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Case Report

## Partial HELLP syndrome: case report

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### ABSTRACT

HELLP syndrome is a complication in pregnancy clustered by haemolysis, elevated liver enzymes, and a low platelet count. It is seen as a serious complication of preeclampsia and eclampsia. Serious manifestations like haemorrhage, infarction, rupture and other hepatic manifestations are usually associated with it. In this case study, 29 years old primigravida is a booked case admitted in ward at 39 weeks 1 day with decreased fetal movement for 2 days. No history of pain abdomen, bleeding per vaginum, discharge per vaginum. Her blood pressure records at the time of admission was 110/72 mmHg and she was normotensive throughout pregnancy. Urine routine examination was negative for urinary protein. However, blood tests showed platelet count of 66,1000/cumm, with ALT of 174 U/L and AST of 123 U/L on peripheral blood film. RBC were predominantly normocytic, normochromic with few macrocytes. WBC has normal morphology. Platelets were reduced on smear. Giant platelets were seen. Ursodeoxycholic acid 300 mg 12 hourly were given to the patient and 3 doses of vitamin K I/M 24 hourly. She was delivered by cesarean section which was performed due to failure of progression of labor with a deflexed head. There was presence of retroplacental clot of 4×3 cm indicating placental abruption, a complication of HELLP syndrome. From this we conclude that we should be careful in suspecting complications of full blown diseases even when the patients are asymptomatic but have atypical laboratory findings.

**Keywords:** Abruption, Low liver enzymes, Partial HELLP, Thrombocytopenia

### INTRODUCTION

HELLP syndrome is a complication in pregnancy clustered by haemolysis with a microangiopathic blood smear, elevated liver enzymes, and a low platelet count.<sup>1</sup> It is seen as a serious complication of preeclampsia and eclampsia.<sup>2</sup> Serious manifestations like haemorrhage, infarction, rupture and other hepatic manifestations are usually associated with HELLP syndrome and severe preeclampsia. A mother with a previous history of HELLP or preeclampsia is at major risk for developing HELLP syndrome.

A variety of genetic variants plays a significant role in increasing risk of HELLP syndrome which have been reported in due course of research, but have no role in

clinical management. There is a hypothesis that the complement cascade is a key mediator in systemic inflammatory disorder like severe preeclampsia/HELLP and it has been observed that women with complement regulatory protein mutations appear to be at increased risk of severe preeclampsia and HELLP syndrome. Fetal long-chain 3-hydroxyacyl CoA dehydrogenase (LCHAD) deficiency appears to be related to the underlying etiology.<sup>3,4</sup> Whereas, nulliparity is not a risk factor for HELLP syndrome in contrast to preeclampsia, as most of the affected patients are multiparous.<sup>5,6</sup>

### CASE REPORT

In this case study, 29 years old primigravida is a booked case admitted in ward at 39 weeks 1 day with decreased

fetal movement for 2 days. No History of pain abdomen, bleeding per vaginum, discharge per vaginum, burning micturition, easy fatigability, headache, blurring of vision, epigastric pain, decreased urine output. Ultrasound scans in the first and second trimester were corresponding to the period of gestation. Her routine antenatal investigations and the blood pressure recordings were normal throughout pregnancy. No maternal risk factors were found. Her past history and family history were insignificant.

**Table 1: HELLP syndrome.**

HELLP syndrome	
	PBF with schistocytes and burr cells.
<b>H-hemolysis</b>	Increased Serum bilirubin $\geq 1.2$ mg/dL (20.52 micromol). Severe anemia, unrelated to blood loss (haemorrhage). Low serum haptoglobin or lactate dehydrogenase (LDH) $\geq 2$ times the upper level of normal (based on laboratory-specific reference ranges).
	<b>EL-elevated liver enzymes</b>
	Aspartate aminotransferase (AST) or alanine aminotransferase (ALT) $\geq 2$ times the upper level of normal
<b>LP-low platelet count</b>	<100,000 cells/microL (mild to severe thrombocytopenia)

**Table 2: Investigations.**

Investigations	On admission	Day after operation
<b>Urine routine for protein</b>	Negative	Negative
<b>Platelet count</b>	66,1000/cumm	1,0,5000/cumm
<b>Liver Function Test</b>	ALT of 174 U/L and AST of 123 U/L	ALT of 161 U/L and AST of 95 U/L
	Bilirubin (T) - 0.20 mg/dl Bilirubin (D) - 0.30 mg/dl	Bilirubin (T) - 0.20 mg/dl Bilirubin (D) - 0.30 mg/dl
<b>Peripheral blood film</b>	RBC: Predominantly normocytic normochromic with few macrocytes WBC: Normal morphology Platelets: Reduced on smear, giant platelets were seen	-
<b>LDH</b>	201 IU/l	



**Figure 1: Retroplacental clot of size 5x3 cm suggesting placental abruption.**

Her blood pressure records at the time of admission was 110/72 mmHg. Urine routine examination was negative for urinary protein. However, blood tests showed platelet count of 66,1000/cumm, with ALT of 174 U/L and AST of 123 U/L on peripheral blood film, RBC were predominantly normocytic normochromic with few macrocytes, WBC had normal morphology, platelets were reduced on smear, giant platelets were seen. Repeat blood tests showed a decrease in platelet count of 60,1000/cumm.

Ursodeoxycholic acid 300 mg 12 hourly were given to the patient and 3 doses of vitamin K I/M 24 hourly. After three doses of dinoprostone gel 0.5 mg instilled through intracervical route, patient went into labour and delivery by cesarean section was performed due to failure of progression of labor with deflexed head, baby delivered as vertex presentation with clear liquor with birth weight 3.5 kg and Apgar score of 9-10 and 9-10 (1-5 min), respectively. There was presence of retroplacental clot of 5x3 cm indicating placental abruption, a complication of HELLP syndrome but the patient's vitals were stable throughout the operation, otherwise placenta and cord both were normal. 2 units FFPs given intraoperatively.

Postoperative period remained uneventful, there was no postpartum haemorrhage or puerperal sepsis. On postoperative day 1 patient's blood samples showed platelet count of 105x1000/cumm, with ALT of 161 U/L and AST of 95 U/L. Patient was discharged on post op day 5 in satisfactory condition with the baby and the baby remained motherside throughout this time.

**DISCUSSION**

As our patient was asymptomatic but had atypical features of HELLP syndrome at the time of delivery at term, with typical absence of raised blood pressure and Hemolysis. Hemolysis is otherwise an essential part of the HELLP syndrome (“H”-hemolysis). Whereas, raised liver enzymes, resulting from the liver involvement (“EL”-

elevated liver enzymes) 3-4 times higher than upper limit, in combination with thrombocytopenia ("LP"- low platelet).

HELLP syndrome is a serious condition that may result in a series of adverse maternal and neonatal consequences.<sup>7,8</sup> Similar cases have been reported showing atypical cases of HELLP syndrome. Up till now, HELLP syndrome is considered as one of the most serious forms of preeclampsia, not as a separate disorder.

## CONCLUSION

We reached the diagnosis of partial HELLP on the basis of the laboratory findings, despite a normal blood pressure and lack of hemolysis with an absence of typical symptoms but presence of concealed retroplacental clot indicating placental abruption, a complication of HELLP syndrome. From this we conclude that we should be careful in suspecting complications of full blown diseases even when the patients are asymptomatic but have atypical laboratory findings.

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