European Journal of Clinical and Biomedical Sciences
2021; 7(6): 155-160
http://www.sciencepublishinggroup.com/j/ejcbs
doi: 10.11648/j.ejcbs.20210706.20
ISSN: 2575-4998 (Print); ISSN: 2575-5005 (Online)

Case Report

HELLP Syndrome with Unusual Features in a Rare Setting

Collins Amadi[1, 2, 4, *], **Bright Amadi**[1, 3, 4]

[1]Department of Chemical Pathology, Rivers State University, Port Harcourt, Nigeria

[2]Department of Chemical Pathology, Pamo University of Medical Sciences, Port Harcourt, Nigeria

[3]Department of Chemical Pathology, University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria

[4]Green Care Medical Consultant's Hospital, Rumuigbo, Port Harcourt, Nigeria

Email address:

collins338@yahoo.com (C. Amadi)

[*]Corresponding author

To cite this article:

Collins Amadi, Bright Amadi. HELLP Syndrome with Unusual Features in a Rare Setting. *European Journal of Clinical and Biomedical Sciences*. Vol. 7, No. 6, 2021, pp. 155-160. doi: 10.11648/j.ejcbs.20210706.20

Received: November 23, 2021; **Accepted**: December 10, 2021; **Published**: December 24, 2021

Abstract: HELLP (Hemolysis, Elevated Liver enzymes, and Low Platelet) syndrome is a rare disorder exclusively associated with pregnancy. It occurs mostly during the antepartum period in about 70% of cases but could also occur during the post-partum period in about 30% of reported cases especially during the first 48 hours following delivery. The syndrome is mostly reported among multiparous Caucasians in association with severe preeclampsia, hypertension, and proteinuria. It is rarely reported within Nigeria and the post-partum variant has never been documented among women of Nigerian origin in the literature to date. Herein, we report a very unusual case of HELLP syndrome in a very rare setting that developed eleven days post-partum without the usual features of hypertension and proteinuria in a 26-year-old G1P1. Upon presentation, she was promptly diagnosed and aggressively managed using standard protocols by a multidisciplinary medical team of specialists. She developed acute kidney injury (KDIGO stage 1) in the course of management of which she required conservative management. Remarkably, with supportive care, all the clinical and laboratory derangements, including hepatic and renal functions, normalized after seven days on admission. Her condition remained stable during weekly follow-up visits for four consecutive weeks. This case and its management protocols reinforce the need for a high index of clinical suspicion for this dreaded disorder and its prompt diagnosis/aggressive management even in very rare circumstances and settings.

Keywords: HELLP, HELLP Syndrome, Class 3 HELLP Syndrome

1. Introduction

The HELLP, the acronym used to describe a constellation of Hemolysis, Elevated Liver enzymes, and Low Platelet syndrome, is an adverse complication at the severe end of hypertensive disorders of pregnancy [1-3]. t occurs in 0.5-0.9% of all pregnancies but at a higher rate when it is associated with severe preeclampsia (10-20%) [4]. The syndrome in its complete form manifests with all the three aforementioned features or partial when it present with only two of these features in the presence of severe preeclampsia [5, 6]. It occurs mostly before delivery (70%) but has been reported to occur during the post-partum period (30%) [4-6].

To date, its etiology has remained elusive despite decade-long research since it was first described in the literature [6]. However, several theories have been proposed on its etiologic background; theories similar to those proposed for preeclampsia, prompting other experts to believe it is a variant or complication of severe preeclampsia [7]. However, it evolves at times (10-20%) without clinical/laboratory features of severe preeclampsia (hypertension/proteinuria) which indicate it could well be a unique disease entity [5-8].

The syndrome is highly prevalent among multiparous Caucasians but rarely reported among women of the Negroid race despite the high rate of its acclaimed precursor pregnancy-related hypertensive conditions (preeclampsia eclampsia) within the Negroid race [9, 10].

Herein, we report a rare case of HELLP syndrome which

presented on the 11th day post-partum without any pre-existing history, clinical and laboratory features of any of the hypertensive disorders of pregnancy.

2. Case Report

A 26-year-old G1P1 was rushed into the emergency room of a privately owned specialist hospital (Green Care Medical Consultants' Hospital, Port Harcourt, Nigeria) on the 19th of August, 2021 on account of severe/excruciating epigastric pain of four hours duration prior to presentation. The pain was colicky, constant, radiates to the lower back, and worsens with meals and on slight movement. The pain had started suddenly while she was lying down and had continued to progressively worsen since onset. She had given birth to a live baby boy (birth weight: 3.1kg) eleven days before presentation in the same privately owned facility where she and her baby were discharged in perfect clinical conditions.

Her pregnancy was initially supervised in a public tertiary hospital (University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria) from 12 weeks gestational age till term (37 weeks) but had to revert to the privately owned facility because of the ongoing industrial action (strike) embarked upon by Medical Doctors in the public sector in Nigeria as that time. Her antenatal period in both hospitals was uneventful/inconspicuous till she had a spontaneous vaginal delivery at 39 weeks. She had no history of hypertension/proteinuria during the antenatal or the delivery period, nor 48 hours after delivery till she was discharged. Past medical history revealed that she had no pre-existing or current liver or blood disorders nor history of ingestion of acetaminophen/non-steroidal anti-inflammatory drugs to toxic levels, sepsis, thrombotic thrombocytopenic purpura, acute fatty liver disease, or abdominal trauma.

On presentation, she was found to be acutely ill-looking, mildly febrile (37.9°C), mildly pale (+), not cyanosed nor edematous but mildly icteric, and overweight (weight: 82kg; height: 1.7m; body mass index: 27.7kg/m^2). She had a blood pressure of 120/80mmHg, pulse rate of 92 beats/minute, and respiratory rate of 24 cycles/minute. Physical examination findings were unremarkable besides the severe (3+) epigastric and right upper quadrant tenderness elicited.

She was immediately admitted into the female medical ward and stabilized. A provisional diagnosis of acute gastritis to rule out acute hepato-biliary disease was initially made. But all medications offered her did not alter her clinical status which warranted a request for further laboratory investigations. The results of those initial urgent laboratory investigations requested are summarized in Table 1 below.

Following the review of the results of the initial investigations, a rare case of class 3 HELLP syndrome using the Mississippi triple class guidelines was made and aggressive multidisciplinary management protocols were instituted to stem the progression of the HELLP syndrome. Following the diagnosis of HELLP syndrome, she was moved into the intensive care unit (ICU).

All the medical consultants including the Obstetrician and Gynecologist, Cardiologist, Intensivist, Anesthetist, Chemical Pathologist, Hematologist, Microbiologist, Gastroenterologist, Nephrologist, and Pulmonologist in the private health facility were assembled. Intravenous fluids (IV), IV dexamethasone 10mg every 12 hours, prophylactic IV Magnesium Sulphate ($MgSO_4$) to prevent seizure, and antipyretics were commenced. Close monitoring of vital signs, the passage of a wide-bore IV cannula, retention of an indwelling urinary catheter for urine input/output evaluation were also instituted.

She remained clinically stable except for moderate/intermittent fever and epigastric pain until day 2 when she became mildly hypertensive (140/80mmHg), developed acute kidney injury (AKI) in association with diminished estimated glomerular filtration rate (eGFR), raised creatinine/urea levels, proteinuria (2+), worsening hyperuricemia, and oliguria while still on adequate IV fluids (Shown in Table 2 below). The AKI was categorized as AKI stage 1 based on the KDIGO (Kidney disease: Improving Global Outcome) recommendations.

The liver parameters worsened on day 2 evidenced by rising ALT/AST activities (Table 2). The hemolytic process seemed to have also worsened on day 2 evidenced by rising LDH activities, total bilirubin, reduced PCV and haptoglobin levels and increased schizocytes/burr cells on peripheral blood smear (Table 2). The platelet count continued to reduce by day 2 but other coagulation parameters remained normal.

Following the development of AKI and hypertension, IV furosemide and IV fluids, anti-hypertensive (labetalol 100 mg thrice daily), and other management protocols were intensified including arrangement for possible blood transfusions. Additionally, the prophylactic IV MgSO4 was temporarily withdrawn as a precaution because of the incident AKI. The plasma magnesium level test ordered on day 2 was found to be normal (Table 2).

On day 3, all the clinical, laboratory, and radiological parameters started to normalize except for worsening anemia for which she received two units of whole blood (Table 2). She continued on her remarkable progress through day 4 when all evaluated clinical and laboratory parameters had normalized (Table 2). On day 5, she remained stable and the only investigation done on day 5 were hemoglobin (Hb) level, PCV, and abdominal ultrasound scan. The Hb level was 10.5g/dL, the PCV value improved to 32%, and her abdominal scan results remained normal (not tabulated).

Upon review by the management team, some of her medications were either stopped or the dosage reduced or tapered off slowly over the following 5th to 7th day on admission. At this point, the management team advised that she should be transferred back into the female medical ward. By the 7th day on admission, she was finally reviewed by the multidisciplinary medical team and certified fit for discharge in good clinical condition but to continue weekly visits for at least four weeks. The summary of her clinical, laboratory and radiological parameters upon discharge including those of the weekly follow-up visits are shown in Table 3.

Table 1. *Summary of various variables obtained at various stages before and at diagnosis of the class 3 HELLP syndrome.*

Variables	4m booking to delivery	Discharge day following delivery	At presentation 9 days after discharge
Blood pressure, mmHg	110/80 – 100/80	110/70	Normal (120/80)
Fetal profiles	Normal	Normal	ND
Blood film for MP	Negative	Negative	Negative
Widal test for enteric fever	ND	ND	Negative
Serology for Hepatitis B/C	Negative	ND	Negative
Serology for Helicobacter Pylori	ND	ND	Negative
E/U/Cr	Normal	Normal	Cr: 75umol/L eGFR: 98 mls/min Others: Normal
Total plasma bilirubin, umol/L	Normal	Normal	62 umol/L (elevated)
Total plasma protein, g/L	ND	ND	Normal
Plasma albumin, g/L	ND	ND	34g/L (Low)
Plasma uric acid level, umol/L	ND	ND	0.9mmo/L (elevated)
Liver enzymes, U/L	Normal	ND	ALT: 140U/L (high) AST: 120U/L (high) LDH: 640U/L (high) Others: Normal
Full blood count/differentials	PCV: 34% - 35% Others: Normal	PCV: 32% Others: Normal	PCV: 29% (Low) PLT: 140 x 10^9cell/L Blood smear: schizocytes + Blood smear: Burr cells + Others: Normal
Prothrombin time, s (10-14)	ND	ND	11 (normal)
Fibrinogen, g/L (2.0-4.0)	ND	ND	2.7 (normal)
D-dimer, ug/L (≤500)	ND	ND	400 (normal)
Haptoglobin, g/L (0.4 – 1.0)	ND	ND	0.5g/L (normal)
Urinalysis/urine MCS	Normal	Normal	Normal findings
24-hour urine volume, mls/day	Normal	Normal	ND
Abdomino-pelvic USS	Normal	Normal	Normal findings

MP: malaria parasite; ND: test not done on that day; E/U/Cr: plasma electrolytes/urea/creatinine; ALT: alanine aminotransferase; AST: aspartate aminotransferase; LDH: lactate dehydrogenase; PCV: parked cell volume;, PLT: platelet count; MCS: microscopy, culture and sensitivity; USS: ultrasound scan

Table 2. *Summary of various variables obtained on different days (day 1 to 4) during management.*

Investigations	At presentation Day 1	Day 2	Day 3	Day 4
Blood pressure, mmHg	Normal (120/80)	140/80 (High)	100/80 (normal)	100/70 (normal)
E/U/Cr	Normal	K^+:5.0 mmol/L (high) U: 6.7 mmol/L (high) Cr: 110umol/L (high) eGFR: 63 mls/min (low) Others:, Normal	K^+:4.2 (normal) U: 4.5 (normal) Cr: 89 (normal) eGFR: 81mls/min Others: Normal	K^+:4.0 (normal) U: 3.9 (normal) Cr: 70 (normal) eGFR: 106mls/min (normal) Others: Normal
RPG, mmol/l	4.0 mmol/L	4.2 mmol/l	4.4 mmol/L	4.3 mmo/L
Total plasma bilirubin, umol/L	62umol/L (high)	86umol/L (high)	57umol/L (reducing)	15umol/l (normal)
Total plasma protein, g/L	Normal	Normal	Normal	Normal
Plasma albumin, g/L	34g/L (low)	30g/L (low)	33g/L	37g/L (normal)
Plasma uric acid level, umol/L	0.9mmo/L (hgh)	1.2mmol/L (rising)	0.8 mmol/L (reducing)	0.40mmol/l (Normal)
Liver enzymes, U/L	ALT: 140U/L (high) AST: 120U/L (high) LDH: 640U/L (high) Others: Normal	ALT: 198 (rising) AST: 160 (rising) LDH: 1100 (rising) Others: Normal	ALT: 130 (reducing) AST: 110 (reducing) LDH: 700 (reducing) Others: Normal	ALT: 40 (normal) AST: 20 (normal) LDH: 260 (normal) Others: Normal
Full blood count/differentials	PCV: 29% (Low) PLT: 140 x 10^9cell/L Blood smear: schistocytes + Blood smear: Burr cells + WBC:7x10^9/L (normal) Others: Normal	PCV: 27% (reducing) PLT: 110 x 10^9cell/L Blood smear: schistocytes ++ Blood smear: Burr cells ++ WBC:8x10^9//L (normal) Others: Normal	PCV: 24% (reducing) PLT:140x10^9cell/L Blood smear: schistocytes + Blood smear: Burr cells negative WBC:8x10^9 (normal) Others: Normal	ND; differed to day 5 PLT:160x10^9cell/L (normal) Blood smear: schistocytes negative Blood smear: Burr cells negative
Prothrombin time, s	11s (normal)	12 (normal)	11 (normal)	Normal
Fibrinogen, g/L	2.7g/L (normal)	2.0 (normal)	2.5 (normal)	Normal
D-dimer, ug/L	400ug/L (normal)	460ug/L (normal)	400ug/L (normal)	Normal
Haptoglobin, g/L	0.5g/L (normal)	0.3g/L (low)	0.6g/L (normal)	Normal
Urinalysis	Normal findings	Proteinuria ++	Proteinuria +	Proteinuria – (normal)
24-hour urine volume, mls/day	ND	400mls/day (oliguria)	1300mls/day	2100mls/day (normal)

Investigations	At presentation Day 1	Day 2	Day 3	Day 4
Plasma magnesium	ND	Normal	ND	ND
Abdominal USS	Normal findings	ND	Normal findings	ND

ND: test not done on that day; E/U/Cr: plasma electrolytes/urea/creatinine; RPG: random plasma glucose; ALT: alanine aminotransferase; AST: aspartate aminotransferase; LDH: lactate dehydrogenase; PCV: parked cell volume;, PLT: platelet count; WBC: white cell count: USS: ultrasound scan

Table 3. *Summary of various variables obtained on the day of discharge (admission day 7) and during weekly follow-up visits.*

Investigations	Discharge day Admission day 7	Follow-up Week 1	Follow-up Week 2	Follow-up Week 3	Follow-up Week 4
Blood pressure, mmHg	120/80 (normal)	110/80	120/70	110/80	110/80
E/U/Cr including eGFR	Normal findings	Normal	Normal	Normal	Normal
RPG, mmol/L	4.4 mmol/L	ND	ND	ND	4.1mmo/L
Total plasma bilirubin, umol/L	12umol/L (normal)	10umol/L	11umol/L	10umol/L	9umol/L
Total plasma protein, g/L	Normal	Normal	ND	ND	Normal
Plasma albumin, g/L	38g/L (normal)	38g/L	ND	ND	Normal
Plasma uric acid level, umol/L	0.4mmo/L (normal)	0.3mmol/L	ND	ND	0.2umol/L (normal)
Liver enzymes, U/L	ALT: 30U/L (normal)	ALT: 25U/L	ALT:20	ALT: 15	ALT: 15U/L (normal)
	AST: 20U/L (normal)	AST: 15U/L	AST: 20	AST: 15	AST: 15U/L
	LDH: 150U/L (normal)	LDH: 160U/L	LDH: 160	LDH: 150	LDH: 140U/L
	Others: Normal	Others: ND	Others: ND	Others: ND	Others: Normal
Full blood count/differentials	Hb: 11g/dL (norml)	ND	ND	ND	Hb: 12g/L (normal)
	PCV: 37% (normal)	PCV: 38%	PCV: 38%	39%	39% (normal)
	PLT: 170 x 10^9/L	180 x 10^9/L	200 x 10^9/L	230 x 10^9/L	270 x 10^9/L
	Others: Normal	Others: Normal	Others: ND	Others: ND	Others: Normal
Prothrombin time, s	12s (normal)	ND	ND	ND	10s (normal)
Fibrinogen, g/L	2.8g/L (normal)	ND	ND	ND	2.9g/L (normal)
D-dimer, ug/L	200ug/L (normal)	ND	ND	ND	170ug/L (normal)
Haptoglobin, g/L	0.7g/L (Normal)	ND	ND	ND	0.7g/L (normal)
Urinalysis	Normal findings	Normal	Normal	Normal	Normal findings
24-hour urine volume, mls/day	2200 mls/day (normal)	ND	ND	ND	ND
Abdominal USS	Normal (normal)	ND	ND	ND	Normal findings

ND: test not done on that week; E/U/Cr: plasma electrolytes/urea/creatinine; RPG: random plasma glucose; ALT: alanine aminotransferase; AST: aspartate aminotransferase; LDH: lactate dehydrogenase; PCV: parked cell volume;, PLT: platelet count; USS: ultrasound scan

3. Discussion

3.1. HELLP Syndrome

HELLP syndrome is the acronym used to describe a constellation of Hemolysis, Elevated Liver enzymes, and Low Platelet syndrome [1-3]. The syndrome is an adverse complication at the severe end of hypertensive disorders of pregnancy [2, 3]. It occurs in 0.5-0.9% of all pregnancies but at a higher rate when it is associated with severe preeclampsia (10-20%) [4].

3.2. Etiology/Pathophysiology

Several researchers identify HELLP syndrome as a variant of severe preeclampsia, but the relationship between these two disorders remains controversial and unproven [7]. Unlike the usual clinical features of preeclampsia, about 30% of women with HELLP syndrome will not present with hypertension or proteinuria, and preeclampsia does not at times manifest before the clinical presentation of HELLP syndrome, as in the index case [5, 6]. The exact pathophysiology of HELLP syndrome remains ill-defined, but evidence from several studies supports the involvement of several theories [7]. Risk factors include multiparty, European descent/Caucasian background, advanced maternal age older than 34 years, and poor pregnancy outcomes, which are all characteristics at variance with the index case [2, 11].

About 70% of the cases are diagnosed during the antenatal period, mostly in the third trimester [4-6]. It is very rare for the clinical features of HELLP syndrome to manifest after 48 hours from delivery, but have been reported to occur more than 10 days post-partum in previous reports, just as it is observed in the index case [12]. The cardinal pathological alterations in HELLP syndrome include vasospasm, endothelial injury, and vascular fibrin deposition with platelet aggregation and consumption [4, 13]. The coagulation distortions lead to hepatic sinusoidal obstruction, intrahepatic vascular congestion with end-organ ischemia, and the resulting hepatic necrosis may occasionally cause hepatic failure, hematoma, or rupture which were not noticed in the index case [4, 13].

Hepatic rupture can occur if intrahepatic pressures do exceed that which the Glisson's capsule can resist [14]. Stretching of the Glisson's capsule has been adduced for the frequent epigastric/right upper quadrant pain/tenderness reported among cases of HELLP syndrome as observed in the index case [14, 15].

3.3. Diagnosis

The diagnosis of HELLP syndrome may be challenging as patients present with ill-defined clinical features that mimic other-like conditions [4, 6, 10]. The diagnosis became more

complicated in situations when classic hypertension and proteinuria are virtually absent as reported in the index case. The clinical features of HELLP syndrome are sometimes mistaken for gastritis, respiratory infection, other hepatobiliary diseases, pyelonephritis, and acute pancreatitis [6]. Same misdiagnosis was made during the initial evaluation of the index case, but that was immediately re-evaluated following the review of the initial laboratory results. The diagnostic criteria for HELLP syndrome are based on a series of laboratory results indicating microangiopathic hemolytic anemia, thrombocytopenia, and hepatic ischemia/necrosis. Currently, there are two major definitions in use in clinical practice: the Mississippi and the Tennessee guidelines [16].

3.4. Investigations

A constellation of very recent delivery, epigastric/right upper quadrant abdominal pain, laboratory features of microangiopathic hemolytic anemia, progressive thrombocytopenia, unexplained deranged liver function test parameters, and some features overlapping preeclampsia strongly indicate post-partum HELLP syndrome [6]. Other medical conditions follow difficult deliveries that may complicate laboratory parameters related to HELLP syndromes such as hemorrhagic shock, organ dysfunction, and coagulopathies, these other conditions must be ruled out using clinical and laboratory protocols [6]. However, the index case had none of these complicating conditions.

The absence of evidence of microangiopathic hemolytic anemia (rising LDH activities, elevated total plasma bilirubin, and presence of schistocytes/burr cells on blood smear) in a post-partum woman is an indicator to seek alternative diagnoses. The index case had all laboratory features of microangiopathic hemolytic anemia which enhanced the prompt diagnosis of her condition [6, 17]. It is essential to also exclude other non-obstetric imitators of post-partum HELLP syndrome during investigation using history, physical examinations, laboratory parameters, and radiological examinations [6, 17]. All these protocols were also carried out on the index case.

It is also essential that investigations are tailored towards complications in post-partum HELLP syndrome [6, 17]. When compared to the antepartum HELLP syndrome, patients who develop post-partum HELLP syndrome have a high tendency of developing acute kidney injury (AKI), pulmonary edema (PE), disseminated intravascular coagulation (DIC), and subscapular liver hematoma [18-20]. These complications heighten the morbidity and mortality in cases of post-partum HELLP syndrome [18-20]. The index case had AKI which was diagnosed and managed promptly but not the other complications. The aggressive management protocols instituted may have presented the emergence of these other life-threatening complications.

3.5. Treatment

The cornerstone of treatment of post-partum HELLP syndrome includes IV fluid replacement, IV dexamethasone 10mg every 12 hours known to control the inflammatory process and accelerate recovery especially in class 1 cases as observed in the index case, antihypertensive for blood pressure control, and prophylactic IV MgSO4 infusion for seizure prophylaxis or control [19-21]. However, other treatment modalities should be dictated by any other complications (AKI, DIC, pulmonary edema, sub-capsular liver hematoma, anemia, etc) that may ensue during the HELLP syndrome evolution or treatment. The index case had AKI and anemia which she received IV fluids, IV furosemide, and blood transfusions [19, 20].

4. Conclusion

We had reported a very unusual case of HELLP syndrome in a very rare setting that developed eleven days post-partum without the usual features of hypertension and proteinuria in a 26-year-old GIP1. Upon presentation, she was promptly diagnosed and aggressively managed using standard protocols by a multidisciplinary medical team of specialists. She developed acute kidney injury in the course of management of which she required conservative management. Remarkably, with supportive care, all the clinical and laboratory derangements, including hepatic and renal functions, normalized after seven days on admission. Her condition remained stable during weekly follow-up visits for four consecutive weeks. This case and its management protocols reinforce the need for a high index of clinical suspicion for this dreaded disorder including prompt diagnosis and aggressive management even in very rare and usual circumstances and settings.

Consent

Written informed consent was obtained from the patient and it is available for review on reasonable request

Conflicts of Interest Statement

The authors declare that they have no competing interests.

Acknowledgements

The authors are very pleased to express the deepest gratitude to the Chief Medical Director of Green Care Consultants Hospital located in the city of Port Harcourt, Nigeria for his unconditional support and guidance in the preparation and completion of this article.

References

[1] Folk DM. Hypertensive disorders of pregnancy: overview and current recommendations. J midwifery women's health. 2018; 63 (3): 289-300.

[2] Poimenidi E, Metodiev Y, Archer NN, Jackson R, Bangash MN, Howells PA. Haemolysis, elevated liver enzymes and low platelets: Diagnosis and management in critical care. J Intensive Care Society. 2021; 0 (0): 1–7. DOI: 10.1177/17511437211025410.

[3] Burwick RM, Moyle K, Java A, Gupta M. Differentiating Hemolysis, Elevated Liver Enzymes, and Low Platelet Count Syndrome and Atypical Hemolytic Uremic Syndrome in the Postpartum Period. Hypertension. 2021; 78 (3): 760-8.

[4] Westbrook RH, Dusheiko G, Williamson C. Pregnancy and liver disease. J Hepatol 2016; 64: 933–45.

[5] Rimaitis K, Grauslyte L, Zavackiene A, Baliuliene V, Nadisauskiene R, Macas A. Diagnosis of HELLP syndrome: a 10-year survey in a perinatology centre. Int J Environ Res Public Health. 2019; 16 (1): 109. DOI: 10.3390/ijerph16010109.

[6] Dusse LM, Alpoim PN, Silva JT, Rios DR, Brandão AH, Cabral AC. Revisiting HELLP syndrome. Clinica Chimica Acta. 2015; 451: 117-20.

[7] McCormick PA, Higgins M, McCormick CA, Nolan N, Docherty JR. Hepatic infarction, hematoma, and rupture in HELLP syndrome: support for a vasospastic hypothesis. J Matern Fetal Neonatal Med. 2021 15: 1-6. DOI: 10.1080/14767058.2021.1939299.

[8] Shelat PM, Vyas RC, Shah SR, Nathwani ND. Fetomaternal outcome in pregnancy with HELLP syndrome. Int Reprod Contracept Obstet Gynecol. 2020; 9 (7): 2860-6.

[9] Socolov D, Ilea C, Lupascu IA, Blidaru I, Luca A, Radescu M, et al. Maternal-fetal prognosis in HELLP syndrome in a level 3 maternal-fetal care centre. Clin Exp Obstet Gynecol. 2021; 43 (3): 374-8.

[10] Fallatah HI, Al-Dabbagh AA, Mimish HL, Al-Sahafi MA, Akbar HO. Liver diseases in pregnancy and outcomes: A retrospective study from Saudi Arabia. Afri J Reprod Health. 2021; 25 (3): 121-9.

[11] Socolov D, Ilea C, Lupascu IA, Blidaru I, Luca A, Radescu M, Carauleanu A, Socolov R. Maternal-fetal prognosis in HELLP syndrome in a level 3 maternal-fetal care centre. Clin Exp Obstetrics Gynecol. 2021; 43 (3): 374-8.

[12] Nakamura K, Inokuchi R, Sonoo T, Hiruma T, Tokunaga K, Doi K. Late postpartum HELLP syndrome over 10 days after delivery. Am J Emerg Med. 2016; 34 (11): 2258-e1.

[13] Booker WA. Hypertensive Disorders of Pregnancy. Clin Perinatol. 2020; 47 (4): 817-33.

[14] MN S. A Maternal Mortality in the Case of HELLP Syndrome: An Avertable Loss. Indian J Forensic Med Toxicol. 2019; 13 (4).

[15] Nam IC, Won JH, Kim S, Bae K, Jeon KN, Moon JI, Cho E, Park JE, Jang JY, Park SE. Transcatheter Arterial Embolization for Spontaneous Hepatic Rupture Associated with HELLP Syndrome: A Case Report. Medicina. 2021; 57 (10): 1055.

[16] Sasamori Y, Tanaka A, Ayabe T. Liver disease in pregnancy. Hepatol Res. 2020: 50 (9): 1015-23.

[17] Wallace K, Harris S, Addison A, Bean C. HELLP syndrome: pathophysiology and current therapies. Curr Pharm Biotech. 2018; 19 (10): 816-26.

[18] Praveen C, Kaul A, Sharma RK. Acute kidney injury in pregnancy. Clin Queries: Nephrology. 2016; 5 (1): 8-15.

[19] Shah FA, Guez G, Patel N, Patel BB. Complicated Post-Partum HELLP Syndrome Causing Acute Renal Failure and a Spontaneous Acute Subdural Hematoma. Cureus. 2021; 13 (2): e13233. DOI: 10.7759/cureus.13233.

[20] Szczepanski J, Griffin A, Novotny S, Wallace K. Acute Kidney Injury in Pregnancies Complicated With Preeclampsia or HELLP Syndrome. Front Med (Lausanne). 2020; 7: 22. DOI: 10.3389/fmed.2020.00022.

[21] Yalcin OT, Sener T, Hassa H, Ozalp S, Okur A: Effects of postpartum corticosteroids in patients with HELLP syndrome. Int J Gynaecol Obstet 1998, 61: 141-148.