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Email : editorijhess@gmail.com

Partial HELLP Syndrome Complicated by Leukocytosis in Pregnant Woman Undergoing Intensive Care in a Hospital : Case Report

Richa Difayana Sari[1)], Hana Iftitah Hidayati[2)], Yuliana Arisanti[3)]
[1)]General Practitioner of Grha Husada Hospital, Gresik
[2)]Department of Obstetric & Gynecology Ibnu Sina General Hospital, Gresik

*Corresponding Author
Email : richadifayana@gmail.com

Abstract

HELLP syndrome (Hemolysis, Elevated Liver enzymes, and Low Platelet count) is a severe obstetric condition that may occur during pregnancy or postpartum, often associated with preeclampsia. Partial or incomplete HELLP syndrome presents a diagnostic challenge when only one or two of the three defining criteria are met. This report describes a 30-year-old woman, gravida 2 para 1 at 33–34 weeks of gestation, who developed eclampsia and partial HELLP syndrome complicated by severe leukocytosis. Despite an emergency cesarean section and intensive care management, including magnesium sulfate, corticosteroids, broad-spectrum antibiotics, and ventilatory support, the patient's condition deteriorated to sepsis, acute respiratory distress syndrome (ARDS), and multi-organ dysfunction. Extreme leukocytosis (>40,000/µL) complicated the diagnostic process, mimicking infectious etiology. This case emphasizes the importance of differentiating inflammatory from infectious leukocytosis in HELLP syndrome and underscores the necessity of a multidisciplinary approach, timely delivery, and intensive monitoring. Even in its partial form, HELLP syndrome remains a life-threatening disorder that requires early recognition and aggressive management to improve maternal outcomes.

Keywords: Partial HELLP Syndrome, Leukocytosis, Eclampsia, Sepsis, Multi-Organ Dysfunction.

INTRODUCTION

HELLP syndrome (Hemolysis, Elevated Liver enzymes, and Low Platelets) is a severe obstetric complication typically arising during pregnancy or the postpartum period. It is often associated with preeclampsia but may also occur independently. While once regarded merely as a variant of severe preeclampsia, recent evidence suggests that HELLP syndrome may represent a distinct clinical entity, given that it can develop in the absence of hypertension or proteinuria.

The *partial* or *incomplete* form of HELLP syndrome refers to cases in which only one or two of the three diagnostic components are present such as elevated liver enzymes and thrombocytopenia without overt hemolysis. This incomplete presentation complicates diagnosis and management, requiring careful differentiation from other hematologic or hepatic disorders such as thrombotic microangiopathies (TMAs), acute fatty liver of pregnancy, or viral hepatitis. The pathogenesis of HELLP syndrome involves systemic endothelial dysfunction, microangiopathic hemolysis, and abnormal activation of the coagulation cascade. These processes lead to hepatic injury and platelet consumption, culminating in multiorgan involvement. Early identification of laboratory abnormalities, such as increased AST, ALT, LDH, and decreased platelet count is crucial for timely intervention to reduce both maternal and perinatal morbidity and mortality.

Leukocytosis during pregnancy is relatively common, especially in late gestation and the postpartum period, reflecting physiological immunomodulation. However, marked or persistent leukocytosis warrants thorough evaluation to exclude infection, hematologic disorders, or inflammatory responses secondary to obstetric complications such as preeclampsia and HELLP syndrome. Reported cases indicate that leukocytosis may complicate the diagnostic process and delay appropriate treatment when misinterpreted as infectious in origin.

The coexistence of *partial HELLP syndrome* and leukocytosis presents a diagnostic and therapeutic dilemma. In such cases, clinicians must determine whether leukocytosis is reactive or pathological, as unnecessary antimicrobial use and invasive investigations may follow. A multidisciplinary approach involving obstetricians, intensivists, and hematologists is essential to guide

targeted management, especially when deciding between conservative therapy and pregnancy termination in the setting of maternal instability. Severe or complicated HELLP syndrome casesparticularly those associated with coagulopathy, hepatic rupture, or renal failure often require intensive care unit (ICU) management. In this setting, treatment focuses on hemodynamic stabilization, correction of coagulopathy, transfusion support, blood pressure control, and magnesium sulfate for seizure prophylaxis. Prompt delivery remains the only definitive treatment in progressive cases. Recent reports underscore the importance of standardized critical care protocols and multidisciplinary coordination to improve outcomes.

In the present case, a pregnant woman developed partial HELLP syndrome complicated by significant leukocytosis, requiring admission to intensive care. This scenario raises key clinical questions: How can clinicians differentiate reactive from pathological leukocytosis in HELLP? What thresholds should trigger antimicrobial therapy or hematologic evaluation? And when should pregnancy termination be prioritized over conservative management? Emerging literature provides frameworks for approaching these complex diagnostic and therapeutic decisions.

Beyond the acute phase, HELLP syndrome has implications for long-term maternal and neonatal health. Women with prior HELLP or preeclampsia are at increased risk for recurrence and cardiovascular complications in subsequent pregnancies. Hence, postpartum follow-up including liver function, hematologic recovery, and preconception counseling are recommended to mitigate future risk.

RESEARCH METHODS

This case report aims to describe the clinical presentation, diagnostic workup, and intensive care management of a patient with partial HELLP syndrome complicated by leukocytosis. By correlating clinical findings with recent evidence, this report contributes to a better understanding of diagnostic challenges and therapeutic strategies for atypical and complicated HELLP presentations in tertiary care settings.

RESULTS AND DISCUSSION

Mrs. P, a 30-year-old woman, gravida 2 para 1 with 33–34 weeks of gestation and postoperative day six after cesarean section, complained of severe dizziness since the morning before hospital admission. No other complaints were noted. She visited a midwife, where her blood pressure was recorded at 222/102 mmHg, and was then referred to the hospital. The patient reported her last menstrual period on November 13, 2024, with an estimated due date of August 20, 2025. She had attended antenatal care four times with a midwife and once with an obstetrician. The patient had a history of preeclampsia during her first pregnancy. Upon admission, her hemodynamic status was stable with a blood pressure of 111/71 mmHg. Fundal height was 23 cm, fetal heart rate was 161 beats per minute, and there were no contractions or abdominal distension. A surgical scar was visible, and vaginal examination showed a closed cervix.

At 10:00 p.m., the patient experienced a seizure lasting 60 seconds, during which she lost consciousness. Her blood pressure reached 215/153 mmHg, and the fetal heart rate dropped to 120 bpm and became irregular. Laboratory results revealed hemoglobin 15.5 g/dL, WBC 25,130/µL, platelets 130,000/µL, and markedly elevated liver enzymes (SGOT 288.4 U/L, SGPT 175.2 U/L). Kidney function and electrolytes were normal. Urinalysis showed +4 proteinuria and negative ketonuria. The patient was diagnosed with eclampsia and HELLP syndrome accompanied by leukocytosis and elevated liver enzymes. An emergency cesarean section was performed after administration of 4 g IV $MgSO_4$, followed by a maintenance infusion of 1 g/hour and nifedipine 10 mg upon regaining consciousness. Antihypertensive management included Adalat Oros 30 mg once daily, Dopamet 250 mg three times daily, and Perdipine infusion starting at 0.5 γ. After surgery, the

patient was transferred to the ICU due to eclampsia complicated by partial HELLP syndrome. On ICU admission, she was weak and sedated, requiring mechanical ventilation (PSIMV mode) and showing signs of severe metabolic derangement. Laboratory tests demonstrated extreme leukocytosis (>40,000/µL), thrombocytopenia (89,000/µL), severe metabolic acidosis (pH 7.18; BE –16.2), and elevated lactate. Intensive supportive therapy was initiated, including antibiotics, corticosteroids, fluid transfusion, antihypertensive combination therapy, and ventilatory support.

Over the following days, the patient's condition fluctuated. On the first postoperative day, her hemoglobin dropped significantly to 5.4 g/dL, requiring PRC transfusion. Although her consciousness improved and ventilator support was briefly reduced, she remained anemic and hypertensive, with worsening liver and kidney function. By the third postoperative day, sepsis had developed, accompanied by hyperkalemia, hypocalcemia, elevated SGOT/SGPT, and increased total bilirubin levels. The patient exhibited fluid imbalance and early signs of multi-organ dysfunction syndrome (MODS). Management continued with broad-spectrum antibiotics, electrolyte correction, and optimization of parenteral nutrition.

By the fifth day, the patient's condition deteriorated further, showing clinical signs of right-sided pneumonia, acute respiratory distress syndrome (ARDS), and severe sepsis. Mechanical ventilation was re-initiated with FiO_2 100%, indicating progressive respiratory failure. Arterial blood gas analysis revealed severe metabolic acidosis (pH 6.93; HCO_3 5 mmol/L) and poor tissue perfusion. Despite maximal intensive therapy including vasopressors, diuretics, carbapenem antibiotics, and full ventilatory support the patient experienced recurrent cardiac arrest on the night of July 24, 2025. After two unsuccessful cardiopulmonary resuscitation (CPR) attempts, she was pronounced dead at 07:50 a.m. on July 25, 2025, with the final diagnosis: Partial HELLP syndrome post-cesarean section complicated by severe sepsis, ARDS, and multi-organ failure.

Discussion

HELLP syndrome, characterized by hemolysis, elevated liver enzymes, and low platelet count, represents one of the most severe complications of pregnancy. It is often associated with preeclampsia or eclampsia and poses significant maternal and fetal morbidity and mortality risks. The condition can appear either during pregnancy or postpartum, with varying degrees of severity. In this case, the patient presented with a *partial HELLP syndrome*, fulfilling only two criteria elevated liver enzymes and thrombocytopenia making diagnosis and management more challenging.[1,6] The pathogenesis of HELLP syndrome involves widespread endothelial injury, platelet activation, and microangiopathic hemolysis. These mechanisms result in hepatic ischemia, fibrin deposition, and hepatocellular necrosis, which in turn elevate liver enzymes. The associated coagulopathy and platelet consumption further contribute to organ dysfunction. In partial HELLP, these processes may be less pronounced initially but can progress rapidly, as seen in this patient's deterioration following surgery. HELLP syndrome occurs in approximately 0.5–0.9% of all pregnancies and in 10–20% of cases of severe preeclampsia.[7] Risk factors include a history of preeclampsia, multiparity, advanced maternal age, and genetic predisposition. This patient had a prior history of preeclampsia in her first pregnancy, which significantly increased her risk for recurrence. The gestational age of 33–34 weeks is consistent with the typical onset period for HELLP syndrome, which usually manifests in the late third trimester.

Partial or incomplete HELLP syndrome may present without the full triad of laboratory findings, which can delay diagnosis. The patient in this case initially presented with eclampsia and severe hypertension, accompanied by elevated transaminases and leukocytosis, without significant hemolysis. The atypical presentation required careful differentiation from other hepatic and hematologic disorders such as acute fatty liver of pregnancy, thrombotic thrombocytopenic purpura (TTP), and viral hepatitis. Leukocytosis is a relatively common physiological change during pregnancy, particularly in response to stress or corticosteroid use. However, in this case, the markedly elevated leukocyte count (>40,000/µL) exceeded normal pregnancy-related ranges and complicated the diagnostic interpretation. Severe leukocytosis may reflect a systemic inflammatory response to endothelial injury or a secondary infection, as later confirmed by sepsis. This finding underscores the

need to assess leukocytosis contextually, considering both infection and sterile inflammation as possible etiologies.

The differential diagnosis for this presentation included preeclampsia with severe features, acute fatty liver of pregnancy, and sepsis-associated hepatic dysfunction. Key laboratory findings supporting HELLP were elevated liver transaminases (SGOT 288.4 U/L, SGPT 175.2 U/L) and thrombocytopenia (platelets 89,000/µL). Absence of hemolysis markers such as elevated LDH or fragmented RBCs suggested a partial variant. The extreme leukocytosis warranted infectious screening, blood cultures, and serial monitoring to differentiate inflammatory from infectious causes. The cornerstone of HELLP syndrome management is stabilization of maternal condition and prompt delivery, as the syndrome often progresses rapidly postpartum. This patient underwent emergency cesarean section after seizure control with magnesium sulfate. Postoperatively, she required intensive monitoring, ventilatory support, and hemodynamic management. Corticosteroids were administered to improve platelet count and hepatic function, while antihypertensive therapy (nifedipine, dopamet, and perindopril infusion) was used to control severe hypertension.

Following ICU admission, the patient developed severe metabolic acidosis, renal impairment, and progressive respiratory failure, consistent with systemic endothelial injury and multiorgan dysfunction. Despite aggressive supportive therapy—including ventilatory management, fluid optimization, transfusions, and antibiotics—the patient's condition continued to deteriorate. The development of ARDS and sepsis indicated a transition from a primarily obstetric to a critical systemic condition requiring multidisciplinary coordination among obstetric, anesthesiology, and internal medicine teams.

Sepsis is a known complication in HELLP syndrome, particularly in postoperative or immunocompromised patients. The presence of pneumonia on imaging and metabolic acidosis (pH 6.93) confirmed the development of severe sepsis and ARDS. The interplay between inflammatory cytokine release and endothelial injury likely amplified the patient's respiratory failure. Despite broad-spectrum antibiotics, vasopressor support, and mechanical ventilation, the patient's condition progressed to refractory shock and cardiac arrest. Notably, HELLP syndrome can worsen after delivery, as seen in this case. The patient's laboratory abnormalities and clinical instability persisted beyond the expected postpartum recovery period. The postpartum surge in inflammatory mediators may explain the continued hepatic and renal dysfunction, as well as the exacerbation of leukocytosis and coagulopathy. This highlights the importance of extended monitoring even after delivery, as postpartum HELLP may progress to MODS and fatal outcomes.

Similar cases in the literature report that partial HELLP syndrome carries comparable mortality and morbidity risks to the complete form, especially when complicated by infection or delayed diagnosis. Studies also indicate that extreme leukocytosis in HELLP syndrome may signal underlying systemic inflammation rather than infection, though distinguishing between the two remains clinically challenging. Timely delivery, early ICU admission, and a structured multidisciplinary approach are repeatedly emphasized as critical determinants of survival. This case illustrates the complexity of managing partial HELLP syndrome complicated by leukocytosis and sepsis. It reinforces the importance of early recognition, aggressive supportive management, and continuous laboratory monitoring. The case also emphasizes the role of multidisciplinary care and postpartum vigilance in preventing progression to MODS and death. Future studies should explore biomarkers that can differentiate inflammatory leukocytosis from infection in HELLP syndrome, to optimize antibiotic stewardship and improve maternal outcomes.

CONCLUSIONS

Effective management of such cases requires prompt recognition of HELLP spectrum disorders, aggressive hemodynamic stabilization, and coordinated multidisciplinary involvement involving

obstetrics, anesthesiology, and internal medicine. Leukocytosis in this setting should be interpreted cautiously, as it may signify either an inflammatory response or an evolving infection. Early ICU admission, close laboratory monitoring, and individualized therapy are key to improving outcomes. Ultimately, this case underscores the life-threatening nature of HELLP syndrome even in its partial form and the necessity for heightened vigilance during the peripartum and postpartum periods. Strengthening antenatal surveillance, optimizing preeclampsia management, and establishing standardized critical care protocols for HELLP syndrome can significantly reduce maternal morbidity and mortality in future cases.

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