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POSTPARTUM HELLP SYNDROME AND CARDIOMYOPATHY IN A PREECLAMPTIC PRIMIGRAVIDA: A CASE REPORT WITH REVIEW OF LITERATURE

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ABSTRACT Peripartum cardiomyopathy (PPCM) is a rare and potentially fatal form of dilated cardiomyopathy that typically develops during the last month of pregnancy or up to five months postpartum. Its exact etiology remains unclear. HELLP syndrome—characterized by Hemolysis, Elevated Liver enzymes, and Low Platelet count—is a severe variant of pre-eclampsia, generally occurring before delivery, but in approximately 30% of cases, it may develop postpartum. Both PPCM and HELLP syndrome are life-threatening conditions that can overlap with pre-eclampsia and significantly increase maternal risk. We present a case of a 19-year-old primigravida at 36 weeks gestation with pre-eclampsia, who underwent emergency lower segment caesarean section (LSCS) for impending eclampsia. In the immediate postpartum period, she developed HELLP syndrome and PPCM, with pulmonary edema and severe left ventricular systolic dysfunction (LVEF <10%). Prompt diagnosis and multidisciplinary management involving obstetricians, anaesthesiologist, and cardiologist led to a successful maternal and fetal outcome. This case highlights the need for vigilance and early recognition of postpartum complications. Timely intervention is crucial in managing combined HELLP syndrome and PPCM to prevent maternal mortality and optimize outcomes, especially in young primigravidas presenting with hypertensive disorders of pregnancy.

KEYWORDS : Peripartum Cardiomyopathy, Postpartum HELLP Syndrome, Pre-eclampsia in Pregnancy, Maternal Morbidity

INTRODUCTION

HELLP syndrome is a severe variant of pre-eclampsia, first described by Weinstein in 1982, characterized by **Hemolysis, Elevated Liver enzymes, and Low Platelet count**. It complicates approximately **0.5–0.9% of all pregnancies** and is seen in **10–20% of cases with severe pre-eclampsia**[1]. This condition typically presents between 27 and 37 weeks of gestation, but around **30% of cases can occur postpartum**, often within the first 48 hours after delivery[2]. Its pathophysiology involves widespread endothelial dysfunction, abnormal placentation, and hepatic microvascular injury, posing serious risks to both mother and fetus, including disseminated intravascular coagulation (DIC), liver rupture, and acute renal failure.

Peripartum cardiomyopathy (PPCM) is a rare and potentially fatal form of **idiopathic dilated cardiomyopathy**, defined by left ventricular systolic dysfunction (LVEF <45%) occurring in the **last month of pregnancy or within five months postpartum**, in the absence of any pre-existing heart disease[3]. The condition presents with signs and symptoms of heart failure, including dyspnoea, orthopnoea, and pulmonary edema. Though the exact etiology remains unclear, proposed mechanisms include angiogenic imbalance, myocarditis, genetic predisposition, oxidative stress, and hormonal triggers such as prolactin cleavage products[4].

Recent studies have suggested that **pre-eclampsia and PPCM may represent overlapping disorders**, sharing similar vascular pathologies and endothelial injury profiles. Women with pre-eclampsia have an increased risk of developing PPCM, and when **HELLP syndrome** complicates this clinical scenario, it creates a **life-threatening postpartum triad** that is rarely reported in the literature but carries high maternal morbidity and mortality[5].

We present a unique case of a 19-year-old primigravida at 36 weeks gestation with pre-eclampsia who underwent emergency cesarean section for impending eclampsia. In the immediate postpartum period, she developed HELLP syndrome followed by **peripartum cardiomyopathy with severe left ventricular dysfunction (LVEF <10%) and pulmonary edema**. This case underscores the **importance of high clinical suspicion, timely diagnosis, and multidisciplinary management**, which proved life-saving for both the mother and the neonate.

CASE REPORT

A 19-year-old primigravida at 36 weeks of gestation presented with a history of elevated blood pressure for one week. She had been prescribed **tablet Labetalol 100 mg BID**, but BP remained uncontrolled. On her first visit to our hospital, she was admitted for further evaluation and monitoring.

She had no complaints of headache, epigastric pain, blurring of vision, or vomiting. On abdominal examination: uterus was term-sized, relaxed, cephalic presentation, and head floating. **Abdominal wall edema** was noted. Per vaginal examination revealed a **posterior, uneffaced cervix with closed os**.

All baseline investigations including complete blood count, liver and renal function tests, and coagulation profile were within normal limits. Obstetric ultrasound showed **a single live intrauterine fetus (SLIUF) of 36 weeks**, estimated fetal weight 3 kg, **AFI 11 cm**, and normal color Doppler. **2D echocardiography showed normal cardiac function with LVEF>60%.**

On the third day, the patient went into **spontaneous labour**. Per vaginal findings showed 2 cm cervical dilation, 10–20% effacement, vertex presentation, station -2, with membranes intact and show present. Six hours later, she complained of **severe epigastric pain and chest discomfort** with **BP rising to 180/110 mmHg**. Two doses of **IV labetalol (20 mg)** were given ten minutes apart along with **magnesium sulfate 4 g loading dose**. In view of impending eclampsia, she was taken for **emergency lower segment cesarean section (LSCS)** and delivered a **2.3 kg female baby**. She remained hemodynamically stable during surgery and was shifted to ICU for post-operative care.

Two hours post-LSCS, **magnesium sulfate** was continued via the **Zuspan regimen (1 g/hour IV infusion)**. However, five hours after delivery, her **BP again spiked to 180/120 mmHg** and she developed **frank hematuria**. Her **SpO_2 dropped to 75–77% on room air**, requiring oxygen via NRBM.

Three hours after LSCS, her **SpO_2 started falling** despite oxygen via NRBM. She was **intubated** and placed on ventilator (VCV mode, 100% FiO_2). **Chest X-ray** showed **bilateral infiltrates and Kerley B lines**, suggesting **pulmonary edema**. A repeat 2D echo revealed **severe left ventricular dysfunction (LVEF <10%)**, consistent with **postpartum dilated cardiomyopathy (PPCM).**

She was managed with **IV furosemide infusion (1 ml/hr)** and **dobutamine infusion (2 ml/hr)** on the advice of the cardiologist. She received **4 units of RDP and 4 units of FFP**.

Repeat investigations showed:

- Hemoglobin: 12.5 g/dL

- TLC: 21,800/mm³
- Platelets: 37,000/mm³
- SGOT: 460 U/L
- SGPT: 241 U/L
- Total bilirubin: 2.07 mg/dL
- LDH: 2853 U/L
- D-dimer: 3720
- INR: 1.5
- Peripheral smear: **Schistocytes +**

These findings were **diagnostic of HELLP syndrome**. Based on **Mississippi classification**, she was classified as **Class I HELLP Syndrome**, characterized by:

- Platelets <50,000/mm³
- AST/ALT ≥ 70 U/L
- LDH > 600 U/L

Table 1 : Mississippi Classification Of HELLP Syndrome

	Class-I (Severe)	Class-II (Moderate)	Class-III (Mild)
Platelets	≤ 50,000/microL	50,000 – 1,00,000 /microL	1,00,000 – 1,50,000/ microL
AST or ALT	≥70 IU/L	≥70 IU/L	≥40 IU/L
LDH	≥600 IU/L	≥600 IU/L	≥600 IU/L

Cardiology consultation confirmed PPCM, and treatment was continued with **dobutamine and furosemide infusion**. Given the risk of **sepsis**, antibiotics were escalated to:

- **Ceftazidime 2g + Avibactam 0.5g 8 hourly**
- **Aztreonam 1g 8 hourly**

Fluid intake was restricted to **1 liter/day**. Serial investigations showed **gradual improvement** in all biochemical parameters. After **48 hours of ventilator support**, the patient was **extubated**, and repeat 2D echo showed **improvement in LVEF to 45%**. She was subsequently shifted to the **HDU**, and eventually discharged on **postoperative day 10** in a stable condition.

DISCUSSION

HELLP syndrome and peripartum cardiomyopathy (PPCM) are individually recognized as serious obstetric complications. However, their co-occurrence, especially in the postpartum period, is exceptionally rare and presents a diagnostic and therapeutic challenge. **HELLP syndrome**-first described by Weinstein in 1982—is considered a severe form of pre-eclampsia. It involves **Hemolysis, Elevated Liver enzymes, and Low Platelet count**, and occurs in 0.5–0.9% of all pregnancies, with a strong association with hypertensive disorders.[1] While it usually occurs between 27 and 37 weeks of gestation, **approximately 30% of HELLP cases present postpartum**, often within 48 hours, as seen in our patient.[2] The **Mississippi classification** is commonly used to stratify HELLP severity; our patient met **Class I** criteria, the most severe form, with platelet counts <50,000/mm³, LDH >600 U/L, and AST/ALT >70 U/L.

Peripartum cardiomyopathy (PPCM) is a rare form of **idiopathic dilated cardiomyopathy**, presenting in the **last month of pregnancy or up to five months postpartum**, characterized by **left ventricular systolic dysfunction (LVEF <45%)**, in the absence of structural heart disease.[3] Its global incidence ranges from 1 in 1000 to 1 in 4000 live births, with higher rates reported in South Asia and Africa.[4] The pathogenesis remains unclear, but proposed mechanisms include **angiogenic imbalance (elevated soluble Flt-1), myocarditis, oxidative stress**, and cardiotoxic prolactin fragments generated by cleavage of the native hormone by cathepsin D.[5]

There is increasing recognition that **pre-eclampsia, HELLP syndrome, and PPCM share overlapping pathophysiological pathways**, particularly endothelial dysfunction, inflammation, and anti-angiogenic imbalance. Studies have shown elevated levels of **sFlt-1 and decreased levels of placental growth factor (PlGF)** in all three conditions, suggesting a shared vascular pathology.[6] This explains the occasional transition from pre-eclampsia to PPCM or the simultaneous presence of both.

In our patient, **the rapid evolution of severe pre-eclampsia into postpartum HELLP syndrome and PPCM with LVEF <10%** highlights the acute hemodynamic burden that the peripartum period can place on the cardiovascular system. This is compounded by systemic inflammation and microvascular endothelial damage-hallmarks of both HELLP and PPCM.

Diagnosis of PPCM is typically based on clinical suspicion and echocardiographic confirmation of reduced ejection fraction, as in our case. **Chest radiograph**, showing pulmonary edema with Kerley B lines, and **echocardiography** are critical tools. However, due to overlapping symptoms such as dyspnoea and chest discomfort-which can also be seen in HELLP, pre-eclampsia, and pulmonary embolism-a high index of suspicion is required.

Management of HELLP syndrome involves **delivery as definitive treatment**, especially in severe or worsening cases. Our patient required **emergency LSCS for impending eclampsia**, which was both therapeutic for HELLP and vital for fetal survival. **PPCM management** includes **diuretics, vasodilators, inotropes (like dobutamine), and fluid restriction**. In our case, the use of **magnesium sulfate**, **IV labetalol**, **dobutamine infusion**, and **ventilatory support**, along with **RDP and FFP transfusions**, was crucial in stabilizing the patient.

Given her deteriorating oxygenation and signs of **sepsis**, **broad-spectrum antibiotics** were initiated early. Infectious triggers are known to exacerbate PPCM and HELLP-related hepatic dysfunction, and thus early antimicrobial coverage is essential.[7]

Our patient had a complicated postpartum course, but **with timely multidisciplinary intervention**, including obstetricians, cardiologists, intensivists, and anesthesiologists, she **survived and recovered fully**. This case reiterates the importance of **early recognition**, **vigilant postpartum monitoring**, and **coordinated care** in high-risk pregnancies.

CONCLUSION

Postpartum HELLP syndrome and peripartum cardiomyopathy (PPCM) are both individually associated with significant maternal morbidity and potential mortality. Their simultaneous occurrence, though rare, can lead to rapid clinical deterioration, particularly in the immediate postpartum period. The overlapping symptomatology can delay diagnosis, thereby increasing the risk of poor outcomes.

In this case, a young primigravida with pre-eclampsia developed both postpartum HELLP syndrome and PPCM with severely reduced ejection fraction, requiring mechanical ventilation, inotropic support, and intensive care. However, prompt recognition and timely intervention by a multidisciplinary team, including obstetricians, intensivists, and cardiologists, proved to be life-saving.

Postpartum HELLP syndrome and PPCM can individually present significant complications, and when combined, may lead to considerable maternal morbidity and even mortality. Therefore, timely detection, aggressive supportive care, and multidisciplinary team management-especially involving the intensivist and cardiologist-are crucial to ensure the best possible outcomes for affected patients.

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