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Hellp's Syndrome - Maternal Morbimortality, Clinical Case Report

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ABSTRACT:

Introduction: HELLP syndrome is a hypertensive disorder of pregnancy associated with severe maternal-fetal complications and representing high morbidity and mortality.

Objective: A clinical history of HELLP syndrome as a complication of severe preeclampsia was analyzed, highlighting the importance of early detection and appropriate treatment.

Clinical case: A young 26-year-old multi-gestation patient from the city of Loja, with a family history of arterial hypertension, was diagnosed with HELLP syndrome and severe preeclampsia in the 33rd week of gestation after five prenatal check-ups and due to the severity of her condition, it was decided to terminate the pregnancy by cesarean section.

Conclusion: The importance and urgency of early identification and treatment of HELLP syndrome and its complications, highlighting the need for an individualized approach based on the patient's laboratory studies and the importance of management in primary care and the patient's care.

KEYWORDS: HELLP syndrome; severe preeclampsia; arterial hypertension; pregnancy; morbidity; mortality.

I. INTRODUCTION

HELLP syndrome is a severe complication of hypertensive disorders of pregnancy and is characterized by the classic triad of hemolysis, elevated liver enzymes, and thrombocytopenia. Worldwide, it is estimated that HELLP syndrome affects 0.1% to 0.9% of pregnancies, as well as 10% to 20% of pregnancies with severe preeclampsia and 50% of cases of eclampsia (Bracamonte-Peniche et al., 2018). It should be added that this syndrome has a higher number of mortality cases, registering up to 24% in pregnant women and 34% in newborns or fetus (Runruil & Loza, 2020).

It is estimated that 27% of women in Latin America who are diagnosed with eclampsia develop Hellp syndrome, with a mortality rate of 14% (Runruil & Loza, 2020). In Ecuador, according to the Ministry of Public Health (MSP), it is estimated that Hellp syndrome occurs for every thousand pregnancies, three pregnant women with this diagnosis, causing cases of maternal mortality, which represents 2% of pregnant women and fetal mortality that represents up to 35% of cases; This is mostly correlated with the week of gestation in which the pregnant woman is at the time of delivery, the recurrence is 27% in subsequent pregnancies, and 30% incidence in pregnancies with hypertensive disorders with previous histories of Hellp syndrome (Andrade & Karanovic, 2017). As for the pathophysiology, the major component of the disturbance in HELLP syndrome involves endothelial damage, one of the most accepted theories being insufficient implantation of cytotrophoblast cells, which infiltrate the decidual portion of the spiral arteries but do not penetrate their myometrial segment. Therefore, these vessels do not transform into vascular channels of high capacitance but remain narrow, resulting in a decrease in placental flow and resulting in high perfusion velocity in the intervillous space, which generates shear stress on the trophoblast (Ramadan et al., 2018). Placental ischemia leads to activation and dysfunction of the maternal vascular endothelium, resulting in increased endothelin and thromboxane production, increased vascular sensitivity to angiotensin II, and decreased formation of vasodilatory agents (nitric oxide and prostacyclins). All these alterations lead to increased vascular resistance, platelet aggregability, coagulation system activation, and endothelial dysfunction, which translate into the symptoms and signs of the disease (Baxter & Weinstein, 2004).

Hemolysis is due to the rapid decrease in the number of erythrocytes, resulting from cell damage due to fibrin deposits generated by endothelial injury with subsequent rupture of red blood cells by contact with the damaged area. The elevation of hepatic enzymes is due to necrosis of the periportal parenchyma with fibrin deposits in the sinusoidal space, these deposits hinder hepatic blood flow, distending the liver. Therefore, the tension caused in Glisson's capsule originates pain in the epigastrium and right hypochondrium. Finally, the low platelet count is due to their increased consumption, platelets are activated and adhere to damaged vascular endothelial cells, which increases platelet turnover with a shorter lifespan (Yadav & Shah, 2017).

Diagnostic criteria are variable and inconsistent. Microangiopathic hemolysis is the hallmark of the triad. Sibai, in 1993, elaborated the diagnostic criteria and also made the classification of the type of HELLP syndrome (Sibai or Tennessee classification), which classifies it as complete or incomplete. The diagnostic criteria for this syndrome are levels of DHL greater than or equal to 600 U/l, AST greater than or equal to 70 U/l, and platelet count less than or equal to 100 000/mm3. The complete type is that which has all three parameters, and the incomplete type is that which has at least one of the above parameters (Sibai BM, 1986). The clinical manifestations may be highly nonspecific, leading to initial misdiagnosis and, thus, inadequate preliminary treatment. The vast majority of patients with this complication may have the same signs and symptoms of preeclampsia-eclampsia. Since HELLP syndrome is primarily a coagulopathic disease in origin, manifestations may commonly begin as epigastric pain, anemia, and platelet consumption by microangiopathic means. Initial symptoms have also been reported as episodes of vomiting and nausea in 50% of patients, and in some cases, headache and visual changes have been documented (Chowdhury et al., 2018). Multiple and varied severe complications have been identified, including cortical blindness, liver rupture, cerebral edema, subarachnoid hemorrhage, and, most commonly of all, hemorrhagic stroke. Hemorrhage usually occurs in cases where the patient's diastolic and systolic pressures rise 30 mmHg and 15 mmHg, respectively, above the mean in late pregnancy; proteinuria and edema, few cases of cortical blindness accompanied by bilateral parieto-occipital anomalies and possible subarachnoid hemorrhage may also occur (Joshi et al., 2010).

II. METHOD. CLINICAL CASE

The patient was 26 years old, with a clinical history of epilepsy diagnosed at 13 years of age under treatment, a family history of maternal aunt arterial hypertension, G3 P0 A1 C2 HV2, with a pregnancy of 33 weeks of gestation, who was transferred to a private hospital (Nataly Clinic), Loja-Ecuador, with symptoms of 18 hours of evolution, consisting of abdominal pain located in the epigastrium of moderate intensity (VAS 8/10), without irradiation, accompanied by abdominal distension, scotomas and moderate pulsating headache. During her prenatal controls (total 5), normal blood pressure figures were found, with normal routine examinations. An ultrasound was also performed, which showed a single cephalic fetus, FHR: 150 bpm, fetal biometry for 31 weeks, fetal weight of 1660 gr, posterior placenta grade I, and normal amniotic fluid. On admission to the level II hospital, physical examination showed a pregnant uterus, single live fetus, uterine height 28 cm, fetal heart rate (FHR) 146 beats per minute, with uterine activity 1/10 minutes, duration 20 seconds, intensity +/4; Genital region no evidence of hydrorrhea, vaginal examination: posterior cervix, softened, ajar; vital signs of: TA 160/115 mm/Hg, FC 92, T° 36.5C°, FR 20, positive proteinuria, saturation 97% (SCORE MAMA 7); paraclinical reported: hemoglobin 13.3 g/dl, hematocrit 38.1%, platelets 285,000, partial urine with traces of protein and negative for infection, creatinine 0. 7 mg/dl, basal glucose 104.50 mg/dl, urea 30.8 mg/dl, uric acid 8.3 mg/dl, TGO 223.6 U/L, TGP 107.1 U/L, lactate dehydrogenase 711.0 U/L. In addition, fetal well-being tests were performed with fetal monitoring rated as class II. Management was started with 0.9% saline solution at 80 cc/hour and then with magnesium sulfate 6 grams of impregnation in 20 minutes, continuing with maintenance drip at 2 grams/hour, hydralazine 5 mg intravenous STAT and termination of pregnancy via cesarean section; due to being far from delivery, and because Bishop score was not favorable and to achieve the best maternal and fetal benefit. Segmental cesarean section plus tubal sterilization was performed, obtaining a male newborn of 1455 grams, size of 42 cm adequate for gestational age, APGAR of 8 at one minute and nine at five minutes, age by Capurro 33 weeks, clear liquid with lumps. Post-surgical management continued with magnesium sulfate at 2 grams/hour until 24 hours were completed, analgesia with tramadol, and prophylactic antibiotic with cefazolin. The patient was sent to the intensive care unit due to the severity of HELLP syndrome. The patient remained normotensive during hospitalization. The toxemic control profile at 4 hours showed hemoglobin 11.4 g/dl, platelets 100,000, normal coagulation time, creatinine 0.9 mg/dl, TGO 200 U/l, TGP 1004 U/l, LDH 650 U/l, total bilirubin 0.9 mg/dl. In-hospital observation was performed for 10 days, finding the patient with diastolic blood pressure elevation and normalization of liver enzymes in daily controls. There were no complications during hospitalization and post-surgical observation, so in view of the adequate clinical evolution and also finding on the last day of in-hospital stay, platelets greater than 120,000 and liver enzymes in the normal range, the newborn was admitted to the neonatal intensive care unit. Ambulatory control was requested for the patient in 1 week for outpatient consultation with a hemogram and creatinine report. During the outpatient control, a week after her discharge, the patient was found with BP 130/90 mm/Hg, physical examination without alterations; the paraclinics reported Hemoglobin of 12.7 g/dl and platelets 322,000. Taking into account the normalization of laboratory tests, proteinuria in 24 hours less than 300 mg/24 hours. It was controlled at 12 weeks post cesarean section, with persistent elevation of diastolic blood pressure, so the patient had arterial hypertension as a result of the complication during gestation.

III.DISCUSSION

The situation described above represents a significant risk for the maternal and infant population. Pregnant mothers who do not receive adequate prenatal medical care are at risk of suffering complications during their pregnancies and deliveries. According to the WHO, early, regular, and comprehensive pregnancy care substantially reduces the risk of complications and maternal and perinatal death; it also favors adequate delivery care and, on the other hand, ensures favorable health conditions for mothers and their children in the periods immediately after birth, as well as reduces the incidence of congenital disability (MSP, 2015).

Pregnant women who develop HELLP syndrome face an increased risk of serious complications, including liver damage, coagulation problems, and dangerously high blood pressure. Sánchez Tapia et al. (2021) refer that this pathology was first described in 1982 by Weinstein and is within the compendium of diseases of placental origin developed during pregnancy in the postpartum period, characterized by hemolysis syndrome, elevated liver enzymes, and thrombocytopenia (HELLP syndrome).

The first component is hemolysis, defined as the presence of microangiopathic hemolytic anemia secondary to platelet consumption at sites of endothelial damage, evidenced by hyperbilirubinemia with predominance of the indirect form and increased LDH (Beckles & Parra, 2015), as evidenced in the clinical case presented, the patient had increased LDH throughout her hospitalization; platelets were within normal parameters. The second component of HELLP syndrome shows elevated liver enzymes due to obstruction of the sinusoids with fibrin, causing periportal or focal parenchymal necrosis. These lesions lead to increased intrahepatic pressure, forming subcapsular hematomas and producing distension of Glisson's capsule (Garcia et al., 2011), which causes the characteristic epigastric or right upper abdominal pain, in the clinical case, there was evidence of elevated liver enzymes. Regarding the severity of Hellp syndrome, the Mississippi classification considers a cut-off point for TGO or TGP with results greater than 40 U/L (Mississippi class III) and in Tennessee, values greater than 70 U/L for the same enzyme (Rivas & Mendivil Ciódaro, 2011). In the case presented, the patient showed on admission TGO and TGP with ranges above 80 U/L, thus fulfilling this criterion of elevated liver enzymes and elevated LDH.

It is important to make an adequate differential diagnosis, especially with pathologies related to acute fatty liver disease, thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, and systemic lupus erythematosus crisis. In the case presented, it was easy to differentiate by the patient's clinical manifestations, such as epigastrialgia, headache, scotoma and also elevated blood pressure, and proteinuria typical of preeclampsia and commonly present in this pathology. This was corroborated with the evolution of the patient towards improvement once the gestation was over, evidenced in the control paraclinical tests performed later during her hospitalization, and this undoubtedly supported the established diagnosis.

IV.CONCLUSION

HELLP syndrome is a serious complication of pregnancy, with high morbidity and maternal-perinatal mortality, especially when diagnosed in early gestational weeks, where the level of fetal viability is low, being necessary for a timely diagnosis in first-level care centers. It is important to mention that cases considered high risk for the mother justify the termination of pregnancy; cases under control allow the pregnancy to evolve until fetal maturity. During hospitalization, the evolution of the patient is observed, and in the event of minimal maternal risk and maximum fetal survival, the health team should consider termination of the pregnancy, making the corresponding interconsultations, especially with the intensive care unit and neonatology.

Another important factor to consider is arterial hypertension that persists at 12 weeks after delivery, also classified as chronic hypertension, with a prevalence of 27.7%, between the ages of 20 to 35 years, with 62.22% (Zayas et al., 2020). Our patient was one whose blood pressure persisted after 12 weeks postpartum. Therefore, the diagnosis of chronic hypertension was established.

REFERENCES

1) Joshi, D., James, A., Quaglia, A., and Westbrook, R. (Febrero de 2010). Liver disease in pregnancy. Lancet., 594-605. https://doi.org/10.1016/S0140-6736(09)61495-1 [Links]
2) (Prevalencia de la hipertensión arterial crónica posparto en pacientes con antecedentes de preeclampsia Prevalence of Postpartum Chronic Arterial Hypertension in Patients with History of Preeclampsia Yarine Leodnell Fajardo Tornés1* https://orcid.org/0000. (s.f.). (Prevalencia de la hipertensión arterial crónica posparto en pacientes con antecedentes de preeclampsia Prevalence of Postpartum Chronic Arterial Hypertension in Patients with History of Preeclampsia Yarine Leodnell Fajardo Tornés1* https://orcid.org/0000.
3) Andrade , N., y Karanovic , N. (2017). Prevalencia del síndrome de HELLP en gestantes con preeclampsia y eclampsia del servicio de Ginecología-Obstetricia del Hospital General Guasmo Sur período enero a junio del 2017. Universidad Católica de Santiago de Guayaquil.
4) Baxter, J., and Weinstein, L. (2004). HELLP syndrome: the state of the art. Obstet gynecol surv. , 59(12). https://doi.org/https://doi.org/10.1097/01.ogx.0000146948.19308.c5 [Links]
5) Beckles, M., and Parra, P. (2015). Beckles Maxwell M, Parra- Ramírez P, Diagnóstico y manejo oportuno del síndrome de HELLP. Acta Médica Costarricense. . ttp://www.redalyc.org/articulo.oa?id=43447102.

6) Bracamonte-Peniche, J., López-Bolio, V., Mendicuti-Carrillo, M., María Ponce-Puerto, J., Sanabrais-López, M., y Méndez-Domínguez, N. (2018). Características clínicas y fisiológicas del síndrome de Hellp. Rev. biomédica, 29(2). https://www.scielo.org.mx/scielo.php?script=sci_arttext&pid=S2007-84472018000200033#:~:text=Respecto%20al%20s%C3%ADndrome%20de%20HELLP,en%20el%20postparto%2C%2031%25.
7) Chowdhury, M., Huq, N., Islam R., R., y Khan, A. (2018). Quality of Care for the Maternal Complications at Selected Primary and Secondary Health Facilities of Bangladesh: Lessons Learned from a Formative Research. Int J Med Health Sci., 5(6). https://waset.org/abstracts/medical-and-health-sciences/80997 [Links]
8) Garcia , E., Grau , E., Torrubiano , Z., y Aguilera , S. (2011). Manejo conservador de un hematoma subcapsular hepático en un síndrome de HELLP. Progresos de Obstetricia y Ginecología, 54(9), 459–61.
9) MSP. (2015). Control Prenatal Guía de Práctica Clínica. Ministerio de Salud Pública del Ecuador. Dirección Nacional de Normatización.
10) Ramadan, M., Badr D., Hubeish, M., and Hijazi, H. (Enero de 2018). HELLP Syndrome, Thrombotic Thrombocytopenic Purpura or Both: Appraising the Complex Association and Proposing a Stepwise Practical Plan for Differential Diagnosis. J Hematol, 7(1). https://doi.org/doi: https://doi.org/10.14740/jh347w
11) Rivas , E., y Mendivil Ciódaro, C. (2011). Síndrome de Hellp: Revisión. Revista Salud Uninorte, 27(2). http://www.scielo.org.co/scielo.php?script=sci_arttext&pid=S0120-55522011000200010
12) Runruil, M., y Loza, M. (2020). Incidencia del síndrome de HELLP incompleto en gestantes cuyo rango de edad va desde los 18 a los 46 años, atendidas en el Hospital Teodoro Maldonado Carbo en el año2018. Hospital Teodoro Maldonado Carbo. http://repositorio.ucsg.edu.ec/handle/3317/14856
13) Sánchez Tapia, M., Chapues Andrade, G., Cáceres Miranda, M., y Medina Sarmiento, M. (2021). Características clínicas del síndrome de HELLP. Recimundo., 5(2), 169–178. https://doi.org/https://doi.org/10.26820/recimundo/5.(2)
14) Sibai BM, T. M.-N. (1986). Resultados materno-perinatales asociados con el síndrome HELLP en preeclampsia-eclampsia grave. 1986; 155:501- 509. Am J Obstet Gynecol .
15) Vigil-De Gracia , P., Rojas, J., Ramos, E., Collantes, J., y Quintero, A. (2015). Incidence of eclampsia with HELLP syndrome and associated mortality in Latin America. Int J Gynecol Obst. , 129(3). https://doi.org/https://doi.org/10.1016/j.ijgo.2014.11
16) Yadav , D., y Shah , A. (Marzo - abril de 2017). HELLP Syndrome: A Brief Review. JBPR. , 6(2). http://jbpr.in/index.php/jbpr/article/view/67/59 [Links]
17) Zayas, B., Granma, B., Tornés, F., Álvarez, A., y Millares, A. (2020). Prevalencia de la hipertensión arterial crónica posparto en pacientes con antecedentes de preeclampsia . Revista Cubana de Obstetricia y Ginecología, 46(2).