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A CASE REPORT ON HELLP SYNDROME

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ABSTRACT

HELLP syndrome is a rare condition that can occur during pregnancy and is associated with high blood pressure. It involves haemolysis (H), elevated liver enzymes (EL) and a low platelet count (LP) – (HELLP syndrome). [1] The estimated incidence in ~0.5% (range 0.17 – 0.85%). The term HELLP was designed in 1982. HELLP syndrome poses significant risks for both the mother and the fetus, increasing the likelihood of adverse complications for both. Treatment for HELLP syndrome typicallu involves the administration of medications to lower blood pressure and prevent seizures. Ultimately, the recommended HELLP syndrome is delivering the baby. We report a 26 years old female patient with HELLP syndrome.

KEYWORDS: HELLP, Preeclampsia, Eclampsia, Pregnancy.

INTRODUCTION

HELLP syndrome is a serious complication of preeclampsia and eclampsia during pregnancy. It is characterized by haemolysis, elevated liver enzymes and a low platelet count, which can be identified through a microangiopathic blood smear. [2] HELLP syndrome can occur as a complication of severe preeclampsia, but recent evidence suggests they may be separate disorders. Some patients with HELLP syndrome may not have preceding hypertension or proteinuria. Having a history of preeclampsia or HELLP syndrome increases the risk of recurrence in future pregnancies. Factors like multiparity, age and genetic predisposition can contribute to the risk. It has also been found that pregnant patients with SARS-CoV-2 infection have a higher risk of developing preeclampsia and

HELLP syndrome. HELLP syndrome affects approximately 0.5% to 0.9% of pregnancies. Most cases, around 70%, occur during the third trimester, while the remaining cases happen within 48 hours after delivery. The mortality rate for women with HELLP syndrome ranges from 0% to 24% and the perinatal death rate can be as high as 37%. [3]

Women may observe symptoms of HELLP syndrome during pregnancy or shortly after giving birth. The most common symptom is abdominal pain, usually in the right upper quadrant or mid epigastric-area, blurred vision, malaise or fatigue, edema (swelling), quick weight gain, nausea, vomiting less commonly blurred vision, headache. In rare cases the patients may also experience uncontrollable nosebleeds, seizures or uncontrollable body shakes. The diagnosis of HELLP syndrome involves a combination of clinical signs, laboratory tests and imaging studies. A laboratory test is typically performed to assess hemolysis, liver enzymes and platelet count. These tests include a complete blood count (CBC), liver function tests and a peripheral blood smear to examine red blood cells for signs of hemolysis. In addition to blood tests, imaging studies such as ultrasound to evaluate the liver, kidneys and the well-being of the baby can be performed and also monitor blood pressure and urine protein levels as these are important indicators of preeclampsia and HELLP syndrome. When it comes to treating HELLP syndrome, the primary focus is on the well-being of both the mother and the baby. The treatment approach depends on the severity of the condition and the stage of pregnancy. In cases nearing full term, delivery of the baby is usually recommended to relieve stress on the mother's body and prevent further complications. If the pregnancy is not yet at full term, the healthcare team may try to manage the condition and prolong the pregnancy while closely monitoring the mother's health. During treatment, healthcare providers closely monitor blood pressure, liver function, platelet count and overall health. Medications may be prescribed to control blood pressure, prevent seizures and manage complications. In severe cases where HELLP syndrome causes significant organ damage or poses a threat to the mother's life, an emergency caesarean section may be necessary, even if the baby is not at full term. [4]

CASE REPORT

A 26 years old primigravida at 37 weeks and 6 days was presented with complaints of labour pain, but on the way to hospital the patient experiences generalised tonic clonic seizure (eclampsia) associated with uprolling of eyes and frothing. Patient had a past medical history of grade 1 fatty liver and bilateral polycystic ovarian morphology. Her personal history shows primary subfertility, now it was a spontaneous conception. The patient had an event of bilateral pedal edema from the 19 weeks of pregnancy and all other investigations were normal except glucose challenge test (185mg/dl) and glucose tolerance test with 100gm glucose, FBS with 82mg/dl, after 1 hour with 151mg/dl, after 2 hour with 164mg/dl, after 3 hour with 122mg/dl which shows the patient had a impaired GTT and was on diet. At 20 weeks scan shows an impression of right uterine artery resistance increased with prediastolic notch but clinically insignificant as placenta is on left side, follow up blood sugars were normal, bilateral pedal

edema persisted hence advised BP check up from local hospital every week. Pulmonary induced hypertension work up was normal.

On the day of admission patient had 2 episodes of seizure during the time of transportation to hospital and after reaching the hospital, associated with tongue biting and frothing. On examination blood pressure was 140/90 mmHg, pedal edema was persisting and also had facial puffiness. During the time of seizure oropharyngeal sucking, head tilt and oxygen inhalation was given. There was a chance for shortness of breath, need for ventillatory support and a possibility of postpartum haemorrhage, disseminated intravascular coagulation, sepsis and pulmonary complication therefore they went for emergency lscs and it was conducted on the admitted date. Inj. Magnesium sulphate 4gm diluted in 12ml normal saline was given for 20 min for eclampsia and Inj. Pantoprazole 40mg IV, Inj. Metochlopromide 10mg IV, Inj. Amoxillin + Clavulanate 1.2g IV, Inj. Levitracetam 500mg IV, Inj. Vitamin K 10mg IV, Inj. Tranexamic Acid 500mg IV, Inj. Hydrocortisone 100mg IV, Inj. Labetalol 20mg IV, Inj. Ethamsylate 250mg IV was given as stat. Inj. Fentanyl 500mcg was given as analgesic for emergency lscs. A male baby of 2.8kg was delivered; baby did not cried at the time of birth and was immediately handled over to neonatal team. Baby was ventilated and shift to NICU after resuscitation but the baby expired after 9 days of delivery. Laboratory investigations showed in the table below.

PARAMETERS	DAY 1	DAY 2	DAY 3	DAY 4	DAY 6	DAY 11
Haemoglobin (mg/dl)	6.4	7.7	8.1	8.6	8.5	9
Platelet count (lakhs/cumm)	90,000		1.10	1.0	1.4	2.67
SGOT	165		55	31	34	24
SGPT	117		91	39	31	24
ALP	177		52	66	79	77
Sr. Creatinine	1.4		2.0	1.5	0.9	0.8
Urea	32		56	42	30	28

CT brain of the patient shows an impression of subarachnoid and intraventricular hemorrhage. Treatment approach for this patient includes T. Labetalol 200mg P/O 1-0-1, T. Nifedipine 10mg P/O 1-1-1, Inj. Cefoperazone sulbactam 1.5g IV 1-0-1, Inj. Pantoprazole 40mg IV 1-0-1, Inj. Levitracetam 500mg IV 1-1-1, Inj. Tranexamic acid 500mg IV 1-1-1, Inj. Meropenem 500mg IV 1-1-1, Neb. Levosalbutamol 0.63mg 1-0-1, Syp. Levodropropizine 5ml P/O 1-1-1, Neb. Budesonide 0.5mg 1-1-1, T. Trypsin chymotrypsin P/O 1-1-1, Syp. Lactulose 15ml P/O 0-0-1, Inj. Placenta extracts 2ml IM 1-0-0, T. Methylprednisolone 4mg P/O 1-1-1 and T. Montelukast + Levocetrizine 5mg + 10mg P/O 0-0-1. After 13 days of treatment patient symptoms has been

improved and discharged with following medications T. Labetalol 200mg P/O 1-0-1, T. Amlodipine 5mg P/O 0-1-0, Inj. Placenta extracts 2ml IM 1-0-0 on alternative days for 10 more days, Syp. Lactulose 15ml 0-0-1, T. Levitracetam 500mg 1-1-1, Syp. Levodropropizine 5ml P/O 1-1-1, T. Trypsin chymotrypsin P/O 1-1-1, T. Pantoprazole 40mg P/O 1-0-1, T. Montelukast + Levocetrizine 5mg + 10mg P/O 0-0-1 and T. Methylprednisolone 4mg P/O 1-1-1.

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