLP Syndrome

<table>
<thead>
<tr>
<th>Laboratory Indices</th>
<th>Trend in Primary 48 Hours</th>
<th>Trend in Continue</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST</td>
<td>10904</td>
<td>1182</td>
</tr>
<tr>
<td>ALT</td>
<td>654</td>
<td>134</td>
</tr>
<tr>
<td>Creatinin</td>
<td>11</td>
<td>15</td>
</tr>
<tr>
<td>LDH</td>
<td>9843</td>
<td>9715</td>
</tr>
<tr>
<td>PLT</td>
<td>42</td>
<td>71</td>
</tr>
</tbody>
</table>

Table 2. Mississippi Classification of HELLP Syndrome

<table>
<thead>
<tr>
<th></th>
<th>Class 1</th>
<th>Class 2</th>
<th>Class 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelets, x 10^9/L</td>
<td>≤ 50,000</td>
<td>50,000-100,000</td>
<td>100,000-150,000</td>
</tr>
<tr>
<td>AST/ALT IU/L</td>
<td>≥ 70</td>
<td>≥ 70</td>
<td>≥ 40</td>
</tr>
<tr>
<td>LDH/ALT IU/L</td>
<td>≥ 600</td>
<td>≥ 600</td>
<td>≥ 600</td>
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Footnotes

Authors’ Contribution: All authors contributed to developing the concept and revising the paper. They all confirmed the final draft for submission and they accept any responsibility regarding the content of the paper.

Competing Interests: The authors declare that they have no competing interests.

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Consent: A written informed consent was obtained from the patient for publication of this case report.

References


