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Review Article

Obstetrics and Gynaecology Section

Clinical Features, Pathophysiology, and Management of HELLP Syndrome: A Narrative Review

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ABSTRACT

Haemolysis, Elevated Liver Enzymes, and Low Platelets (HELLP), collectively referred to as HELLP syndrome, is a serious complication of Pre-Eclampsia (PE) that can occur anytime after the 28th week of pregnancy or postpartum. Common symptoms include upper right abdominal pain, nausea, and generalized weakness, which can delay diagnosis. Early identification of HELLP syndrome is critical to prevent severe maternal and foetal complications, such as liver rupture, Disseminated Intravascular Coagulation (DIC), and placental abruption. Placental dysfunction, systemic inflammation, and endothelial injury contribute to the development of this syndrome. Recent studies also suggest that antiangiogenic molecules and long non-coding Ribonucleic Acids (RNAs) may play a role in its pathophysiology. Platelet counts are monitored to classify HELLP syndrome as complete or partial, and additional classification is based on the Mississippi and Tennessee criteria. The differential diagnosis includes Acute Fatty Liver of Pregnancy (AFLP), thrombotic thrombocytopenic purpura, and haemolytic uraemic syndrome, each requiring a distinct management approach. Prompt delivery is the mainstay of treatment, often supplemented with corticosteroids to promote foetal lung maturity, along with management of maternal hypertension and seizures. Emerging therapies, such as plasma exchange and eculizumab, may be considered when conventional measures fail. Notably, HELLP syndrome without hypertension requires careful monitoring due to the need for rapid diagnosis and intervention. The purpose of the present review is to provide an overview of the clinical manifestations, pathophysiology, diagnostic challenges, and newer treatment strategies for HELLP syndrome, aiming to improve maternal and foetal outcomes through timely detection and intervention.

Keywords: Antiangiogenic molecules, Corticosteroids, Differential diagnosis, Endothelial injury, Haemolysis elevated liver enzymes and low platelets, Thrombocytopenia

INTRODUCTION

Pre-Eclampsia (PE) is a multisystem disorder of pregnancy, characterised by new-onset hypertension and proteinuria or organ dysfunction after 20 weeks of gestation in women without pre-existing hypertension [1]. HELLP syndrome is a severe complication of PE that typically occurs in the third trimester or postpartum period [1,2] and is associated with a heightened risk of adverse maternal and foetal outcomes [3]. HELLP syndrome occurs in approximately 0.2-0.8% of all pregnancies, with PE present in 70-80% of cases [2,3]. Early diagnosis and timely management are essential, as HELLP syndrome can rapidly progress to life-threatening complications, including liver rupture (1.8%), DIC (21%), and placental abruption (16%) [4,5]. The present review provides a comprehensive overview of the clinical features, diagnostic criteria, pathophysiology, classification, complications, differential diagnosis, and treatment approaches for HELLP syndrome. It also highlights emerging molecular insights and novel therapeutic strategies that may guide future management.

Clinical Features and Diagnostic Criteria of HELLP Syndrome

Clinical features and diagnostic criteria of HELLP syndrome are summarised in [Table/Fig-1] [6-10].

Category	Details
Definition	HELLP stands for Haemolysis, Elevated Liver Enzymes, and Low Platelet count; a severe form of PE [6].
Timing	Typically occurs in the third trimester or postpartum [6].
Key clinical features	- Right upper quadrant or epigastric pain (due to liver involvement) - Nausea and vomiting - Headache - Fatigue - Visual disturbances - High blood pressure and proteinuria (not always present) [7,8]
Misleading symptoms	May mimic gastroenteritis or viral hepatitis, delaying diagnosis [9]
Haemolysis-related signs [6]	- Fatigue - Pale skin
Liver Involvement [6,9].	- Elevated liver enzymes - Jaundice - Rarely, liver rupture
Thrombocytopenia effects [10].	Increased risk of bleeding, especially during delivery
Diagnostic criteria [10].	- Haemolysis: Elevated LDH, presence of schistocytes - Liver Enzymes: Elevated AST and ALT - Platelets: <100,000/mm^3

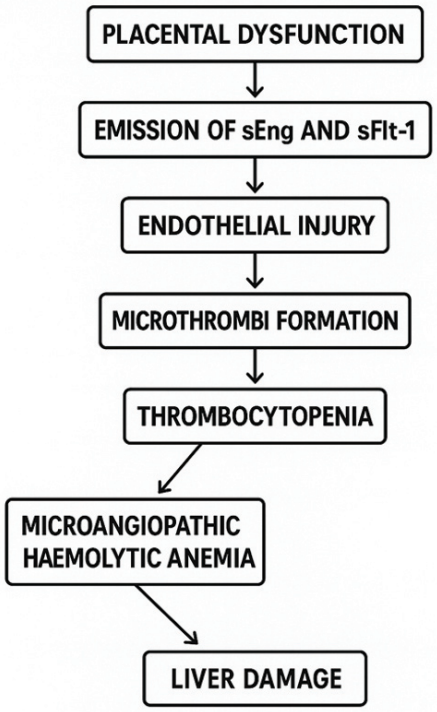
[Table/Fig-1]: Clinical features and diagnostic criteria of HELLP syndrome [6-10].
LDH: Lactate dehydrogenase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase

Pathophysiology and Emerging Molecular Insights in HELLP Syndrome

The pathophysiology of HELLP syndrome involves a complex interplay of placental dysfunction, systemic inflammation, and endothelial injury [8,11]. Inadequate remodeling of the spiral arteries in the placenta can result in hypoxia and the release of antiangiogenic factors, including Soluble Endoglin (sEng) and Soluble Fms-like Tyrosine kinase-1 (sFlt-1) [11-13]. These factors compromise the endothelial walls, increasing vascular permeability and promoting vascular injury [12,14].

Platelets adhere to the injured endothelium, forming microthrombi, which reduces circulating platelet levels and leads to thrombocytopenia [14]. As red blood cells pass through the narrowed and damaged small blood vessels, they become fragmented, resulting in microangiopathic haemolytic anaemia, a

process also observed in conditions such as sepsis [15]. Since the liver has a rich blood supply, it is often affected early, resulting in elevated liver enzymes, and, in severe cases, hepatocellular injury or liver cell death [9,16]. This combination of factors contributes to the characteristic symptoms seen in HELLP syndrome. Certain Long Non-Coding RNAs (lncRNAs) may influence inflammation and cell growth in the placenta, leading to vascular dysfunction and abnormal placental positioning [17]. The pathophysiology of HELLP syndrome is depicted in [Table/Fig-2].



[Table/Fig-2]: Pathophysiology of HELLP syndrome.

Classification and Severity Grading of HELLP Syndrome

HELLP syndrome is classified into two types: complete and partial, depending on the presence of its defining features-haemolysis, elevated liver enzymes, and thrombocytopenia [18]. Complete HELLP syndrome is diagnosed when all three criteria are present, whereas partial HELLP syndrome presents with only one or two of these features [18]. Additionally, HELLP syndrome can be classified using the Mississippi and Tennessee criteria [19,20]. A summary of HELLP syndrome classification is shown in [Table/Fig-3] [6,18-21]. Maternal and neonatal complications associated with HELLP syndrome are described in [Table/Fig-4] [4,6,7,9,18-21].

Category	Details / Criteria	Maternal Complications	Foetal Complications
Types of HELLP [18]	Complete HELLP: All 3 features (haemolysis, ↑ liver enzymes, ↓ platelets) Partial HELLP: 1-2 features present [18].	Complete HELLP → higher risk of hepatic rupture, DIC, MODS, placental abruption, haemorrhage due to thrombocytopenia, jaundice [18,21].	Both types → IUGR, preterm birth, hypoxia, intrauterine foetal demise (partial HELLP usually less severe) [6,18].
Mississippi classification (20).	Platelet-based: - Class I: <50,000/µL - Class II: 50,000-100,000/µL - Class III: 100,000-150,000/µL [20].	Class I patients are most prone to hepatic rupture, DIC, and maternal death (mortality 1-30%) [18,20]	Higher risk of IUGR, hypoxia, and preterm birth with lower maternal platelet counts [20]
Tennessee classification [19].	Lab-based: - LDH >600 U/L - AST ≥70 U/L - Platelets <100,000/µL [19].	Identifies patients with significant hepatic injury and coagulopathy, prone to haemorrhage and MODS [18,19].	Correlates with increased preterm delivery and perinatal mortality [6,19].

[Table/Fig-3]: Classification of HELLP syndrome summarising diagnostic criteria, maternal and foetal complications, and clinical outcome correlations [6,18-21].
MODS: Multiple organ dysfunction syndrome; IUGR: Intrauterine growth restriction; LDH: Lactate dehydrogenase; AST: Aspartate aminotransferase

Category	Complications
Maternal complications	
Haematologic	Haemolysis → fatigue, pallor; thrombocytopenia → bleeding tendency (especially during delivery); Disseminated Intravascular Coagulation (DIC), more frequent in complete HELLP [18-20].
Hepatic	Elevated liver enzymes, jaundice, hepatic rupture (rare 1-1.8%, but highest maternal mortality), liver necrosis [7,9].
Cardiologic	Multiple Organ Dysfunction Syndrome (MODS), particularly in complete HELLP [19].
Pulmonary	Pulmonary oedema, acute respiratory distress secondary to DIC/MODS [21].
Renal	Acute kidney injury, oliguria/anuria due to microangiopathy and endothelial dysfunction [21].
Neurological	Seizures, cerebral oedema, intracranial haemorrhage, hypertensive encephalopathy [4,21].
Maternal mortality	Ranges from 1% to 30%, depending on severity and complications [18].
Neonatal complications	
Growth-related	Intrauterine Growth Restriction (IUGR) due to placental insufficiency [6].
Prematurity-related	Preterm birth (spontaneous or iatrogenic due to maternal status) [4,6].
Hematologic	Foetal hypoxia resulting from reduced placental perfusion [6].
Mortality	Intrauterine foetal demise in severe cases [6].

[Table/Fig-4]: Maternal and neonatal complications of HELLP syndrome [4,6,7,9,18-21].

Differential Diagnosis of HELLP Syndrome

Accurate differentiation of HELLP syndrome from similar pregnancy-related conditions is essential, as management strategies differ. Both AFLP and HELLP syndrome typically present in the third trimester with nausea, vomiting, abdominal pain, and elevated liver enzymes [22]. According to the Swansea criteria, AFLP diagnosis emphasises symptoms such as hypoglycaemia and encephalopathy, which are not prominent in HELLP syndrome [22,23]. Lower antithrombin activity (<65%) generally suggests AFLP and should be considered in the diagnostic process [22,24]. Differential diagnoses and distinguishing clinical features are summarised in [Table/Fig-5] [22,23,25,26].

Treatment Approaches and Emerging Therapies in HELLP Syndrome

The management of HELLP syndrome focuses on early diagnosis, maternal stabilisation, and a multidisciplinary approach involving obstetrics, anaesthesiology, haematology, and intensive care [27]. Supportive measures include close haemodynamic monitoring, careful fluid management, transfusion therapy, and targeted organ-specific interventions [5,27]. The only definitive treatment after maternal or foetal compromise is delivery [27].

The timing of delivery is critical and depends on gestational age and maternal-foetal stability. For pregnancies beyond 34 weeks, expedited delivery is recommended due to high maternal risk and favourable neonatal outcomes [28,29]. Before 34 weeks, short-term expectant management may be considered to allow administration of corticosteroids for foetal lung maturation, but only in stable patients under tertiary care [29]. A meta-analysis of late-preterm PE comparing immediate delivery to expectant management showed that immediate delivery resulted in fewer maternal complications {1.1% vs. 3.1%, Relative Risk (RR) 0.36, 95% Confidence Interval (CI) 0.15-0.87} but higher rates of neonatal respiratory distress syndrome (20.9% vs. 17.1%, RR 1.22, 95% CI 1.01-1.47) [29].

Antihypertensive therapy is crucial in managing severe hypertension and reducing the risk of maternal cerebrovascular complications [30]. First-line agents include intravenous labetalol, hydralazine, and oral nifedipine, titrated to maintain systolic blood pressure <160 mmHg and diastolic blood pressure <110 mmHg, while preserving uteroplacental perfusion [30]. Seizure prophylaxis with magnesium

Condition	Similarities with HELLP Syndrome	Distinguishing Features	Diagnostic criteria / markers	Management
Acute Fatty Liver of Pregnancy (AFLP) [22,23].	Nausea, vomiting, abdominal pain, elevated liver enzymes	Hypoglycaemia, hyperammonemia, coagulopathy	Swansea criteria: hypoglycaemia, encephalopathy; antithrombin activity <65%	Supportive care; delivery if indicated
Thrombotic Thrombocytopenic Purpura (TTP) [25]	Microangiopathic haemolytic anaemia, thrombocytopenia	Neurological symptoms, markedly decreased	ADAMTS13 deficiency	Plasma exchange
Haemolytic Uraemic Syndrome (HUS) [25,26]	Microangiopathic haemolytic anemia, thrombocytopenia	Predominant renal involvement; often post-infectious	Kidney dysfunction; recent GI infection	Plasma exchange, complement inhibitors
HELLP syndrome [26]	—	Hypertension, proteinuria, liver damage in pregnancy	Elevated liver enzymes, haemolysis, low platelets	Prompt delivery

[Table/Fig-5]: Differential diagnosis of HELLP syndrome and distinguishing clinical features [22,23,25,26].

sulfate is also essential [31]. Duley L et al., demonstrated that magnesium sulfate, compared to placebo or diazepam, reduces the risk of eclampsia by more than half (RR 0.41, 95% CI 0.29-0.58) and lowers maternal mortality (RR 0.54, 95% CI 0.26-1.10) [31].

The use of corticosteroids in HELLP syndrome is controversial. Corticosteroids reduce systemic inflammation and stabilise endothelial cells, leading to temporary improvements in platelet count and liver enzymes [32,33]. However, a meta-analysis of randomised trials (n=485) showed no statistically significant reduction in maternal mortality (RR 0.95, 95% CI 0.28-3.25) or maternal morbidity (RR 1.36, 95% CI 0.45-4.10) [33]. Similarly, a pooled review of seven Randomised Controlled Trial (RCTs) (n≈485) found no significant improvements in maternal morbidity (RR 1.36, 95% CI 0.45-4.10) or incidence of eclampsia (RR 1.16, 95% CI 0.76-1.77) [32]. While corticosteroids improve laboratory indices, their primary clinical benefit is in promoting foetal lung maturation before preterm birth rather than improving maternal outcomes [33].

Blood product support in patients with HELLP syndrome is individualised [25]. Platelet transfusion is used to maintain platelet counts above 50,000/µL in patients requiring cesarean delivery or major surgery. It can also be considered for patients with platelet counts below 20,000-30,000/µL or those with active bleeding [27,34]. When coagulopathy or haemorrhage is present, fresh frozen plasma, cryoprecipitate, and red blood cells are administered according to standard obstetric transfusion protocols [34].

In patients unresponsive to standard management, some adjunctive therapies have been investigated [28,35]. Therapeutic Plasma Exchange (TPE) has been associated with improved outcomes in severe postpartum HELLP syndrome or cases overlapping with Thrombotic Microangiopathy (TMA), although evidence is limited to case series [35]. TPE is considered when there is no clinical improvement within 24 hours of delivery despite maximal supportive care (Class-I HELLP, progressive organ failure, or strong suspicion of TMA) [36]. Early initiation (within 24 hours) is generally linked to better laboratory and clinical outcomes [35,36].

Eculizumab, a complement inhibitor, has been used in HELLP associated with atypical haemolytic uraemic syndrome. Its use is reserved for highly selected cases after multidisciplinary discussion, ideally guided by genetic testing, and requires prompt meningococcal vaccination. The extremely high cost and potential need for prolonged therapy should also be considered [37]. Continuous Renal Replacement Therapy (CRRT), sometimes combined with Inhaled Nitric Oxide (iNO) in critically ill patients with respiratory failure, has been reported as an experimental approach to improve oxygenation. It serves as a temporising measure, with improvements in oxygenation often observed within 24-48 hours [38].

Certain lncRNAs, such as LINC-HELLP (Long intergenic non-protein coding RNA associated with HELLP syndrome), have been identified in affected families and regulate invasion by extravillous trophoblasts. Mutations in these lncRNAs can disrupt placental development and increase the risk of HELLP syndrome [39]. Other dysregulated lncRNAs in HELLP and PE include Nuclear Paraspeckle Assembly Transcript 1 (NEAT1), which promotes inflammation via Nuclear Factor kappa-light-chain-enhancer of activated B cells (NF-κB) signaling, and Metastasis Associated Lung Adenocarcinoma Transcript 1 (MALAT1), which regulates trophoblast migration and endothelial function [17]. Antisense oligonucleotides or Small interfering RNA (siRNAs) targeting pathogenic lncRNAs show promise as experimental therapies, and lncRNA profiling may aid in early diagnosis [17]. However, no clinical trials have yet been conducted, and available data are limited to in-vitro studies and small patient groups, so their use remains investigational [17,39].

Atypical HELLP Syndrome without Hypertension

Although HELLP syndrome is generally considered a severe form of PE, 10-20% of cases occur without hypertension, which can complicate diagnosis and management [40,41]. Normal blood pressure and urinary protein levels may delay recognition, increasing the risk of serious complications such as liver rupture, DIC, and kidney failure [28,41]. Systemic endothelial dysfunction, platelet activation, and microangiopathic haemolysis are not necessarily dependent on hypertension [42]. Therefore, any pregnant or postpartum woman presenting with elevated liver enzymes, low platelet counts, and signs of haemolysis should be closely monitored, even in the absence of hypertension, to prevent diagnostic and treatment delays [5,42].

CONCLUSION(S)

Prompt diagnosis and management of HELLP syndrome are essential, as the condition can be life-threatening for both mothers and their infants. While hypertension is commonly associated with PE, cases without this symptom should not be underestimated. A deeper understanding of the complex pathophysiological mechanisms, including the roles of antiangiogenic factors and lncRNAs, can facilitate faster diagnosis and more individualised treatment. Further research is needed to elucidate the molecular mechanisms underlying HELLP syndrome and to develop biomarkers and therapeutic strategies to improve maternal and foetal outcomes.

REFERENCES

[1] Chappell LC, Cluver CA, Kingdom J, Tong S. Pre-eclampsia. Lancet. 2021;398(10297):341-54.
[2] Adorno M, Maher-Griffiths C, Grush Abadie HR. HELLP Syndrome. Crit Care Nurs Clin North Am. 2022;34(3):277-88.
[3] Petca A, Miron BC, Pacu I, Dumitrașcu MC, Mehedințu C, Șandru F, et al. HELLP Syndrome-Holistic Insight into Pathophysiology. Medicina (Kaunas). 2022;58(2):326.
[4] Gasem T, Al Jama FE, Burshaid S, Rahman J, Al Suleiman SA, Rahman MS. Maternal and fetal outcome of pregnancy complicated by HELLP syndrome. J Matern Fetal Neonatal Med. 2009;22(12):1140-43.
[5] Dusse LM, Alpoim PN, Silva JT, Rios DRA, Brandão AH, Cabral ACV. Revisiting HELLP syndrome. Clin Chim Acta. 2015;451(Pt B):117-20.
[6] Zhang X, Meng HB, Cao RY, Cai LY. HELLP syndrome: A case report and literature review. Zhonghua Xue Ye Xue Za Zhi. 2024;45(S1):117-19.
[7] Pécourt M. Highlights on HELLP syndrome. J Med Vasc. 2021;46(5-6):207-08.
[8] Sandvoß M, Potthast AB, von Versen-Höynck F, Das AM. HELLP syndrome. Reprod Sci. 2017;24(4):568-74.
[9] Nghiemová HY, Mojhová M, Zikán M. Severe hepatic rupture in HELLP syndrome. Ceska Gynekol. 2023;88(2):100-05.
[10] Perronne L, Dohan A, Bazeries P, Guerrache Y, Fohlen A, Rousset P, et al. Hepatic involvement in HELLP syndrome: An update with emphasis on imaging features. Abdom Imaging. 2015;40(7):2839-49.

[11] Vigil-De Gracia P. HELLP syndrome. Ginecol Obstet Mex. 2015;83(1):48-57.
[12] Iannaccone A, Reisch B, Mavarani L, Darkwah Oppong M, Kimmig R, Mach P, et al. Soluble endoglin versus sFlt-1/PlGF ratio: Detection of preeclampsia, HELLP syndrome, and FGR in a high-risk cohort. Hypertens Pregnancy. 2022;41(3-4):159-72.
[13] Gardikioti A, Venou TM, Gavriilaki E, Vetsiou E, Mavrikou I, Dinas K, et al. Molecular Advances in Preeclampsia and HELLP Syndrome. Int J Mol Sci. 2022;23(7):3851.
[14] Erez O, Romero R, Jung E, Chaemsaithong P, Bosco M, Suksai M, et al. Preeclampsia and eclampsia: The conceptual evolution of a syndrome. Am J Obstet Gynecol. 2022;226(2S):S786-S803.
[15] Gupta M, Feinberg BB, Burwick RM. Thrombotic microangiopathies of pregnancy: Differential diagnosis. Pregnancy Hypertens. 2018;12:29-34.
[16] von Salmuth V, van der Heiden Y, Bekkers I, van Runnard Heimel P, Spaanderman MA, Peeters LL, et al. The role of hepatic sinusoidal obstruction in the pathogenesis of the hepatic involvement in HELLP syndrome: Exploring the literature. Pregnancy Hypertens. 2020;19:37-43.
[17] Amirlatifi S, Kooshari Z, Salmani K, Fallah Ziyarani M, Azizi S, Ghotbi E, et al. Evaluation of long noncoding RNA (LncRNA) in pathogenesis of HELLP syndrome: Diagnostic and future approach. J Obstet Gynaecol. 2023;43(1):2174836.
[18] Jenkins J, Ferozuddin AA, Mourad J, Abdulla ZZ, Oviedo A. Incongruence of Hemolysis, Elevated Liver Enzyme, Low-Platelet Count Syndrome (HELLP) and preeclampsia criteria in pregnancy: Implications for medical education and obstetrics training. Cureus. 2024;16(8):e67211.
[19] Li BY, Yang HX. Comparison of clinical features in antepartum and postpartum HELLP syndrome. Zhonghua Fu Chan Ke Za Zhi. 2022;57(12):907-13.
[20] Martin JN Jr, Brewer JM, Wallace K, Sunesara I, Canizaro A, Blake PG, et al. Hellp syndrome and composite major maternal morbidity: Importance of Mississippi classification system. J Matern-Fetal Neonatal Med. 2013;26(12):1201-06.
[21] Wang YQ, Wang J, Ye RH, Zhao YY. Investigation of diagnosis and treatment of hemolysis-elevated liver enzymes-low platelet counts (HELLP) syndrome: Clinical analysis of 59 cases. Chin Med J (Engl). 2010;123(10):1273-77.
[22] Byrne JJ, Seasely A, Nelson DB, Mcintire DD, Cunningham FG. Comparing acute fatty liver of pregnancy from hemolysis, elevated liver enzymes, and low platelets syndrome. J Matern-Fetal Neonatal Med. 2022;35(7):1352-62.
[23] Goel A, Jamwal KD, Ramachandran A, Balasubramanian KA, Eapen CE. Pregnancy-related liver disorders. J Clin Exp Hepatol. 2013;4(2):151-62.
[24] Minakami H, Morikawa M, Yamada T, Yamada T, Akaishi R, Nishida R. Differentiation of acute fatty liver of pregnancy from syndrome of hemolysis, elevated liver enzymes and low platelet counts. J Obstet Gynaecol Res. 2014;40(3):641-49.
[25] Giannubilo SR, Marzioni D, Tossetta G, Ciavattini A. HELLP syndrome and differential diagnosis with other thrombotic microangiopathies in pregnancy. Diagn Basel Switz. 2024;14(4):352.
[26] Kappler S, Ronan-Bentle S, Graham A. Thrombotic Microangiopathies (TTP, HUS, HELLP). Hematol Oncol Clin North Am. 2017;31(6):1081-103.
[27] Beucher G, Simonet T, Dreyfus M. Management of the HELLP syndrome. Gynecol Obstet Fertil. 2008;36(12):1175-90.
[28] Haram K, Svendsen E, Abildgaard U. The HELLP syndrome: Clinical issues and management. A Review. BMC Pregnancy Childbirth. 2009;9:8.
[29] Chatzakis C, Liberis A, Zavlanos A, Petousis S, Tsakmaki E, Dinas K, et al. Early delivery or expectant management for late preterm preeclampsia: A meta-analysis of randomized controlled trials. Acta Obstet Gynecol Scand. 2021;100(8):1392-400.
[30] Jain R, Jogi SR. Effectiveness and safety of intravenous labetalol in severe pre-eclampsia and eclampsia at a teaching institution in Chhattisgarh. J Fam Med Prim Care. 2024;13(9):3788-91.
[31] Duley L, Gülmezoglu AM, Henderson-Smart DJ, Chou D. Magnesium sulphate and other anticonvulsants for women with pre-eclampsia. Cochrane Database Syst Rev. 2010;2010(11):CD000025.
[32] Sun WJ, Hu J, Zhang Q, Shan JM. Administration of corticosteroid therapy for HELLP syndrome in pregnant women: Evidences from seven randomized controlled trials. Hypertens Pregnancy. 2023;42(1):2276726.
[33] Kasem AF, Alqenawy HB, Elgendi MA, Ali RR, Ahmed RH, Sorour MN, et al. Corticosteroids for improving patient-relevant outcomes in HELLP syndrome: A systematic review and meta-analysis. BMC Pregnancy Childbirth. 2024;24(1):487.
[34] Kirkpatrick CA. The HELLP syndrome. Acta Clin Belg. 2010;65(2):91-97.
[35] Chowdhry M, Agrawal S, Gajulapalli SP, Thakur UK. Therapeutic plasma exchange in HELLP syndrome: A life savior. Asian J Transfus Sci. 2022;16(1):106-10.
[36] Erkurt MA, Sarici A, Kuku I, Berber I, Kaya E, Bicim S, et al. The effect of therapeutic plasma exchange on management of HELLP Syndrome: The report of 47 patients. Transfus Apher Sci. 2021;60(5):103248.
[37] Lokki AI, Haapio M, Heikkinen-Eloranta J. Eculizumab treatment for postpartum HELLP syndrome and aHUS-case report. Front Immunol. 2020;11:548.
[38] Azimi-Ghomi O, Miller G, Guida C, Marimon A, Boneva D, McKenney M. HELLP! The novel use of continuous renal replacement therapy and nitric oxide in the treatment of acute respiratory failure in HELLP syndrome. Case Rep Crit Care. 2021;2021:8895319.
[39] van Dijk M, Thulluru HK, Mulders J, Michel OJ, Poutsma A, Windhorst S, et al. HELLP babies link a novel lincRNA to the trophoblast cell cycle. J Clin Invest. 2012;122(11):4003-11.
[40] El-Agwany AS, Abdelsadek AA. A rare case of normotensive HELLP syndrome complicated with massive ascites: Spontaneous resolution. Egypt J Anaesth. 2016;32(1):155-58.
[41] Sibai BM. Diagnosis, controversies, and management of the syndrome of hemolysis, elevated liver enzymes, and low platelet count. Obstet Gynecol. 2004;103(5 Pt 1):981-91.
[42] Abildgaard U, Heimdal K. Pathogenesis of the syndrome of hemolysis, elevated liver enzymes, and low platelet count (HELLP): A review. Eur J Obstet Gynecol Reprod Biol. 2013;166(2):117-23.

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