A case report of HELLP syndrome

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Abstract

HELP syndrome is a serious complication in pregnancy characterized by hemolysis, elevated liver enzymes and low platelet count. This case demonstrated the importance of rapid and early diagnosis and treatment of HELLP syndrome to reduce maternal and perinatal mortality and morbidity. 26 years old, 2nd gravida with 31 weeks of gestation with severe pre-eclampsia was admitted to Department of Obstetrics and Gynecology at Civil Hospital, Ahmedabad. Patient suddenly developed epigastric pain, blood tinge urine (not frank hematuria) and decrease urine output within 24 hours of admission. Investigations revealed platelet count 44,300, serum bilirubin 12, direct bilirubin 3.44, and indirect bilirubin 8.56, SGPT 193.5 and was diagnosed as a HELLP syndrome class 1. She underwent cesarean section and there was dramatic improvement of her symptoms and all blood investigations (S. bilirubin, Platelet count) were declined to normal limit within 48 hours post operatively. HELLP syndrome, a variant of severe pre-eclampsia, if diagnosed and manage timely ensure favorable maternal and perinatal outcome.

Key words

HELP syndrome, Pre-eclampsia, Hemolysis, Diagnosis, Treatment.

Introduction

HELP Syndrome (H – Haemolysis, EL - Elevated liver enzymes, LP - Low platelet count) is a serious complication of severe pre-eclampsia. Its incidence is reported as 0.5-0.9% of all pregnancies, and 10-20% of women with severe pre-eclampsia. Incidence of maternal and perinatal mortality and morbidity is very high in this case. Timely diagnosis and management of HELLP syndrome reduces the maternal and perinatal mortality and morbidity [1].

Case report

26 years old, 2nd gravida female with 31 weeks of gestation presented to us with chief
HELLP syndrome

complaints of bilateral pedal edema since one month. She had a history of severe pre-eclampsia in past pregnancy and underwent lower section cesarean section (LSCS) for transverse lie before three years. Her blood pressure was 150/100 mmHg and had bilateral pedal edema. Rest of the findings of general examination was normal. Random urine albumin was +2 by dip stick method. Obstetrical examination found abdominal wall edema, previous cesarean section scar, 28-30 weeks size uterus, cephalic presentation, regular fetal heart sound (FHS) and relaxed, per vaginal examination showed os closed. On admission, hemogram (Hemoglobin 9 gm/dl, Platelet count 3,68,000 cells/cumm), liver function test (serum bilirubin 8.56, INR 1.01) including liver enzymes and prothrombin time were within normal limit. Ultrasound for fetal well being showed 31 weeks mature intrauterine fetus with early diastolic notch in right uterine artery on Doppler study.

Patient was kept on antihypertensive (T. Methyl dopa and T. Nifedipin) and 2 doses of steroids given for fetal lung maturity. Next day, she suddenly developed epigastric pain, blood tinge urine (not frank hematuria) and decrease urine output. At that time, blood pressure was 160/100 mmHg and urine albumin was +2. In a view of HELLP syndrome, repeat investigations were sent which revealed hemoglobin 11.2 gm/dl, platelet count 64,300 cells/cumm, bilirubin 12 mg/dl, direct bilirubin 3.44, indirect bilirubin 8.56, INR 1.01, SGPT 193.5. At that time, blood components were given (1 pint PCV, 8 pint FFP, 12 pint Platelets, 4 pint Cryoprecipitates). Non stress test was done which was equivocal. Decision of emergency LSCS was taken. After counseling, the patient was posted for surgery. Emergency LSCS was done, delivered a male child 1.5 kg. Per operative blood components were given. Post operative period was uneventful. Her investigations revealed hemoglobin 7 gm/dl, platelet 1,1700 cells/cumm, bilirubin 3.72 mg/dl, direct 1.42 and indirect 2.35, SGPT 85.6, INR 0.98. Patient was given total 5 pint PCV, 21 pint Platelets, 15 pint Cryoprecipitates, and 8 pint FFP.

Discussion

HELLP syndrome is a life-threatening pregnancy complication of pre-eclampsia. The severity of HELLP syndrome is measured according to the blood platelet count of the mother and divided into three categories.

Mississippi classification of HELLP syndrome [2]

<table>
<thead>
<tr>
<th>Class 1 (Severe)</th>
<th>Class 2 (Moderate)</th>
<th>Class 3 (Mild)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelets ≤50,000/µL</td>
<td>50,000-100,000/µL</td>
<td>100,000-150,000/µL</td>
</tr>
<tr>
<td>AST or ALT ≥70 IU/L</td>
<td>≥70 IU/L</td>
<td>≥40 IU/L</td>
</tr>
<tr>
<td>LDH ≥600 IU/L</td>
<td>≥600 IU/L</td>
<td>≥600 IU/L</td>
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</tbody>
</table>

The pathogenesis of HELLP syndrome is not completely understood. Those currently considered important includes

- Placental implantation with abnormal trophoblastic invasion of uterine vessels.
- Immunological maladaptive tolerance between maternal, paternal (placental) and fetal tissues.
- Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy.
- Genetic factors including inherited predisposing genes as well as epigenetic influences.

Hemolysis, one of the major characteristics of the disorder, is due to a microangiopathic hemolytic anaemia. Elevation of liver enzymes
HELLP syndrome reflects the hemolytic process as well as liver involvement. Hemolysis contributes substantially to the elevated levels of LDH, whereas enhanced aspartate aminotransferase (AST) and alanine aminotransferase (ALAT) levels are mostly due to liver injury. Decreased platelet count in the HELLP syndrome is due to their increased consumption. Platelets are activated, and adhere to damaged vascular endothelial cells, resulting in increased platelet turnover with shorter lifespan [3, 4, 5].

HELLP syndrome typically occurs between 27 weeks of gestation and delivery or immediately postpartum in 15%-30% of cases [6, 7, 8, 9]. HELLP syndrome has been shown to occur in older maternal age groups, with a mean age of 25 years. In contrast, pre-eclampsia is most common in younger patients (mean age, 19 years) [9]. The recurrence rate is 2-27% in subsequent pregnancies [10, 11]. Maternal mortality ranges from 1-3%, with a perinatal mortality rate of 35% [12].

Maternal morbidity includes
- Disseminated intravascular coagulation (DIC) (20%)
- Placental abruption (16%)
- Acute renal failure (7%)
- Pulmonary edema (6%) [12]

Conclusion

Definitive treatment for women with HELLP syndrome is delivery of the baby. Transfusion of some form of blood product (red cells, platelets, plasma) is often needed. Corticosteroids can be used to improve fetal lung maturation in the very preterm pregnancy. Timely diagnosis and management of HELLP syndrome either by induction and delivery by vaginal route or by cesarean section is beneficial and prevents complications in mother and fetus.

References


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