Case Report

Abdelazim and AbuFaza ELLP syndrome as a variant of HELLP syndrome: Case reports

Ibrahim A. Abdelazim[1,2], Mohannad AbuFaza[1]

[1]Department of Obstetrics and Gynecology, Ahmadi Hospital, Kuwait Oil Company, Ahmadi, Kuwait, [2]Department of Obstetrics and Gynecology, Ain Shams University, Cairo, Egypt

Abstract

Background: The hemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome is a serious complication in pregnancy occurring in 0.5–0.9% of all pregnancies and in 10–20% of cases with severe pre-eclampsia. Previous studies described HELLP syndrome without hemolysis without any further details. **Objectives:** This report represents the criteria for the diagnosis of Abdelazim and AbuFaza elevated liver enzymes, low platelet count (ELLP) syndrome as a variant of HELLP syndrome. **Case Reports:** A 39-year-old woman, pregnant 32 weeks' gestation, previous five cesarean sections, admitted with severe pre-eclampsia (blood pressure 160/110 mmHg, proteinuria +3, 700 mg proteins/24 h urine, and protein/creatinine ratio ≥0.9 in spot urine sample). Laboratory investigation showed elevated liver enzymes, low platelet (PLT) count, and no evidence of hemolysis. A 31-year-old woman, pregnant 33^{+4} weeks' gestation, previous one cesarean section, admitted with severe pre-eclampsia (blood pressure 170/120 mmHg, proteinuria +2, 1200 mg proteins/24 h urine, and protein/creatinine ratio 1.1 in spot urine sample). Laboratory investigations showed elevated liver enzymes, low PLT count, and no evidence of hemolysis. Both patients delivered by cesarean section after stabilization of their blood pressure and dexamethasone for induction of fetal lung maturity and $MgSO_4$ for prevention of eclampsia. Both patients had uneventful intraoperative and postoperative stay in the hospital. The liver enzymes and the PLT count were completely normal on the 5th postoperative day, and they were discharged from the hospital in good general condition. **Conclusion:** Abdelazim and AbuFaza ELLP syndrome is variant of HELLP syndrome without hemolysis in women with severe pre-eclampsia. Abdelazim and AbuFaza ELLP syndrome diagnostic criteria are as follows: (1) Elevated liver enzymes; (2) Low PLT count; and (3) Absence of hemolysis (normal total and unconjugated bilirubin, absence of schizocytes, and polychromatic red cells in peripheral blood smear, and normal reticulocyte count).

Keywords: Abdelazim, AbuFaza, ELLP, HELLP, hemolysis

Introduction

Weinstein in 1982 defined the HELLP syndrome (H = Hemolysis, EL = Elevated liver enzymes, and LP = Low platelets) syndrome.[1] HELLP syndrome considered a complication of severe pre-eclampsia.[2-5] The diagnosis of HELLP syndrome requires the presence of the three major components of the syndrome, whereas partial or incomplete HELLP syndrome consists of only one or two elements of the triad (H or EL or LP).[6]

HELLP syndrome is a serious condition in its complete form and associated with maternal and fetal risks.[7,8]

HELLP syndrome occurs in about 0.5–0.9% of all pregnancies and in 10–20% of severe pre-eclampsia.[9] The majority of women with the HELLP syndrome have had hypertension and proteinuria, which may be absent in 10–20% of the cases.[5]

Women with partial HELLP syndrome have absent one or two elements of the HELLP triad (H or EL or LP). However, partial

Address for correspondence: Prof. Ibrahim A. Abdelazim, Department of Obstetrics and Gynecology, Ain Shams University, Cairo, Egypt and Ahmadi Kuwait Oil (KOC) Company Hospital, P.O. Box: 9758, Ahmadi - 61008, Kuwait.
E-mail: dr.ibrahimanwar@gmail.com

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or incomplete HELLP syndrome may progress to complete form of the disorder.[4]

The three components of the HELLP syndrome are hemolysis, elevated liver enzymes, and thrombocytopenia. Hemolysis is one of the major component of the disorder and is due to a microangiopathic hemolytic anemia. Red cell fragmentation caused by high-velocity passage through damaged endothelium of the small vessels. The presence of fragmented schizocytes in the peripheral blood smear reflects the hemolytic process.[10] Polychromatic red cells in blood smears and increased reticulocyte count reflect the compensatory release of immature red cells from the bone marrow.[11]

Hemoglobin released from hemolysis of the red cells converted to unconjugated bilirubin in the spleen or may be bound in the plasma by haptoglobin. The hemoglobin-haptoglobin complex is cleared quickly by the liver, leading to low or undetectable haptoglobin levels in the blood.[11] Low haptoglobin concentration can be used to diagnose hemolysis and is the preferred marker of hemolysis.[12-14] Thus, the diagnosis of hemolysis supported by high Lactate dehydrogenase (LDH) concentration and the presence of unconjugated bilirubin, but the low or undetectable haptoglobin concentration is more specific indicator of hemolysis.[12-14]

Hemolysis contributes substantially to the elevated levels of LDH, whereas increased asparate aminotransferase (AST) and alanine aminotransferase (ALT) levels are mostly because of liver injury. Plasma glutathione S-transferase-A1 (GST-A1) is more sensitive indicator for acute liver damage than AST and ALT.[14]

Thrombocytopenia [platelets (PLTs) $<150 \times 10^3/mm^3$] in pregnancy may be caused by gestational thrombocytopenia, immune thrombocytopenic purpura (ITP), pre-eclampsia, and HELLP syndrome.[15] Decreased PLT count in the HELLP syndrome is due to increased PLT consumption.[10]

At present, there are two major definitions for diagnosing the HELLP syndrome. In the Tennessee Classification System, Sibai proposed strict criteria for "true" or "complete" HELLP syndrome, which includes PLTs $\leq 100 \times 10^3/mm^3$, AST ≥70 IU/L, and LDH ≥600 IU/L.[5] Intravascular hemolysis diagnosed by abnormal peripheral blood smear, increased serum bilirubin (≥20.5 μmol/l or ≥ 1.2 mg/100 ml), and elevated LDH levels (>600 IU/l).[16]

The Mississippi-Triple Class System of HELLP syndrome depending on the low PLT count during the course of the syndrome.[4] Class 1 and Class 2 associated with hemolysis (LDH >600 IU/l) and elevated AST (≥70 IU/l), whereas Class 3 requires LDH >600 IU/l and AST ≥40 IU/l, in addition to the specific PLT count.[17] Class 3 HELLP syndrome considered as a clinical transition stage or a progression phase of the HELLP syndrome.[17]

The HELLP syndrome can be diagnosed simply on biochemical evidence.[18-20] Some authors require the presence of severe pre-eclampsia together with the biochemical markers to diagnose HELLP.[20-23]

Case Reports

A 39-year-old woman, pregnant 32 weeks' gestation, previous five cesarean sections, diabetic on oral hypoglycemic medications, admitted to Ahmadi hospital, Kuwait oil company (KOC) with severe pre-eclampsia (blood pressure 160/110 mmHg measured twice 6 h apart, lower limbs edema, headache, exaggerated ankle and knee reflexes, proteinuria +3 using urine dipsticks, 700 mg proteins/24 h urine, and protein/creatinine ratio ≥0.9 in spot urine sample).

Laboratory investigation showed elevated liver enzymes (ALT 211 IU/l, AST 432 IU/l, and LDH 670 IU/l), low PLT count 67×10^3, and no evidence of hemolysis.

Ultrasound fetal assessment showed single intrauterine pregnancy matching with her dates and estimated fetal weight (EFW) 1.700 Kg.

A 31-year-old woman, pregnant 33^{+4} weeks' gestation, previous one cesarean section on oral thyroxine for treatment of hypothyroidism, admitted to Ahmadi hospital, KOC with severe pre-eclampsia (blood pressure 170/120 mmHg measured twice 6 h apart, lower limbs edema, headache, epigastric pain, proteinuria +2 using urine dipsticks, 1200 mg of proteins/24 h urine and protein/creatinine ratio 1.1 in spot urine sample).

Laboratory investigations showed elevated liver enzymes (ALT 292 IU/l, AST 437 IU/l, and LDH 720 IU/l), low PLT count 56×10^3, and no evidence of hemolysis.

Ultrasound fetal assessment showed single intrauterine pregnancy matching with her dates with EFW 1.850 Kg.

Both patients delivered by cesarean section after stabilization of their blood pressure and dexamethasone for induction of fetal lung maturity[24] and $MgSO_4$ for fetal neuroprotection and for prevention of eclampsia.[25] Both patients had uneventful intraoperative and postoperative stay in the hospital. The liver enzymes and the PLT count were completely normal on the 5th postoperative day, and they were discharged from the hospital in good general condition.

Discussion

The HELLP syndrome may be misdiagnosed as viral hepatitis, cholangitis, and other serious conditions such as idiopathic thrombocytopenic purpura (ITP), acute fatty liver of pregnancy (AFLP), hemolytic uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP), and systemic lupus erythematosus (SLE).[16]

AFLP typically occurs between the 30th and 38th gestational weeks with history of malaise, vomiting, epigastric or right upper abdominal pain, and jaundice, whereas hypertension and proteinuria are usually absent. AFLP usually associated with low grade disseminated intravascular coagulation (DIC), prolonged prothrombin time, and partial thromboplastin time, low fibrinogen, and anti-thrombin concentrations.[14]

ITP is a syndrome with thrombocytopenia, which may be manifested by bleeding disorder with purpura and petechiae. Pregnancy does not increase the incidence of ITP and even with a very low PLT count, most cases of ITP not associated with neither maternal nor fetal morbidity.[26,27]

HUS and TTP are thrombotic microangiopathies that share some characteristics of the HELLP syndrome such as endothelial injury, PLT aggregation, micro-thrombi, thrombocytopenia, and anemia.[28]

The microvascular injury in HUS affects mainly the kidneys with signs and symptoms of renal failure. However, most cases of HUS appear in children and adolescents and caused by specific enterotoxin produced by *Escherichia coli* O157:H7.[29]

TTP is an extremely rare condition during pregnancy characterized by neurological dysfunction, abdominal pain, and bleeding. The spectrum of neurological abnormalities include headache, visual disturbances, transient paresis, and seizures.[30]

SLE is an autoimmune disorder characterized by deposits of antigen-antibody complexes in capillaries of multiple organs (kidneys, lungs, heart, liver, and brain). The clinical and laboratory findings in lupus nephritis are similar to those of severe pre-eclampsia. Antiphospholipid antibodies (APA; lupus anticoagulant and/or anticardiolipin antibodies) are present in 30–40% of the cases, whereas thrombocytopenia occurs in 40–50% and hemolytic anemia in 14–23% of women with SLE. Cerebral symptoms may develop because of vasculitis and/or cerebro-vascular occlusion that might lead to seizures.[5] APA associated with recurrent thrombosis and pregnancy loss. Antiphospholipid syndrome may also occur as a primary disease unrelated to SLE.[31]

Clinical symptoms such as headache, visual changes, epigastric pain, and nausea-vomiting have been suggested to be better predictors of adverse maternal outcome in HELLP syndrome.[32]

Spontaneous rupture of a sub-capsular liver hematoma in pregnancy is a rare, but life threatening complication that occurs in about 1% to < 2% of the cases with the HELLP syndrome.[33]

Abruptio-placentae, DIC, and subsequent severe post-partum bleeding are more common serious maternal complications with HELLP syndrome.[5] Bilateral permanent visual loss associated with retinopathy is a rare ophthalmic complication of HELLP syndrome.[34] In the literature, there are several case reports of cerebral bleeding associated with the HELLP syndrome.[35,36] Wound hematoma and infection are frequent complications in women with the HELLP syndrome undergoing cesarean delivery.[37]

Abruptio-placentae associated with the HELLP syndrome increases the risk of DIC as well as the risk of renal failure and blood transfusion.[38] Cerebral hemorrhage or stroke found to be the primary cause of death in 26% of HELLP syndrome.[39] The perinatal mortality rate related to the HELLP syndrome is ranging from 7.4% to 34%.[39] According to Gul *et al.*, the perinatal mortality was 34% before 32 weeks' gestation and 8% after the 32nd week.[40] Prematurity, placental insufficiency, with or without intrauterine growth restriction, and abruptio-placentae are the main causes of neonatal death.

The studied women had severe pre-eclampsia diagnosed by (1) High blood pressure ≥160/110 mm Hg measured twice 6 h apart. (2) Significant proteinuria using urine dipsticks, ≥500 mg of proteins/24 h urine, and protein/creatinine ratio ≥0.9 in spot urine sample. (3) Symptoms in form of headache, epigastric pain, and/or exaggerated ankle and knee reflexes.

Laboratory investigations of the studied women showed elevated liver enzymes (ALT, AST, and LDH) and low PLT count. The presence of hemolysis excluded by normal bilirubin (total and unconjugated) and absence of schizocytes, and polychromatic red cells in blood smear, and normal reticulocyte count.[10]

Chhabra *et al.*, described the partial HEELP as a syndrome contains only one or two of the three components HELLP syndrome.[41] Roelofsen *et al.*, described the ELLP syndrome as HELLP syndrome without hemolysis in a retrospective study investigating the maternal-fetal outcome after pregnancies complicated by (H) ELLP syndrome.[42]

A number of studies have included women with HELLP syndrome without evidence of hemolysis without further details.[43,44]

Abdelazim and AbuFaza ELLP syndrome is variant of HELLP syndrome without hemolysis in women with severe pre-eclampsia. Abdelazim and AbuFaza ELLP syndrome diagnostic criteria are as follows: (1) Elevated liver enzymes; (2) Low PLT count; and (3) Absence of hemolysis (normal total and unconjugated bilirubin, absence of schizocytes, and polychromatic red cells in peripheral blood smear, and normal reticulocyte count).

In general, there are three major options for the management of women with severe pre-eclampsia and HELLP syndrome.[14]

These include (1) Immediate delivery if HELLP syndrome diagnosed at ≥34 weeks' gestation; (2) Delivery after stabilization of the maternal condition within 48 h if HELLP syndrome diagnosed at 27–34 weeks' gestation; and (3) Expectant (conservative) management for >48–72 h may be

considered if HELLP syndrome diagnosed before 27 weeks' gestation.[14]

Both studied women delivered by cesarean section after stabilization of their blood pressure and dexamethasone for induction of fetal lung maturity[24] and $MgSO_4$ for fetal neuroprotection and for prevention of eclampsia.[25] Both patients had uneventful intraoperative and postoperative stay in the hospital. The liver enzymes and the PLT count were completely normal on the 5th postoperative day, and they were discharged from the hospital in good general condition.

This report represents the diagnostic criteria of Abdelazim and AbuFaza ELLP syndrome as a variant of HELLP syndrome without hemolysis, and large studies are going on to confirm the diagnostic criteria of the syndrome.

Conclusion

Abdelazim and AbuFaza ELLP syndrome is variant of HELLP syndrome without hemolysis in women with severe pre-eclampsia. Abdelazim and AbuFaza ELLP syndrome diagnostic criteria are as follows: (1) Elevated liver enzymes; (2) Low PLT count; and (3) Absence of hemolysis (normal total and unconjugated bilirubin, absence of schizocytes, and polychromatic red cells in peripheral blood smear, and normal reticulocyte count).

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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