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HELLP SYNDROME WITH PERSISTENT SEVERE THROMBOCYTOPENIA: A CASE REPORT

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Abstract: INTRODUCTION: HELLP syndrome is a severe form of pre-eclampsia, referring to a group of clinical syndromes with hemolysis, elevated liver enzymes, and thrombocytopenia in pregnant women. It involves inadequate placentation during early pregnancy, associated with the effects of hepatic and coagulation cascades. It is associated with severe disease with rapid onset of clinical manifestations that often coexist with fetal growth restriction, with childbirth being the efficient and definitive treatment of HELLP syndrome. METHODOLOGY: We present a case of a patient with Pre-eclampsia aggravated by HELLP Syndrome, approved by the Ethics Committee, CAAE 52415521.6.0000.8069.
Keywords: HELLP Syndrome; pre-eclampsia; thrombocytopenia

INTRODUCTION

The HELLP syndrome is a severe form of pre-eclampsia, which refers to a group of clinical syndromes with hemolysis, elevated liver enzymes, and thrombocytopenia in pregnant women. Relatively low incidence 0.2-0.8%, and in 70-80% of cases coexist with pre-eclampsia, but rapid evolution and high risk to maternal and child health [1].

It is believed that there are combinations of multiple factors associated with the emergence of HELLP syndrome, genetic variants, as well as maternal and environmental factors that act simultaneously in the etiological mechanisms [1].

The syndrome also involves inadequate placentation during early pregnancy, associated with the effects of hepatic and coagulation cascades. Currently, inflammatory cytokines derived from the placenta and poor immunological adaptation are considered important triggers of the disease [2].

The classification of HELLP syndrome is based on two classifications, the first, from Tennessee, which is widely used for diagnosis, being the presence of microangiopathic hemolytic anemia with abnormal blood smear and low serum haptoglobin, elevated LDH levels above 600 IU /L and AST above 70 IU/L (both enzyme levels more than twice the upper limit of normal values) or bilirubin greater than 1.2 mg/dL and platelet count below $100 \times 10\ 9\ L\ -1$ as criteria for diagnosis. The second classification, from Mississippi, highlights the disorder's severity according to the platelet count's lowest point. Current literature describes a clinically less severe entity, referred to as incomplete HELLP syndrome, with only two criteria [2-3].

The typical clinical symptoms of the disease are pain in the right upper abdominal or epigastric quadrant, which may be associated with nausea and vomiting. Upper abdominal pain may be fluctuating, cramping-like. Patients report a history of malaise a few days before presentation. Up to 30–60% of women experience headache and 20% visual symptoms. However, HELLP syndrome can also present with nonspecific symptoms or subtle signs of preeclampsia or nonspecific viral syndrome-like symptoms. Symptoms generally progress continuously and their intensity changes frequently and spontaneously [4].

The onset of HELLP syndrome before 28 weeks of gestation accounts for approximately 20-30% of cases. It is associated with severe disease with rapid onset of clinical manifestations that often coexist with fetal growth restriction. In these cases, childbirth is the efficient treatment of HELLP syndrome [4].

METHODOLOGY

We present a case of a patient with Pre-eclampsia aggravated by HELLP Syndrome, approved by the Ethics Committee, CAAE 52415521.6.0000.8069.

DESCRIPTION OF CASE

Pregnant woman, 21 years old, mixed race, G5P0A4, gestational age (GA) 33.1 weeks, admitted complaining of scotomas and epigastric pain, already using magnesium sulfate, started by the transport team. During admission, she reported using acetylsalicylic acid (AAS) at a dose of 150mg/day, methyldopa at a dose of 1.5g/day, and enoxaparin sodium at a dose of 40mg/day started prenatally; indicated by a previous history of anti-phospholipid antibody syndrome (APS), acute myocardial infarction (AMI). She also reported having used corticosteroids for lung maturation at 26.0 weeks of gestation. The admission exams showed the following data: platelets 15,000/mm3; creatinine 1.5mg/dL; Lactic dehydrogenase: 1,767U/L; uric acid: 9.2mg/dL; ALT: 22U/L; AST: 29U/L, Labistix: 4+ proteins. Obstetric USG: Weight: 1713g (6% percentile); amniotic fluid index (AI): 7.1cm, GA 33.1 weeks; fetal heart rate of 159 bpm, Fundic placenta, grade II. Doppler with zero diastole and normal ductus venosus. FullPIERS 6.3%. The patient was admitted to an intensive care unit (ICU) bed for clinical stabilization and laboratory curve. It was decided to interrupt the pregnancy after requesting a reservation of blood products from the hospital's blood bank, however, it was reported that there was no stock of platelets in the entire state network. The patient progressed with clinical and laboratory worsening, and urgent termination of the pregnancy via discharge was indicated. The surgery was performed uneventfully, a Penrose drain was installed in the subaponeurotic region to monitor possible postpartum bleeding, the live newborn, the female patient, and APGAR scores; which in turn were measured in the first and fifth minutes as 4 and 8, respectively. The patient was referred to the Intensive Care Unit for monitoring and completion of the magnesium sulfate regimen. During her stay in the ICU, she remained stable and with laboratory improvements, except for thrombocytopenia, which persistently remained between 18,000-30,000/mm3. On the 17th day postpartum, pulse therapy with methylprednisolone 1g was indicated for 3 days, with levels improving to 145,000/mm3. After stabilization of platelet levels, the patient was discharged from the ICU to a rooming-in ward, followed by discharge from the hospital after the baby presented ideal weight. Patient and baby remain under follow-up at the service's pediatric and hematology outpatient clinics.

CONCLUSION

Thrombocytopenia in pregnant women requires urgent diagnosis and may be the first symptom of HELLP syndrome in cases of the absence of other prodromal clinical symptoms. An extension of diagnosis and treatment is necessary when disorders persist for more than three days.

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