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Pregnancy Complicated by HELLP Syndrome in a Patient with Morbid Obesity

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Abstract

Hemolysis, Elevated Liver enzymes, and Low Platelets count (HELLP) syndrome is a severe pregnancy complication associated with an impaired placental function. Rapid progression and non-specific symptoms render a definitive diagnosis exceptionally challenging. However, in spite of the fact that there is no specific treatment for HELLP syndrome, the delivery appears to be beneficial both for the fetus and the mother.

Our paper presents a case of a woman who developed HELLP syndrome at the 31st week of gestation. On admission, the pregnancy had already been complicated by gestational diabetes mellitus, third-degree obesity, and uncertain gestational age. Due to the BMI equal to 63.48 kg/m^2 (weight 162.5 kg height 1.6 m), cardiotocography was impossible, and the range of the ultrasound examination was limited. It is vital to note that in this case two pathologies were observed, which affected both the fetus and the mother. Initially, the patient presented with gestational diabetes which resulted in maternal hyperglycemia and excessive fetal growth. Furthermore, secondary placental insufficiency developed which manifested as HELLP syndrome. In turn, impaired placental function reduced maternal glucose levels due to inadequate insulinase activity and restricted the fetal growth. Therefore, establishing the diagnosis entailed the understanding of the two co-existing antagonist effects, concealing the potential symptoms of the disease. The treatment involved corticosteroids to manage fetal maturation as well as timely delivery. Cesarean section due to the fetal distress was performed in the 31st week of gestation-neonatal weight was 1360 g, and Apgar scores were 7,7,8,8.

The neonate in good condition was discharged on the 41st day following the cesarean section. Our case confirmed the relevance of liver and placental impairment biomarkers in HELLP syndrome diagnosis, particularly in view of the limited diagnostic tools in patients with obesity.

Keywords: HELLP syndrome; Obesity; Morbid obesity; Preterm delivery

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Introduction

Hemolysis, Elevated Liver enzymes, and Low Platelets (HELLP) syndrome is a severe multisystemic complication of pregnancy. It affects 0.5% to 0.9% of pregnancies, and nearly 25% of cases complicated by preterm preeclampsia [1,2]. Patients usually develop HELLP syndrome between the 27th and 37th week of gestation [1], presenting with non-specific symptoms, such as epigastric pain, or emesis. Notably, impaired placental function leading to materno-fetal imbalance is characteristic for patients suffering from HELLP syndrome, and maybe relevant for the diagnosis. In fact, in the described case, establishing the correct diagnosis was even more challenging considering the patient's comorbidities, as well as obesity. Nevertheless, no specific treatment for HELLP syndrome exists, and the treatment usually involves corticosteroids to manage fetal maturation, maternal hypotensive therapy, and timely delivery [3]. On the other hand, patients with obesity are additionally at risk of developing gestational diabetes and fetal macrosomia, although extensive growth of the fetus maybe limited by placental insufficiency.

Case Presentation

A 31-year-old Caucasian woman presented to the obstetric emergency department with hypertension which did not respond to the prescribed treatment. The patient was admitted to the hospital in the 31st week of gestation of her first pregnancy. The first obstetric consultation was performed in the 26th week of gestation, since the patient had previously been unaware she was pregnant, due to irregular menstrual periods and obesity - the patient weighed 162.5 kg and was 1.6 m tall, with the BMI amounting to 63.48 kg/m^2. Additionally, the gestational age was uncertain as a result of irregular menstrual bleeding, and the patient's blood pressure on admission was 170/105

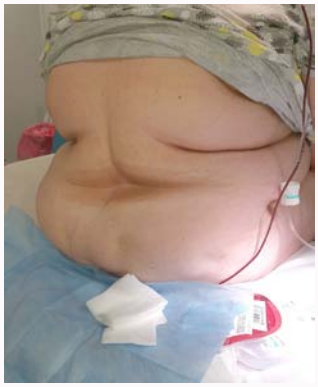

Figure 1: Preparation for anesthesia prior to the cesarean section.

mmHg.

We modified the existing hypotensive therapy, methyldopa 250 mg 3 times daily to 4 times daily, 500 mg methyldopa and nitrendipine 20 mg every 12 h. Due to the patient's obesity, CTG monitoring could not be performed, thus, we opted for Fetal Heart Rate (FHR) monitoring every 2 h. Additionally, we started antenatal corticosteroids therapy in order to stimulate fetal maturation, and we also ordered blood tests to control Hemolysis, Elevated Liver enzymes, Low Platelets (HELLP) syndrome, and multiple organ dysfunction.

On the basis of the interview, we found that during her first obstetric consultation in the 26th week of gestation, one of the ordered tests included Oral Glucose Tolerance Test (OGTT). The OGTT results were abnormal at each time point, indicating that the patient's condition fulfilled Gestational Diabetes Criteria (GDM) [4,5]. Her fasting glucose was 107 mg/dL; one hour after ingestion of 75 g glucose it reached 211 mg/dL, whereas after two hours it amounted to156 mg/dL. Her primary care obstetrician diagnosed gestational diabetes mellitus and recommended a special diet for GDM patients, as well as a regular glucose control treatment, which the patient did not comply with. She was diagnosed with gestational hypertension two weeks later; hence, she was prescribed 150 mg of aspirin as a potential prevention of preeclampsia.

Following hypotensive therapy modification, the patient's blood pressure remained below 140/90 mmHg. Glycosylated hemoglobin was 5.62%, indicating adequate glycemic levels despite the lack of ambulatory glycemic control. The patient did not require insulin intake to maintain euglycemia, and treatment of hypertension was successful. Nevertheless, laboratory findings revealed placental insufficiency. Daily urine protein loss exceeded 10 g per 24 h, where 0.3 g per 24 h constitutes a key proteinuria cut-off point. During the first three days of hospitalization, the platelet count dropped from 166 G/L to 40 G/L, and lactate dehydrogenase increased from 459000 U/L to 683000 U/L. The above mentioned results led us to the diagnosis of HELLP syndrome and to the decision of performing the cesarean section.

Every aspect of the cesarean section in patients with a BMI equal to 63.48 kg/m^2 constitutes a challenge (Figure 1). The Pfannenstiel incision allows operators to avoid dissecting most of the abdominal fat tissue, although the estimated blood loss during the surgery did not exceed 500 ml additionally, due to the risk factors, thromboprophylaxis was necessary in order to avoid postpartum complications. The neonate was born prematurely in the 31st week of gestation, with the body mass of 1,360 gm and Apgar scores 7,7,8,8. At birth, the neonate was within the third centile for the gestational age, which allows for the estimation of the fetal growth restriction, most likely due to the placental insufficiency. The neonate's arterial blood gas parameters measured in the umbilical artery presented pH of 7.28, and the base excess equal to -4.96. For the first ten days of life, the neonate remained in a serious condition in the intensive care unit. He required parenteral nutrition for the first 12 days and a single dose of pulmonary surfactant. After 41 days, the neonate was discharged from the neonatology department in good condition.

The mother's condition improved every day following the delivery. An early phase of wound healing was uncomplicated. Blood pressure was approximately 135/80 mmHg with reduced antihypertensive medications at the time of the patient's discharge. Laboratory findings improved in the days following the delivery; platelets increased to 156 G/L, lactate dehydrogenase decreased to 420 U/L. The patient was discharged four days following the delivery in good condition.

Discussion

The differential diagnosis of HELLP syndrome is challenging, particularly with a limited number of diagnostic tools due to the patient's obesity. The clinical presentation of HELLP syndrome varies among patients, therefore, it is extremely difficult to develop univariate diagnostic criteria based on the symptoms. Furthermore,

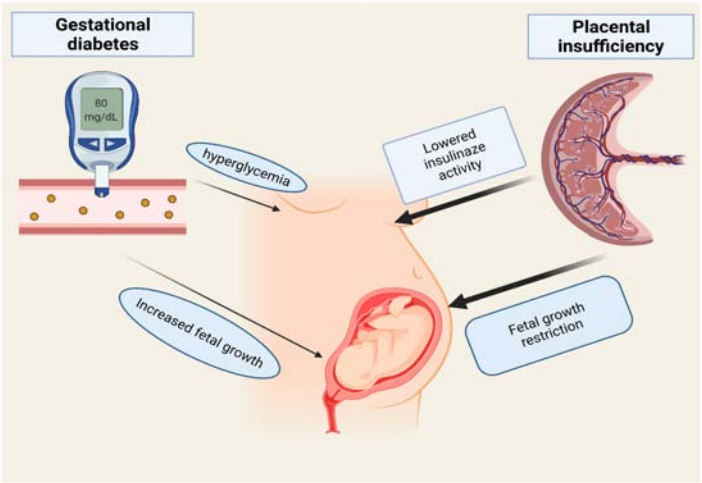

Figure 2: Antagonist effects of the placental insufficiency and gestational diabetes.

other complications of pregnancy may involve liver dysfunction which could mimic HELLP syndrome [6-8]. Hence, the typical differential diagnosis includes such disorders as thrombocytopenia of pregnancy, antiphospholipid syndrome, acute fatty liver of pregnancy, hemolytic uremic syndrome, viral hepatitis [3,9].

Although the pathophysiology of HELLP syndrome remains unclear, it is classified as a severe manifestation of preeclampsia, which usually manifests in the second half of pregnancy, with the number of cases increasing with each gestational week [8,10]. Abnormal placentation leads to the release of soluble toxic agents into the maternal circulation. These toxins result in vasculitis, endothelial dysfunction, and systemic maternal diseases [10]. In a normal pregnancy, the trophoblast migrates deeply into the uterine wall and spiral arteries inside the myometrium. However, the impaired trophoblast penetration leads to an insufficient arterial blood supply for the developing placenta, resulting in local ischemia. A reduced blood flow through uterine arteries can be observed during screening in the first trimester of pregnancy, indicating abnormal placentation and increasing the risk of placental insufficiency [11].

Two antagonist mechanisms complicated the patient's pregnancy. First, she was diagnosed with gestational diabetes, which was superimposed by third-degree obesity. Moreover, gestational diabetes leads to hyperglycemia which may cause an increased fetal growth. Nonetheless, due to the placental insufficiency, fetal growth was restricted despite elevated maternal glucose levels. Regardless of her obesity and gestational diabetes, the patient did not require insulin treatment. We hypothesize that the underlying explanation for this mechanism is the placental insufficiency, which resulted in a reduced placental insulinase activity. This, in turn, contributed to lower glycemia than would have been expected on the basis of the OGTT results and the mother's body weight.

Although the treatment of preeclampsia and HELLP syndrome is based on hypotensive therapy [12,13], the severity of the HELLP syndrome does not correlate with the level of hypertension [9], which indicates that both Mississippi and Tennessee classification systems are based on biochemical laboratory parameters [14]. Furthermore, liver and placental biomarkers represent promising options which could contribute to the identification of novel biochemical factors pointing to the HELLP syndrome development [15,16]. In fact, biochemical evidence of placental impairment predicts fetal growth restriction and maternal systemic disease. Additionally, it is applicable in patients with obesity where the ultrasound diagnosis is less reliable. We believe that screening tests involving biomarkers of placental and liver dysfunction may contribute to a decreased morbidity due to HELLP syndrome and, therefore, require further research.

Author Contribution

Conceptualization, DB, E.W-O.; Literature review DB, E.W-O. Original draft preparation DB E.W-O; Writing-Review and Editing E.W-O. Figures DB.

References

1. Dusse LM, Alpoim PN, Silva JT, Rios DRA, Brandão AH, Cabral ACV. Revisiting HELLP syndrome. Clin Chim Acta. 2015;451(Pt B):117-20.
2. Aloizos S, Seretis C, Liakos N, Aravosita P, Mystakelli C, Kanna E, et al. HELLP syndrome: Understanding and management of a pregnancy-specific disease. J Obstet Gynaecol. 2013;33(4):331-7.
3. Wallace K, Harris S, Addison A, Bean C. HELLP syndrome: Pathophysiology and current therapies. Curr Pharm Biotechnol. 2018;19(10):816-26.
4. Wender-Ożegowska E, Bomba-Opoń D, Brązert J, Celewicz Z, Czajkowski K, Gutaj P, et al. Standardy polskiego towarzystwa ginekologów i położników postępowania u kobiet z cukrzycą. Ginekologia i Perinatologia Praktyczna. 2017;2(5):215-29.
5. Diagnostic criteria and classification of hyperglycaemia first detected in pregnancy: A World Health Organization Guideline. Diabetes Res Clin Pract. 2014;103(3):341-63.
6. Ibdah JA, Bennett MJ, Rinaldo P, Zhao Y, Gibson B, Sims HF, et al. A fetal fatty-acid oxidation disorder as a cause of liver disease in pregnant women. N Engl J Med. 1999;340(22):1723-31.
7. Sibai BM. Imitators of severe pre-eclampsia/eclampsia. Clin Perinatol. 2004;31(4):835–52, vii–viii.
8. Baxter JK, Weinstein L. HELLP syndrome: The state of the art. Obstet Gynecol Surv. 2004;59(12):838-45.
9. Rimaitis K, Grauslyte L, Zavackiene A, Baliuliene V, Nadisauskiene R, Macas A. Diagnosis of HELLP syndrome: A 10-year survey in a perinatology centre. Int J Environ Res Public Health. 2019;16(1):109.
10. Rana S, Lemoine E, Granger JP, Karumanchi SA. Preeclampsia: Pathophysiology, challenges, and perspectives. Circ Res. 2019;124(7):1094-112.
11. Rolnik DL, Wright D, Poon LCY, Syngelaki A, O'Gorman N, de Paco Matallana C, et al. ASPRE trial: Performance of screening for preterm pre-eclampsia. Ultrasound Obstet Gynecol. 2017;50(4):492-5.
12. Amaral LM, Wallace K, Owens M, LaMarca B. Pathophysiology and current clinical management of preeclampsia. Curr Hypertens Rep. 2017;19(8):61.
13. Committee Opinion No. 623: Emergent therapy for acute-onset, severe hypertension during pregnancy and the postpartum period. Obstet Gynecol. 2015;125(2):521-5.
14. Martin JN, Brewer JM, Wallace K, Sunesara I, Canizaro A, Blake PG, et al. Hellp syndrome and composite major maternal morbidity: Importance of Mississippi classification system. J Matern Fetal Neonatal Med. 2013;26(12):1201-6.
15. Alese MO, Moodley J, Naicker T. Preeclampsia and HELLP syndrome, the role of the liver. J Matern Fetal Neonatal Med. 2021;34(1):117-23.
16. van Lieshout LCEW, Koek GH, Spaanderman MA, Heimel PJVR. Placenta derived factors involved in the pathogenesis of the liver in the syndrome of haemolysis, elevated liver enzymes and low platelets (HELLP): A review. Pregnancy Hypertens. 2019;18:42-8.