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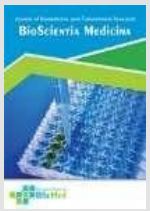

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Intensive Care Management of Eclampsia with HELLP Syndrome: A Case Report

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A B S T R A C T

Background: The incidence of HELLP syndrome is approximately 0.1-1% of all pregnant women and 5.5% of patients admitted to the ICU. Hemolysis, elevated liver enzymes, and thrombocytopenia are the characteristics of HELLP syndrome. **Case Presentation:** A 38-year-old patient with diagnosed G4P3A0L3 31-32 weeks of preterm pregnancy + eclampsia on $MgSO_4$ regimen, HELLP syndrome + twice previous Sectio cesarean + breech presentation. On physical examination, the general condition was blood pressure 199/103 mmHg. The results of laboratory tests post-op were hemoglobin 7,1 g/dL, leukocytes 15.930, hematocrit 24%, and platelets 38,000. The results of other laboratory tests showed decreased albumin levels (Alb 2,4), increased levels of total bilirubin 14,7, direct bilirubin 10,8 and indirect bilirubin 3,9, increased liver enzyme SGOT 2001 SGPT 513. **Conclusion:** HELLP syndrome is a threatening clinical problem. Appropriate and adequate management, especially in the Intensive care unit, is needed to prevent severe complications to reduce morbidity and mortality rates in patients with HELLP syndrome.

1. Introduction

The incidence of HELLP syndrome is approximately 0.1-1% of all pregnant women and 5.5% of patients admitted to the ICU, with a mortality rate of 66%.[1,2] The prevalence of HELLP syndrome out of all complications is 4% to 12%. Hemolysis, elevated liver enzymes, and thrombocytopenia are the characteristics of HELLP syndrome.[3,4] HELLP syndrome is a life-threatening hypertensive disease associated with severe preeclampsia and eclampsia with three characteristic symptoms and common causes of ICU admission among all obstetric patients.[5]

HELLP syndrome is a dangerous health problem worldwide. The maternal mortality rate due to HELLP syndrome is 3.5%-24.2%, and the perinatal mortality rate is 7.7%-60%. The main cause of maternal death due to HELLP syndrome is caused by coagulopathy problems, such as disseminated intravascular coagulation, pulmonary and cerebral edema, liver hemorrhage, placental abruption, and hypovolemic shock.[5,6] Because HELLP syndrome is life-threatening, intensive care management is needed when more than two organ systems fail or require mechanical ventilator support.[5]

Intensive care is required in approximately 10% of patients with preeclampsia with complications, especially HELLP syndrome. Management of HELLP syndrome patients is complex and requires multidisciplinary care from obstetricians, intensive care physicians, and ICU nurses. Further studies are needed to reduce maternal mortality due to HELLP syndrome by minimizing complications and damage

and factors that affect the length of ICU treatment.[1,7]

2. Case Presentation

A 38-year-old patient with diagnosed G4P3A0L3 31-32 weeks of preterm pregnancy + Eclampsia on $MgSO_4$ regimen, HELLP syndrome + twice previous Sectio cesarean + breech presentation. The patient had been hospitalized in a previous hospital for about two days and had been given a regimen of $MgSO_4$, methyldopa 3x500 mg, and dexamethasone 2x6 mg 1 week ago. The patient checked her blood pressure with a midwife and got 196/110, and then she went to the hospital. She had been checked laboratory with thrombocyte 53.000 SGOT/SGPT: 441/138. Because of a limited facility, the patient was referred to General Hospital for further management with an IV line ($MgSO_4$ regiment) and a catheter inserted. The patient had no complaints of headache, epigastric pain, blurred vision and signs of labor. The patient controls the obstetrician every month from one month of gestation. The patient was known to have high blood pressure at seven months of gestation, received 3x500 mg of methyldopa, and did not take medication regularly.

On physical examination, the general condition was blood pressure 199/103 mmHg, pulse 99x per minute, respiratory rate 22x per minute, oxygen saturation 98% with nasal cannula 5 lpm. There is an icteric sclera and skin, uterine fundus height of 23 cm, no contractions and fetal heart rate of 150-155x per minute. There was no vaginal bleeding. On laboratory examination, found thrombocytopenia and urine protein +2. Ultrasonography found 31-32 weeks according to biometry, fetal alive, singleton, intrauterine, and breech presentation. The patient was diagnosed with G4P3A0L3 31-32 weeks of preterm pregnancy + severe preeclampsia on a maintenance dose of $MgSO_4$ regimen from another institution + HELLP syndrome + twice previous CS + breech presentation + complete lung maturation from other institution.

A cardiologist consulted the patient and received nicardipine therapy starting at 0.5 mcg/KgBB/min with a target TDS <160mmHg if the target achieves change with Methyldopa 3x500mg. According to Lee's revised score, MACE 2; is low risk (0.9%). The internist also consulted the patient to receive 10 units of thrombocyte transfusion therapy. The surgical operation should be done with blood pressure <140mmHg. The patient underwent stabilization and termination of pregnancy by Caesarean section. The patient was treated at ICU after the termination of pregnancy.

Day one at intensive care, the patient was composmentis, Blood Pressure: 145/72 mmHg, Heart Rate: 78 x/m, Temperature: 36,5, Respiratory Rate: 20, SpO_2: 99%, from abdominal examination uterine fundal palpated two fingers below umbilical, contraction (+), Genitalia: vaginal bleeding normal, drip oxytocin 20 IU 20 tpm, Ceftriaxone 2 x 1 gram IV, Nicardipine 5 mg/ hour, Methyldopa 3x500mg, Adalat oros 1x30 mg. The results of laboratory tests post-op were hemoglobin 7,1 g/dL, leukocytes 15.930, hematocrit 24%, and platelets 38,000. The results of other laboratory tests showed decreased albumin levels (Alb 2,4), increased levels of total bilirubin 14,7, direct bilirubin 10,8 and indirect bilirubin 3,9, increased liver enzyme SGOT 2001 SGPT 513 and increased procalcitonin >1,96. The patient was given a transfusion of PRC and platelets.

On the second day, the patient was composmentis, and the vital sign was normal; the laboratory results also found hyponatremia, severe anemia, and thrombocytopenia. Bilirubin levels are still elevated, and liver enzymes are decreased. A blood gas analysis was carried out with the results pH 7.519, pCO_2 27.9, pO_2 116.2, SO_2% 99, HCO3 6.5, BEecf -0.2, PO_2/FIO_2 569.6. On the fourth day in ICU, routine blood tests showed hemoglobin of 6.2 g/dL and platelets of 88,000. A blood gas analysis was carried out with the results pH 7.44, pCO_2 30, pO_2 97, SO_2% 98, HCO_3 20.6, BEecf -3.7. From the abdominal ultrasound, there is no intraabdominal bleeding. The patient was given three units of PRC transfusion and two units of platelets.

On the fifth day, in the ICU, the vital sign was normal; the laboratory result was hemoglobin 9,6 g/dL, platelet 132.000, and procalcitonin 1.51; a blood gas analysis was carried out with the results pH 7.52, pCO_2 26, pO_2 116.1, SO_2% 99.3, HCO_3 21.5, BE-0.1. The patient was given of PRC transfusion. On the sixth day in the ICU, the laboratory result improved with hemoglobin11.0, platelet 105.000, albumin 2.9, procalcitonin 1.05, liver enzymes are within normal; SGOT 12, SGPT 19. then the patient moved to the obstetric and gynecology high care unit.

3. Discussion

Termination of pregnancy is the primary management of HELLP syndrome. Studies show that most cases improve laboratory parameters within 72 hours after delivery. However, if there is no improvement in laboratory data after 72 hours of labor, there may be a tendency to worsen the disease condition, including organ failure. If not managed appropriately and adequately will worsen the outcome.[8]

The main principle of HELLP syndrome management is to optimize the mother's condition and prevent maternal and infant mortality. In HELLP syndrome, microvascular endothelial damage causes intravascular platelet activation. This activation causes the secretion of serotonin and thromboxane A, which will cause vasospasm, and the agglutination process, thus exacerbating the endothelial damage. The syndrome may be a complication or progression of severe preeclampsia.[9,10]

The results of laboratory tests post-op were hemoglobin 7,1 g/dL, leukocytes 15.930, hematocrit 24%, and platelets 38,000. The results of other laboratory tests showed decreased albumin levels (Alb 2,4), increased levels of total bilirubin 14,7, direct bilirubin 10,8 and indirect bilirubin 3,9, increased liver enzyme SGOT 2001 SGPT 513. HELLP syndrome was diagnosed according to the following criteria: the presence of hemolysis (total bilirubin > 1.2mg/dL or serum LDH > 600IU/L or decreased hemoglobin and hematocrit levels), elevated liver enzymes (AST> 70 IU/L and ALT > 70 IU/L) and thrombocytopenia (< 150000 cells/mm^3.[3]

The pathogenesis of HELLP remains unclear, in HELLP syndrome there is abnormal remodeling of the spiral artery, systemic endothelial dysfunction, defective trophoblast differentiation, hypoxia, ischemia, hypoperfusion, immunological and genetic factors may play a role. HELLP syndrome has more severe liver inflammation and abnormal coagulation system.[2] HELLP syndrome is a complex multisystem disorder and requires multidisciplinary care. In cases with HELLP syndrome requiring mechanical ventilation assistance aims to maintain adequate oxygenation while minimizing metabolic activity in other organs.[11]

The patient had no complaints of headache, epigastric pain, blurred vision and signs of labor. The patient controls the obstetrician every month from one month of gestation. The patient was known to have high blood pressure at seven months of gestation. On physical examination, may have ascites or extremity edema. Right upper quadrant or epigastric tenderness is noted.

Clinical symptoms of HELLP syndrome may vary. Patients may present with central epigastric pain or right upper quadrant abdominal pain, nausea, vomiting, and fatigue. HELLP syndrome symptoms related to abnormalities can include jaundice, leg swelling, increased abdominal circumference, headache, and visual disturbances. Patients may experience severe bleeding, acute kidney injury, placental abruption, liver hematoma, or retinal ablatio.[12]

While in the ICU, blood pressure is closely monitored. Vigilance against the development of possible complications such as pulmonary edema and stroke is also required. On physical examination, patients have hypertension with a blood pressure >199/103 mmHg. The American College of Obstetricians and Gynecologists (ACOG) recommends antihypertensive therapy for severe preeclampsia and HELLP syndrome if blood pressure is ≥160/110 mmHg. Because the uncontrolled increase in high

blood pressure will cause cerebrovascular injury leading to hypertensive encephalopathy, if there is too high an increase in intracranial pressure, it can result in intracranial hemorrhage or cerebral edema. Hypertensive management may include antihypertensive drugs (IV bolus dose of 5-10 mg hydralazine given over 2 minutes or IV bolus dose of 20-80 mg labetalol over 2 minutes or oral dose of 10-20 mg nifedipine) used to maintain blood pressure within a safe range (140 150/90 100) without compromising brain perfusion and uteroplacental flow. Second-line alternatives include labetalol or nicardipine infusion. Current ACOG guidelines recommend a target blood pressure range of 140-150/90- 100 mmHg.[5,13]

Postoperatively, the patient required mechanical ventilator support and was admitted to the Intensive care Unit, where she was cared for by a multidisciplinary team consisting of obstetricians, anesthesiologists, hypertensive renal physicians, hematology department and intensive care nurses. Despite propofol sedation, anticonvulsant drugs such as lorazepam, levetiracetam and phenytoin were required to control seizures.[14]

The results of laboratory tests post-op were hemoglobin 7,1 g/dL, leukocytes 15.930, hematocrit 24%, and platelets 38,000. For patients with class I HELLP syndrome, Platelet transfusion is recommended for severe thrombocytopenia or platelets <50,000/µL. In addition, patients experiencing active bleeding with thrombocytopenia and platelet count <20,000/µL should receive platelet transfusion to prevent excessive bleeding during labor. However, repeated platelet transfusions are not necessary due to the short half-life of platelets.[5]

Transfusion of blood products is permitted depending on the patient's clinical condition and laboratory findings. In cases with tachycardia, tachypnea, and cerebral hypoxia problems, blood product transfusion is performed according to the patient's haemoglobin (Hb) level. Hemodynamics can be stabilised in most medical and surgical patients if the metabolic problem is resolved.[1]

In managing HELLP syndrome, start transfusion if the haemoglobin level is 7-8 g/dl. One unit of FFP is given for every unit of PRC. Platelet transfusion is started when platelet levels are ≤20,000-25,000/µL in vaginal delivery and when platelets are <50,000/µL in cesarean section and patients with class I HELLP syndrome. We prefer apheresis platelet transfusion.[1]

In HELLP syndrome, serious complications that may occur are coagulopathy, bleeding, and DIC. Rapid hemostatic management and appropriate and rapid clinical and laboratory monitoring by a multidisciplinary team can improve outcomes in HELLP syndrome. It is recommended to start transfusion if there is clinical suspicion of coagulopathy.[15]

The results of laboratory tests post-op were showed decreased albumin levels (Alb 2,4), increased levels of total bilirubin 14,7, direct bilirubin 10,8 and indirect bilirubin 3,9, increased liver enzyme SGOT 2001 SGPT 513. In HELLP syndrome, fluid management is more challenging if there is bleeding, coagulopathic disorders, or hypovolemia problems. Improvement of the systemic circulation after bleeding is also significant; this can be managed by administering blood products to avoid coagulopathy if bleeding is ongoing. Supplemental fluid therapy may replace fluid loss with close hemodynamic monitoring. In HELLP syndrome, the problem of oliguria is also a concern. However, if too much fluid is given to force urine output, it can cause excess iatrogenic fluid and pulmonary oedema. It is crucial to monitor volume status and hemodynamics to avoid excessive resuscitation. This monitoring can also be assisted by echocardiographic assessment, volume status assessment, serial clinical examinations, and metabolic status assessment (lactate and central venous oxygen saturation).[14]

The causes of maternal death in HELLP syndrome are usually mainly due to renal impairment, hepatic haemorrhage, coagulopathy, and hypovolemic shock. Sudi Ataskhoei showed that the median period of ICU stay with preeclampsia and HELLP syndrome was three days. However, if there are complications and

severe disorders, the length of treatment can be longer, around 5 - 7 days.[2,16]

4. Conclusion

HELLP syndrome is a threatening clinical problem. Appropriate and adequate management, especially in the Intensive care unit, is needed to prevent severe complications to reduce morbidity and mortality rates in patients with HELLP syndrome.

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