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POSTPARTUM HELLP SYNDROME- A CASE SERIES

Obstetrics & Gynaecology

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ABSTRACT

HELLP commonly results as a complication of preeclampsia, recognized by an acronym HELLP refers to a syndrome characterized by haemolysis, elevated liver enzymes, and a low platelet count. It occurs in 0.5%–0.9% of all pregnancies. Women with postpartum HELLP syndrome have significantly higher incidences of complications like pulmonary oedema, renal failure, disseminated intravascular coagulation, and subcapsular liver haematoma. Even the maternal mortality and morbidity is found to be more in postpartum HELLP when compared to antepartum HELLP. So is the much need of early diagnosis and management. This case series includes 3 cases who were managed in our hospital.

KEYWORDS

jaundice, HELLP, low platelet, postpartum HELLP, AKI

INTRODUCTION

HELLP commonly results as a complication of preeclampsia, recognized by an acronym HELLP refers to a syndrome characterized by haemolysis, elevated liver enzymes, and a low platelet count. Haemolysis in these cases results from cell passage through small vessels partially obliterated with fibrin deposits (microangiopathic haemolytic anaemia)[1]. Common complications of the disease are disseminated intravascular coagulation (DIC), placental abruption, pulmonary oedema, subcapsular liver hematoma, retinal detachment, laparotomy due to severe intra-abdominal bleeding and maternal death. Other complications include acute respiratory distress syndrome, shock, sepsis, kidney failure and incision site bleeding in patients with thrombocytopenia[2]. About 70% of the cases happen before delivery and the remainder within 48 hours after delivery. Although there are no differences in laboratory findings between HELLP syndrome before and after delivery, women with postpartum HELLP syndrome have significantly higher incidences of complications and postpartum HELLP is associated with more maternal mortality and morbidity than antepartum HELLP[3]. Absence of symptoms should not rule out this syndrome, and it is recommended to consider risk of postpartum HELLP syndrome during follow ups[2]. Postpartum HELLP presents with atypical presentation, associated with complications and hence timely diagnosis and appropriate management is utmost important.

Case Series

There are 3 cases in this case series, who were all admitted, delivered in our hospital and were diagnosed as HELLP syndrome during the postpartum period. These patients were admitted over a period of 3 years.

Case-1

A 24-year-old primigravida with 9 months ANC with complaints of pain in abdomen for 6 hours admitted in our hospital in view of labour pain. She was referred to our hospital in view of severe oligohydramnios (AFI-1cm). She had no complaints of oedema, epigastric pain, blurring of vision, headache, nausea, vomiting. She was not a known case of hypertension and other major surgical or medical illness. On examination, BP- 110/78 mmhg, PR-98bpm, Uterus 36 weeks, longitudinal lie, Foetal heart sounds- 150bpm, regular, contractions+. Per vaginum examination- os closed, uneffaced. Her investigations were within normal limits. Her CTG was suspicious, so she underwent emergency caesarean section in view of severe oligohydramnios with unfavourable cervix. Caesarean section was uneventful. After 24 hours, patient suddenly developed uneasiness, altered sensorium, epigastric pain, on BP-130/84mmhg, PR-102bpm, pallor+, icterus+, urine albumin was nil, normal vision and normal deep tendon reflexes. She was shifted to ICU, investigated for jaundice as her total bilirubin came out 4.1mg/dl. Her investigations revealed haemoglobin 8.9g%, platelets-52,000/cmm, on PS there were anisopoikilocytosis, schistocytes, LDH was 780U/L, AST-120IU and ALT-144IU, coagulation profile and renal function testes were within normal limits, negative for all hepatitis viral markers. Her USG abdomen showed normal. She was diagnosed as HELLP syndrome though she was normotensive. Her LFTs worsened on day 2 in ICU, she was on supportive therapy, monitored in ICU, she improved clinically. She was transfused with 2 PRBC as her haemoglobin was 7.2g/dl on the next day. Platelets started to rise on day 4 of admission and even Liver function tests improved. On day 8, all her investigations were within normal limits and she was discharged**.**

Case-2

A 27-year-old G3P1L1A1 with 8 months ANC admitted with complaints of headache and vomiting. She had complaints of headache, nausea, vomiting (2 episodes) since that morning. She was married for 7 years; first pregnancy was uneventful and delivered by LSCS 3 years ago for indication- impending eclampsia. In present pregnancy, she was diagnosed with chronic hypertension at 3rd month of ANC and was on tab labetalol 100mg three times daily since then. One month ago, she was also started on tab nicardia 20mg twice daily along with labetalol as her BP She was also a case of pre-eclampsia during her first pregnancy. Pulse- 90/min, BP- 150/90mmhg and systemic examination detected no abnormality. Uterus-32 weeks, longitudinal lie, FHR- 146bpm, regular, previous LSCS scar present, no scar tenderness, no contractions felt. Per vaginum examination- Os posteriorly placed, closed, uneffaced. Her vision was normal, deep tendon reflexes were normal with urine albumin ++. Provisional diagnosis was made as G3P1L1A1 at 31 weeks with previous 1 caesarean section with chronic hypertension with superimposed preeclampsia not in labour. Investigations were within limits. As her BP was not under control and continued warning signs, she was taken for emergency caesarean section in view of previous LSCS with impending eclampsia. The surgery was uneventful but patient developed anuria on postoperative day 2. She was shifted to ICU for further management and she received 4 platelets, 4 FFP. She developed icterus too and her biochem reports were haemoglobin-8.9g/dl, platelet-24,000/cmm, blood urea-98mg/dl, s. creat-3.6mg/dl, total bilirubin-5.2mg/dl, direct bilirubin-3mg/dl, indirect bilirubin-2.2mg/dl, SGOT-120IU/dl, SGPT-98IU/dl, ALP-122IU/dl, LDH-747IU/L, with normal coagulation profile. The serum creatinine levels kept increasing and her haemoglobin fell to 6.1g/dl on postop day-3 for which blood transfusion was done. Arterial blood gas analysis suggested of metabolic acidosis with compensatory respiratory alkalosis with USG suggested of bilateral grade 2 renal parenchymal disease. She had to undergo haemodialysis in view of anuria with creatinine 4.8mg/dl with electrolyte imbalance. Platelets and liver function tests improved by day 6 but renal function kept worsening. She underwent 9 cycles of haemodialysis. On postoperative day 26 she went home with good health.

Case-3

A 28-year-old G3P2L2 with 9 months of ANC brought with complaints of headache. She had no complaints of pain in abdomen,

epigastric pain, blurring of vision, no history of convulsion. She was diagnosed with hypertension at 7th month and was om treatment. Pulse- 98/min, BP-144/98 mmhg, height of uterus 36 weeks, longitudinal lie, Foetal heart sounds- 144bpm, no contractions, multiparous os, uneffaced. Her vision was normal, deep tendon reflexes were normal, urine albumin 2+. All Investigations were within normal limits. Patient was induced with PGE2 gel for preeclampsia at term and delivered vaginally. After 36 hours, patient suddenly developed epigastric pain, breathlessness, icterus, altered sensorium with blood pressure 160/110 and urine albumin ++. Hence, she was shifted to ICU, started on injection magnesium sulphate. Injection furosemide was given as there were crepts on auscultation and chest X ray suggested of pulmonary oedema. Her investigations revealed (table no.1) hb-8.8g%, platelet-72,000/cmm with bilirubin- 3.4mg/dl, and raised AST/ALT, LDH was 670U/L, coagulation profile and renal function tests were within normal limits. She was diagnosed and treated as postpartum HELLP syndrome. Her antenatal investigations were within normal limits. Her platelets kept falling in ICU and investigations got worsen on following day. Ultrasound study of abdomen revealed increased echogenicity in the right lobe of the liver and no other abnormality detected. She was managed conservatively, needed 2 PRBC transfusion as her haemoglobin fell to 6.9g/dl. Symptoms improved after 24 hours in ICU with supportive therapy and eventually biochemistry reports showed improvement and on day 9, all her investigations were within normal limits.

Table no.1 – clinical presentation and management

	Case-1	Case-2	Case-3
Known case of hypertension	No, normotensive	Chronic HTN with superimposed preeclampsia	Severe pre-eclampsia
Mode of delivery	LSCS	LSCS	Vaginal delivery
Time of diagnosis (After delivery)	24 hours	48 hours	36 hours
Presenting complaints	Altered sensorium, epigastric pain	Anuria	Breathlessn ess
Associated complication	-	ARF, underwent 9 cycles of dialysis	Pulmonary oedema
ICU stay duration	3 days	12 days	4 days

DISCUSSION

Though there is much understanding about the pathophysiology and treatment of HELLP, not much literature is available regrading postpartum HELLP syndrome. HELLP syndrome, mostly occurs in the presence of Pre-eclampsia. However, approximately 20% of HELLP syndrome cases can occur without Pre-eclampsia.

Clinical Presentation-

The usual presentation of HELLP is epigastric pain, nausea, vomiting, still all these are non-specific complaints. The diagnosis especially becomes difficult HELLP syndrome in normotensive pregnancies. Presence of severe epigastric pain, low platelet counts with high ALT and AST levels arouse suspicion for HELLP syndrome4. Unlike antepartum HELLP, postpartum HELLP will not have typical presentation thus delaying the diagnosis and treatment. Like in our cases (table no.1) case-1 presented with uneasiness, epigastric pain, altered sensorium, case-2 presented with anuria and case-3 presented with breathlessness. Despite many authors having shown that HELLP syndrome is a complication of preeclampsia or eclampsia, Martin et al study5 observed that hypertension and proteinuria may be absent or its slight even if it is present. Likewise in our case series, 2 out of 3 cases had pre-eclampsia whereas case-1 had normal blood pressure throughout. 30% of the HELLP syndrome occurs in postpartum period especially within 48 hours. Our cases presented after 24 hours, 48 hours and 36 hours however case reported by Cakmak et al6, presented after 60 hours. Another case report by Maryam et al2 also presented within few hours after caesarean section. There are few case reports who had presented with subcapsular haematoma and intraparenchymal liver haematoma.7 which was managed conservatively.

Diagnosis-

There are no differences in the diagnostic criteria of HELLP syndrome during antepartum and postpartum period. However, the severity is more in postpartum HELLP syndrome. women with postpartum HELLP syndrome have significantly higher incidences of complications, such as pulmonary edema, renal failure, disseminated intravascular coagulation, and subcapsular liver haematoma3. Diagnosis is done by lab investigations. There are currently two classification system diagnostic criteria for HELLP syndrome: The Tennessee classification system (platelets < 100× 10 9 , AST > 70 IU, LDH > 600 IU/l or other signs of haemolysis like abnormal peripheral smear finding, raised serum bilirubin >1.2g/dl, low serum haptoglobin) and the Mississippi classification system (Class I: platelets < 50× 10 9 , AST > 70 IU, LDH > 700 IU/l; Class II: 50×10 9 < platelets < 100× 10 9 , AST or ALT > 70 IU, LDH > 600 IU/l; Class III: 100×10 9 < platelets < 150× 10 9 , AST or ALT > 40 IU, LDH > 600 IU/l)2. Tennesse also classifies HELLP into complete HELLP syndrome if there is presence of all the three parameters abnormal (AST/ALT, LDH, Platelets) and incomplete syndrome if there are one or two of the three as abnormal. In our case series, all of them were diagnosed according Tennesse criteria, all of them had complete HELLP syndrome (table no.1). Diagnosis of postpartum HELLP is difficult and delayed as they have atypical, because of absence of classical signs of preeclampsia, it can confuse physicians and lead to delay in diagnosis. Hypertension is absent in 20% of patients with HELLP syndrome, and 5-15% of pregnant patients have mild or no proteinuria. Early detection of haemolysis is more sensitive than determination of serum haptoglobin. Increase in AST and ALT often occurs before platelet count falls8. The differential diagnosis to be considered during diagnosis are acute fatty liver, thrombotic thrombocytopenic purpura, haemolytic uraemic syndrome. Imaging of abdomen (ultrasound) is to be done to look for hepatic subcapsular haematoma or intraparenchymal liver haematoma.

Management-

HELLP syndrome is associated with serious maternal morbidity, especially when it arises in the postpartum period3. In postpartum HELLP most of the cases reported in literature needed conservative management only. Common complications of the disease are disseminated intravascular coagulation (DIC), placental abruption, pulmonary oedema, subcapsular liver hematoma, retinal detachment, laparotomy due to severe intra-abdominal bleeding and maternal death. Other complications include acute respiratory distress syndrome, shock, sepsis, kidney failure and incision site bleeding in patients with thrombocytopenia2. As most of them have associated complications, ICU management and supportive therapy is the mainstay. Supportive treatment includes antihypertensives, control of blood pressure, magnesium sulphate, liver protecting agents accordingly. Steroids has no role in treatment of thrombocytopenia in HELLP syndrome according to recent guidelines. Like in our cases, one had acute renal failure and the other had pulmonary oedema. All three of them needed ICU admission. Surgical intervention is indicated in cases of rupture of subcapsular haematomas. Blood transfusion is needed whenever there is anaemia because of haemolysis. Coagulation defects need platelets and fresh frozen plasma transfusion to combat disseminated intravascular coagulation. The choice of treatment in nonbleeding intraparenchymal hematoma with stable hemodynamic is conservative management. Timely diagnosis and management are important.

CONCLUSION

Postpartum HELLP should be always be considered especially in cases of preeclampsia. Because early diagnosis and treatment of HELLP prevents complications. If left untreated, postpartum HELLP syndrome and its complications could be life threatening as the evolution of the disease is rapid but with few clinical symptoms. Blood pressure monitoring in the immediate postoperative period is crucial as there are chance of patient developing pre-eclampsia as well as HELLP syndrome in the postpartum period in a previously normotensive pregnancy.

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