

Case Report

Sudden Postpartum HELLP Syndrome Resurgence: Case Report

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• HELLP syndrome; Preeclampsia; Postpartum

Abstract

We present a case of acute HELLP (Haemolysis, Elevated Liver enzymes, Low Platelets) syndrome at 28 gestational weeks. Shortly after delivery, all parameters initially improved. At 48 hours postpartum, we observed a resurgence of complete HELLP syndrome with a platelet drop to 17.000/mm³ requiring frozen platelet transfusion. With this case report, we want to draw attention on the risk of resurgence of HELLP syndrome in postpartum even after initial improvement.

ABBREVIATIONS

HELLP syndrome: Haemolysis Elevated Liver enzymes; LP: Low Platelets; AST: Aspartate Aminotransferase; ALT: Alanine transaminase; LDH: Lactate Deshydrogenase; Bpm: Beats per minute

INTRODUCTION

HELLP (Haemolysis, Elevated Liver enzymes, Low Platelets) syndrome is a disease of the pregnancy following early abnormal placentation and microcirculation injuries. It is described as a severe form of preeclampsia but can also be seen without hypertension and proteinuria [1-3]. It happens in 2 to 9 pregnancies in 1000 [1,3,4], before the 27th gestational week in 10% of cases, and after delivery, particularly up to 48 hours postpartum, in 30% [1,4]. In most women, the biological parameters worsen immediately after delivery with an improvement trend on the third day [4]. We present a case of early prenatal HELLP syndrome with an improvement of the blood tests shortly after the delivery followed by a sudden resurgence of HELLP syndrome at 48 hours postpartum.

CASE PRESENTATION

A 29-years-old primigravida presented at 28 weeks and 4 days of gestation with epigastric pain. She suffered from gastroesophageal reflux and heartburn for about a week but epigastric pain became unbearable despite painkillers. She reported no other symptom (no headache, no vision troubles, no nausea, no vomiting, no urinary problem, no oedema). She had a history of abdominal superficial venous thrombosis four years ago, with normal thrombophilia testing.

In the emergency room, her blood pressure was 120/85 mmHg. Physical examination was pertinent with reproductive

epigastric pain and positive Murphy sign. Cervix examination was reassuring, with a posterior, long and closed cervix. Biological findings included normal haemoglobin (14,6g/dl), low platelet count (111.000/mm³), leucocytosis (14.100/mm³), mild inflammatory syndrome (C-reactive protein 20mg/L, normal <10) and elevated liver enzymes (aspartate aminotransferase (AST) 163 IU/L (normal range: 15-37), alanine transaminase (ALT) 182 IU/L (normal range: 30-65), lactate deshydrogenase (LDH) 363 IU/L (normal range: 85-245)) with normal bilirubin values. Other parameters including coagulation, renal function and ions were normal. Proteinuria was absent on dipstick testing. A vaginal swab was also normal. An abdominal ultrasound showed no abnormality of the liver, biliary duct or pancreas.

Foetal ultrasound revealed an estimated foetal weight of 1144g (P17), normal umbilical and middle cerebral artery doppler testing, a biophysical profile of 8/8 and a normal aspect of the placenta. The cardiotocography showed normal variability and reactivity of the foetal heart rate, mean 135bpm, with unfelted regular contractions per 3 minutes.

The patient was admitted in the maternal intensive care unit for suspected HELLP syndrome. She was managed with intramuscular corticosteroid treatment (12mg betamethasone) for foetal maturation and regular blood tests.

Six hours after admission, a new blood sample was taken but the laboratory results arrived late, while the patient felt better, had lesser abdominal pain and had normal parameters. However, laboratory results eventually revealed thrombocytopenia (70.000/mm³), mild anisocytosis, normal haemoglobin (12,7g/dl), no identified schizocytes, increased liver enzymes (AST 245 IU/L, ALT 239 IU/L, LDH 407 IU/L), normal haptoglobin values and normal renal function. The results of the blood tests at 12 hours arrived soon thereafter and showed a continuing drop

of the platelet count ($49.000/\text{mm}^3$) and a continuing increase of the liver enzymes (AST 349 IU/L, ALT 337 IU/L, LDH of 561 IU/L, with normal γ GT and bilirubin values), associated to low haptoglobin value ($<8 \text{ mg/dl}$), normal haemoglobin ($12,8\text{g/dl}$), leucocytosis ($12.700/\text{mm}^3$), normal coagulation testing and normal renal function.

Facing this acute HELLP syndrome, intravenous magnesium sulfate treatment for foetal cerebral protection was started and an emergency foetal extraction by caesarean section was performed. The patient gave birth to a viable male weighing 980 grams with Apgar's scores of 4 and 9 at 1 and 5 minutes, respectively. Thrombophilia prophylaxis by subcutaneous low-molecular-weight heparin was started after the delivery.

After delivery, the initial clinical and biological evolution was good. The blood tests at 36 hours after the placental extraction showed: haemoglobin $11,7 \text{ g/dl}$, regression of thrombopenia ($109.000/\text{mm}^3$) and decreasing of hepatic cytolysis (AST 107 IU/L, ALT 188 IU/L, LDH 421 IU/L). Meanwhile, at 48 hours postpartum, although asymptomatic, the patient suffered from a sudden resurgence of complete HELLP syndrome: haemolysis (haemoglobin $8,5\text{g/dl}$, haematocrit 24%, LDH 1556 IU/L, low haptoglobin, $2/1000$ schizocytes, elevated bilirubin (total bilirubin $1,18 \text{ mg/dl}$ (normal range $0,20\text{-}1,00$)), elevated liver enzymes (AST 1302 IU/L, ALT 1040 IU/L, γ GT 93 IU/L (normal range: $5\text{-}55$) and low platelet count ($17.000/\text{mm}^3$). She was transferred to the intensive care unit for 24 hours, where she received one unit of frozen platelets. Hepatic ultrasound was performed and was normal.

The patient was again admitted in the maternal intensive care unit at 72 hours postpartum. The blood tests improved progressively. Magnesium sulfate treatment was stopped at five days postpartum. A new blood test was performed at 14 days postpartum and was normal. The patient continued thrombophilia prophylaxis by low-molecular-weight heparin during six weeks after delivery. Immune and coagulation tests were performed and were normal.

DISCUSSION

We report a case of acute complete HELLP syndrome in a primigravida at 28 weeks of gestation with epigastric and right upper quadrant pain as only clinical symptom. The patient was treated with a single dose of corticosteroids, intravenous magnesium sulfate and emergency delivery. Her blood tests quickly improved in the early postpartum but showed a sudden resurgence of complete HELLP syndrome at 48 hours postpartum with a drop of the platelets down to $17.000/\text{mm}^3$, justifying a single platelet transfusion. All parameters recovered progressively afterwards. Magnesium sulfate treatment was stopped at five days postpartum.

HELLP syndrome is a multifactorial disease, with a combination effect of multiple gene variants, metabolic factors, immune maladaptation, inflammatory response and environmental factors [3]. It typically concerns patients who suffer from severe preeclampsia, but it can be seen without hypertension and proteinuria [1-3], such as in our case report. The etiopathology of HELLP syndrome is still unclear but early abnormal placentation and microcirculation injuries are involved

[1-3]. This leads to microangiopathic haemolytic anaemia, liver microcirculation injuries and endothelial injuries. The triad 'haemolysis', 'elevated liver enzymes' and 'low platelets' is required to diagnose complete HELLP syndrome [1].

The differential diagnosis can be challenging because there is an overlap between different diseases, namely preeclampsia, acute fatty liver, thrombotic thrombocytopenic purpura, haemolytic uremic syndrome and antiphospholipid syndrome [1,2,4]. Other diseases involving the liver such as cholecystolithiasis and viral hepatitis can also be confused with HELLP syndrome [1,5]. Liver imaging by ultrasound or magnetic resonance may be helpful to make the correct diagnosis [1].

Blood tests are essential to diagnose HELLP syndrome. Microangiopathic haemolytic anaemia leads to decreasing haemoglobin, haematocrit and haptoglobin values, increasing of non-conjugated bilirubin (total bilirubin $> 1,2\text{mg/dl}$) and LDH levels ($>600 \text{ IU/L}$) and identification of schizocytes. Liver microcirculation injuries explain elevation of AST ($> 70 \text{ IU/L}$) and ALT values and hepatic function impairment. Endothelial injury leads to activation and aggregation of platelets. Increase of platelet peripheral consumption results in thrombocytopenia ($<100.000/\text{mm}^3$) [2,3]. The Tennessee (complete or incomplete HELLP syndrome) and Mississippi (3 classes based on platelet count, AST/ALT and LDH values) classifications can be useful for the diagnosis of HELLP syndrome [5].

Patients may have symptoms also seen in preeclampsia, such as pain in the right upper quadrant, epigastric pain, nausea, vomiting, oedema, headache, visual impairments [1,2,4]. The symptoms may exacerbate during the night and recover during the day [4].

HELLP syndrome can be complicated by hepatic haemorrhage or hepatic rupture [1]. Other complications are also seen in preeclampsia, such as eclampsia, cerebral ischemia, retroplacental hematoma, intrauterine growth restriction, foetal distress and premature delivery [2,4].

Delivery is the definitive treatment [3-5]. Other treatments are symptomatic. They are useful to continue the pregnancy, at least to allow foetal maturation by corticosteroids in the premature. Corticosteroids may also help to increase the platelet count [1,4,6]. Magnesium sulfate can be indicated in cases before 32 weeks of amenorrhea for foetal cerebral protection, or to prevent eclampsia crisis in severe preeclampsia with neurologic symptoms [2]. Platelet transfusion [2] can be given when the platelet count is lesser than $50.000/\text{mm}^3$.

CONCLUSION

HELLP syndrome is an insidious disease of the pregnancy. Evolution can be very rapid and severe with few clinical symptoms. The only definitive treatment is delivery. After delivery, it is important to be aware of the risk of sudden resurgence of HELLP syndrome even after an initial improvement. It is important to refer these patients in maternal intensive care units with trained teams to manage the possible severe complications.

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