Clinical and Experimental Obstetrics & Gynecology

Clin. Exp. Obstet. Gynecol. **2025**; 52(3): 33370
https://doi.org/10.31083/CEOG33370

Original Research

Comparison of Clinical Characteristics and Pregnancy Outcomes in Partial and Complete HELLP Syndrome: An Eight-Year Retrospective Study From a Tertiary Hospital in China

Hao Gu[1,†], Jiaying Chen[1,†], Hongqin Wu[1], Minhui Jiang[1], Ying Gu[2], Yaling Feng[1,*]

[1]Department of Women Health Care, Jiangnan University Affiliated Wuxi Maternity and Child Health Care Hospital, 214002 Wuxi, Jiangsu, China
[2]Department of Obstetrics, Jiangnan University Affiliated Wuxi Maternity and Child Health Care Hospital, 214002 Wuxi, Jiangsu, China
*Correspondence: 13600182648@163.com (Yaling Feng)
[†]These authors contributed equally.

Academic Editor: George Daskalakis
Submitted: 27 November 2024 Revised: 16 January 2025 Accepted: 13 February 2025 Published: 17 March 2025

Abstract

Background: Hemolysis, elevated liver enzymes, and low platelet count syndrome (HELLP syndrome), characterized by hemolysis (H), elevated liver enzymes (EL), and a low platelet count (LP), is a severe obstetric complication. We analyzed the clinical characteristics of complete and partial HELLP syndrome. **Methods**: We conducted a retrospective study to collect data on 96 pregnant women with preeclampsia accompanied by HELLP syndrome. HELLP syndrome was diagnosed based on the Tennessee Classification System. General characteristics, clinical manifestations, laboratory results, complications, as well as maternal and neonatal outcomes were analyzed to compare complete and partial HELLP syndrome. **Results**: Among the 96 pregnant women with HELLP syndrome, 76% (73/96) were diagnosed with partial HELLP syndrome, while 24% (23/96) were diagnosed with complete HELLP syndrome. No statistically significant differences were found in maternal and disease characteristics between the partial and complete HELLP groups (all $p > 0.05$). The main symptoms of HELLP syndrome were headache and epigastric pain. Regarding diagnostic measures, the complete HELLP group had lower platelet counts (PLT) and higher total bilirubin (TBil), lactate dehydrogenase (LDH), alanine transaminase (ALT), and aspartate transaminase (AST) levels compared to the partial HELLP group. For non-diagnostic measures, the complete HELLP group showed higher white blood cell counts and D-dimer levels. No statistically significant differences were observed in the remaining laboratory indexes (all $p > 0.05$). Similarly, there was no statistically significant differences in the incidence of maternal pregnancy complications and fetal demographic features between the two groups (all $p > 0.05$). **Conclusions**: The distinction between partial and complete HELLP syndromes primarily lies in specific laboratory indexes. Both syndromes can lead to severe perinatal complications, including eclampsia, uteroplacental apoplexy, and fetal demise. Clinical diagnosis does not require strict adherence to all three criteria: H, EL, and LP. Special attention should be given to patients with partial HELLP syndrome, who require immediate treatment and intervention.

Keywords: complete HELLP syndrome; partial HELLP syndrome; preeclampsia; perinatal outcomes; clinical features

1. Introduction

Hemolysis, elevated liver enzymes, and low platelet count syndrome (HELLP syndrome), first named by Weinstein [1] in 1982, is a severe complication of preeclampsia characterized by hemolysis (H), elevated liver enzymes (EL), and low platelet count (LP). The incidence of HELLP syndrome is approximately 0.2%–0.8% [2]. It is a multisystemic disease with complications including liver and kidney insufficiency, thrombosis, eclampsia, and postpartum hemorrhage. In addition to these maternal complications, HELLP syndrome can result in preterm birth, fetal growth restriction, and placental abruption, significantly affecting fetal health. The maternal mortality rate is approximately 9.7%, with neonatal mortality reaching 37% [3]. Thus, HELLP syndrome poses a grave threat to the lives of both mothers and infants.

The specific diagnostic laboratory values for HELLP syndrome vary across countries and regions [4]. However, many physicians assess disease severity based on three main criteria. Complete HELLP syndrome is defined by the presence of all three criteria, whereas partial HELLP syndrome is diagnosed when one or two criteria are met [5]. Aydin *et al.* [6] suggested that complete and partial HELLP syndromes may represent a continuum in the natural progression of the disease. The debate continues regarding whether partial and complete HELLP syndromes constitute a continuum of the same condition or represent distinct conditions. The pathogenesis of HELLP syndrome remains unclear. Enhancing awareness of this condition is crucial for reducing maternal and infant mortality. This article reviews the clinical features of partial and complete HELLP syndromes to strengthen understanding and management of the condition and improve perinatal outcomes.

2. Materials and Methods

2.1 Study Design and Diagnostic Criteria for HELLP Syndrome

Ninety-six patients with HELLP syndrome were retrospectively enrolled from January 2016 to December 2023. HELLP syndrome was diagnosed using the Tennessee Classification System, which is globally recognized. This system includes the following laboratory findings: (1) hemolysis, defined by abnormal peripheral smear, elevated bilirubin ($\geq$20.5 µmol/L), and increased lactate dehydrogenase (LDH $\geq$600 U/L); (2) elevated liver enzymes, defined as increased aspartate aminotransferase (AST $\geq$70 U/L) and elevated LDH; and (3) low platelet count (PLT $<100 \times 10^9$/L) [5]. Complete HELLP syndrome was diagnosed when all three criteria were met, while partial HELLP syndrome was diagnosed when one or two criteria were met. Exclusion criteria included: (1) patients with conditions such as thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, acute fatty liver of pregnancy, or other diseases causing hemolysis, abnormal liver function, or platelet reduction; and (2) patients transferred to other hospitals without pregnancy termination at our facility or who had delivered before admission. Patient enrollment is shown in Fig. 1. Ultimately, 96 patients met the inclusion criteria. This study adhered to the Declaration of Helsinki and was approved by the Ethics Committee of Wuxi Maternal and Child Health Hospital (No. 2024-06-0507-14). All participants provided informed consent.

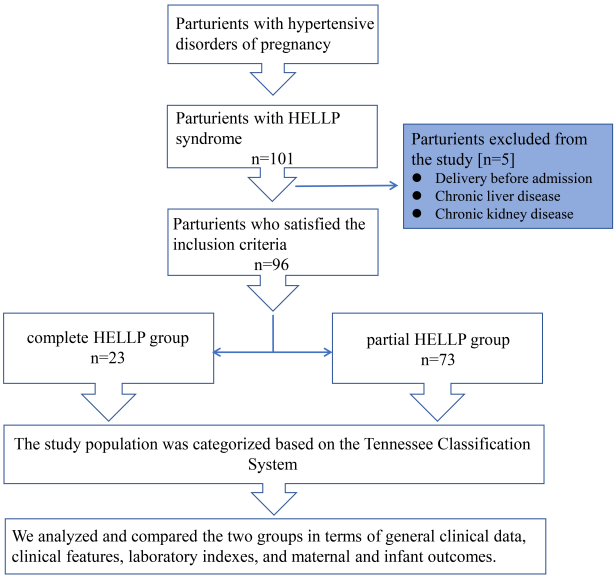

Fig. 1. Schematic representation of the study population selection. HELLP, hemolysis, elevated liver enzymes, and low platelet count syndrome.

2.2 Data Collection

Clinical indicators collected included gestational age, body mass index (BMI) at admission, gravidity, primiparity, family history of hypertension, and history of cesarean section. Disease course and comorbidities included gestational age at delivery, systolic and diastolic blood pressure at admission, mean arterial pressure at admission, and gestational diabetes mellitus. Neonatal outcomes included assisted reproductive rate, preterm birth rate, stillbirth rate, and Apgar scores. Laboratory indicators corresponding to the most severe period of hospitalization included alanine transaminase (ALT), AST, LDH, total bilirubin (TBil), platelet count, hemoglobin, white blood cell count, and plasma fibrinogen. Maternal perinatal outcomes included eclampsia, placental abruption, and renal function impairment.

2.3 Statistical Analysis

IBM SPSS Statistics for Windows, version 27.0 (IBM Corp., Armonk, NY, USA) was used for statistical analyses. The Shapiro-Wilk test was applied to test for normality. Differences between groups were assessed using the independent sample *t*-test for normally distributed continuous variables, the Mann-Whitney U test for non-normally distributed continuous variables, and the Chi-square test for categorical variables. Fisher's exact test was applied for categorical variables with an expected frequency of less than 5. Statistical significance was set at $p < 0.05$. Normally distributed continuous variables are presented as mean $\pm$ standard deviation (SD), while non-normally distributed variables are expressed as median (25th–75th percentile) [median (P25–P75)]. Categorical variables are presented as percentages.

3. Results

3.1 Comparison of General Clinical Data between the Two Groups

Among the 96 patients with HELLP syndrome, 73 were in the partial HELLP group (76%) and 23 were in the complete HELLP group (24%). The ages of the partial HELLP group and the complete HELLP group were similar (30.3 *vs.* 31.8), though the difference was not statistically significant ($p = 0.267$). Among the 96 pregnant women, there were seven twin pregnancies, but no triplet or higher-order pregnancies. Six of these pregnancies were in the partial HELLP group, and one was in the complete HELLP group, with no statistical difference between the two groups. Additionally, there were no significant differences between the two groups for maternal BMI before delivery, gravidity, primiparity, chronic hypertension, history of preeclampsia, family history of hypertension, or history of cesarean section (Table 1).

Table 1. Comparison of general clinical data in partial and complete HELLP syndrome.

	Partial HELLP group (n = 73)	Complete HELLP group (n = 23)	Statistic values	*p*-values
Maternal age, years	30.3 ± 5.7	31.8 ± 5.2	$t = -1.117$	0.267
Maternal BMI before delivery, kg/m^2	27.8 ± 4	28.4 ± 3.8	$t = -0.556$	0.580
Gravidity	1 (1~2)	2 (0~3)	Z = –0.463	0.643
Primiparity, n (%)	46 (63%)	14 (61%)	$\chi^2 = 0.034$	0.853
Chronic hypertension, n (%)	11 (15%)	3 (13%)	—	>0.999
History of preeclampsia, n (%)	7 (10%)	5 (22%)	—	0.152
Family history of hypertension, n (%)	13 (18%)	6 (26%)	—	0.383
History of caesarean section, n (%)	12 (16%)	4 (17%)	—	>0.999
Twin pregnancies, n (%)	6 (8%)	1 (4%)	—	>0.999
Single pregnancy, n (%)	67 (92%)	22 (96%)	—	>0.999

BMI, body mass index.

Table 2. Comparison of the features and additional diseases of hypertensive disorders of pregnancy in partial and complete HELLP syndrome.

	Partial HELLP group (n = 73)	Complete HELLP group (n = 23)	Statistic values	*p*-values
Delivery week, weeks	33.6 ± 3.6	33.0 ± 4.2	$t = 0.575$	0.566
Highest SBP before delivery, mmHg	162.3 ± 24.8	164.6 ± 21.2	$t = -0.404$	0.687
Highest DBP before delivery, mmHg	104.9 ± 16.3	104.1 ± 14.0	$t = 0.202$	0.841
Highest MAP before delivery, mmHg	124.0 ± 18.5	124.3 ± 15.8	$t = -0.062$	0.951
According to early-onset or late-onset preeclampsia				
Early-onset, n (%)	53 (73%)	16 (70%)	$\chi^2 = 0.080$	0.778
Late-onset, n (%)	20 (27%)	7 (30%)		
According to antenatal or postpartum HELLP syndrome				
Antenatal, n (%)	62 (85%)	15 (65%)	—	0.068
Postpartum, n (%)	11 (15%)	8 (35%)		
Discharge time after delivery, days	5 (4~7)	5 (4~7)	Z = –0.707	0.879
Oligohydramnios	6 (8%)	0	—	0.330
Gestational diabetes mellitus, n (%)	20 (27%)	6 (26%)	$\chi^2 = 0.015$	0.902
Hypothyroidism, n (%)	7 (10%)	0	—	0.191
Placenta previa, n (%)	2 (3%)	1 (4%)	—	0.565

SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure.

3.2 Comparison of Features of Hypertension and Underlying Diseases between the Two Groups

According to the features of hypertensive disorders during pregnancy, both groups had similar gestational ages, systolic blood pressure, diastolic blood pressure, and mean arterial pressure ($p > 0.05$). The incidence of early-onset preeclampsia was higher than that of late-onset preeclampsia in both groups. In the partial HELLP group, antenatal HELLP syndrome accounted for 85%, and postpartum HELLP syndrome accounted for 15%; in the complete HELLP group, antenatal HELLP syndrome accounted for 65%, and postpartum HELLP syndrome accounted for 35%. There was no significant difference between the two groups ($p = 0.068$). Regarding underlying diseases, gestational diabetes mellitus was the most common. In two groups, a total of 26 patients had gestational diabetes (Table 2).

3.3 Comparison of Laboratory Indices, Clinical Symptoms, and Signs of Maternal HELLP Syndrome in the Two Groups

HELLP syndrome primarily presents with nonspecific clinical manifestations such as abdominal pain, nausea, vomiting, chest pain, and general discomfort. There was no difference in the incidence of these clinical symptoms between the two groups (Table 3). Therefore, laboratory indices play an important role in the diagnosis of HELLP. All three indicators met the inclusion criteria for the complete HELLP group; otherwise, the partial HELLP group was included. The two groups showed significant differences in ALT, AST, LDH, TBil, and platelet counts ($p < 0.05$). In terms of other laboratory indices, the white blood cell count and D-dimer level in the complete HELLP group were higher than those in the partial HELLP group ($p < 0.05$). There were no significant differences in hemoglobin, plasma fibrinogen, thrombin time, globulin, albumin, alkaline phosphatase, uric acid, serum creatinine, blood urea nitrogen, or blood calcium levels ($p > 0.05$) (Table 4).

Table 3. Comparison of clinical symptoms and signs in partial and complete HELLP syndrome.

	Partial HELLP group (n = 73)	Complete HELLP group (n = 23)	Statistic values	*p*-values
Headache or visual symptoms, n (%)	20 (27%)	8 (35%)	$\chi^2 = 0.462$	0.497
Upper abdominal pain, n (%)	19 (26%)	4 (17%)	$\chi^2 = 0.716$	0.397
Chest distress, n (%)	8 (11%)	2 (9%)	—	>0.999
Nausea or vomiting, n (%)	7 (10%)	3 (13%)	—	0.699
Vaginal bleeding, n (%)	2 (3%)	1 (4%)	—	0.565
Absent and/or reversed end-diastolic flow in the umbilical artery, n (%)	6 (8%)	3 (13%)	—	0.444

Table 4. Comparison of laboratory indexes in partial and complete HELLP syndrome.

	Partial HELLP group (n = 73)	Complete HELLP group (n = 23)	Statistic values	*p*-values
ALT, U/L	55.0 (22.5~122.0)	133.2 (71.0~378.0)	Z = –3.301	0.001
AST, U/L	51.9 (31.5~106.5)	168.9 (97.0~380.3)	Z = –4.219	<0.001
LDH, U/L	355.0 (271.4~534.1)	765.0 (456.0~1304.8)	Z = –4.399	<0.001
TBil, μmol/L	8.4 (6.2~11.3)	26.2 (22.5~37.5)	Z = –7.206	<0.001
PLT, $\times 10^9$/L	85.0 (72.0~99.0)	60.0 (41.0~68.0)	Z = –3.143	0.002
Hemoglobin, g/L	118.6 ± 16.0	118.7 ± 19.0	*t* = –0.034	0.973
White blood cell, $\times 10^9$/L	10.2 (7.7~13.1)	13.1 (10.4~15.1)	Z = –2.356	0.018
Fib, g/L	3.5 (2.71~4.45)	3.3 (2.6~4.3)	Z = –0.631	0.528
Thrombin time, s	17.6 (16.55~18.6)	18.3 (16.7~21.1)	Z = –1.722	0.085
D-dimer, mg/L	3.5 (1.9~8.2)	8.8 (4.6~10.6)	Z = –3.013	0.003
Globulin, g/L	24.1 (21.2~27.9)	24.3 (21.8~30.0)	Z = –0.610	0.542
Albumin, g/L	30.2 (27.5~35.1)	29.5 (28.2~32.7)	Z = –0.215	0.830
Alkaline phosphatase, U/L	134.1 (92.2~169.5)	121.3 (100.2~175.0)	Z = –0.103	0.918
Uric acid, μmol/L	437.5 (382.6~539.2)	472.0 (382.8~633.0)	Z = –1.137	0.255
Scr, μmol/L	68.0 (54.0~78.3)	68.1 (52.1~100.0)	Z = –0.760	0.447
BUN, mmol/L	5.6 (4.3~7.8)	5.8 (3.7~7.9)	Z = –0.077	0.938
Ca, mmol/L	2.1 ± 0.2	2.1 ± 0.2	*t* = 0.626	0.533

ALT, alanine aminotransferase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; TBil, total bilirubin; PLT, platelet; Fib, fibrinogen; Scr, serum creatinine; BUN, blood urea nitrogen.

3.4 Comparison of Neonatal and Maternal Outcomes

Due to improvements in diagnostic and treatment techniques, there were no maternal deaths in either the partial or complete HELLP group. The incidences of eclampsia, placental abruption, uteroplacental apoplexy, postpartum hemorrhage, and renal function injury were higher in the complete HELLP group than in the partial HELLP group; however, these differences were not statistically significant ($p > 0.05$). Regarding neonatal outcomes, there were no cases of three or more fetuses in either group, and no significant differences were observed between the two groups in embryo transfer rate, fetal growth restriction rate, preterm birth rate, neonatal birth weight, or neonatal mortality rate ($p > 0.05$) (Tables 5,6).

4. Discussion

The specific pathogenesis of HELLP syndrome remains unclear. Petca *et al.* [2] demonstrated that approximately 70%–80% of HELLP syndrome cases are secondary to preeclampsia. As a serious complication of preeclampsia, the main pathological changes are consistent with those seen in preeclampsia [7], such as vasospasm and reduced organ perfusion; however, the underlying mechanism of HELLP syndrome development remains unclear. The inflammatory immune system is abnormally activated in HELLP syndrome [8]. Some studies have found that patients with HELLP syndrome have a higher neutrophil-to-lymphocyte ratio and a lower platelet-to-lymphocyte ratio due to an increase in neutrophils in peripheral blood samples [9,10]. One study discovered that neutrophil extracellular traps promote endothelial cell activation and further increase the risk of thrombosis through the action of interleukin-1a (IL-1a) and cathepsin G [11]. This may be related to infection status; however, the specific causes remain unknown. In this study, patients with HELLP exhibited abnormal white blood cell counts, with the complete HELLP group showing higher white blood cell counts. Inflammation has been speculated as a potential cause of HELLP syndrome. However, due to limited data, experimental results related to neutrophils, monocytes, immune cells, and complement in white blood cells were not addressed in this study and require further investigation.

Table 5. Comparison of maternal outcomes in partial and complete HELLP syndrome.

	Partial HELLP group (n = 73)	Complete HELLP group (n = 23)	Statistic values	*p*-values
Maternal death, n (%)	0	0	—	—
Admission to the ICU, n (%)	23 (32%)	9 (39%)	$\chi^2 = 0.457$	0.499
Eclampsia, n (%)	3 (4%)	2 (9%)	—	0.590
Pre-admission eclampsia, n (%)	1 (1%)	1 (4%)	—	0.424
Placental abruption, n (%)	3 (4%)	3 (13%)	—	0.147
Uteroplacental apoplexy, n (%)	2 (3%)	1 (4%)	—	0.565
Postpartum hemorrhage, n (%)	5 (7%)	2 (9%)	—	0.672
Acute kidney injury, n (%)	4 (5%)	2 (9%)	—	0.627

ICU, intensive care unit.

Table 6. Comparison of perinatal infant outcomes in partial and complete HELLP syndrome.

	Partial HELLP group (n = 73)	Complete HELLP group (n = 23)	Statistic values	*p*-values
IVF-ET, n (%)	12 (16%)	3 (13%)	—	>0.999
FGR, n (%)	19 (26%)	6 (26%)	$\chi^2 = 0.000$	0.995
Preterm birth, 28 $\leq$ gestational weeks $<$ 37, n (%)	48 (66%)	12 (52%)	$\chi^2 = 1.376$	0.241
Full-term birth, 37 $\leq$ gestational weeks $<$ 42, n (%)	14 (19%)	7 (30%)	$\chi^2 = 1.297$	0.255
Birth weight, g	1794.7 $\pm$ 703.8	1770.9 $\pm$ 950.9	$t = 0.129$	0.898
Perinatal death, n (%)	9 (12%)	4 (17%)	—	0.504
Perinatal survival, n (%)	64 (88%)	19 (83%)	—	0.504
1 min Apgar	8 (6~10)	6 (2~10)	Z = –1.483	0.138
5 min Apgar	9 (8~10)	8 (7~10)	Z = –1.917	0.055

Note: For twins, the fetus with more serious general condition was selected for statistics, and the number of cases was recorded as 1 case.
IVF-ET, *in vitro* fertilization-embryo transfer; FGR, fetal growth restriction.

HELLP syndrome is believed to result from multiple factors, pathways, and mechanisms [12]. The annual delivery count at Wuxi Maternal and Child Health Hospital is approximately 10,000, with the incidence of HELLP syndrome estimated at 0.1%. Advanced age is a key risk factor for HELLP syndrome [13]. In this study, 23 patients (23.9%) were aged over 35 years, and the average age of the 96 pregnant women was 30.7 years, higher than the optimal childbearing age recommended by the National Health Commission. Obesity (BMI $\geq$30 kg/m^2) is a known risk factor for HELLP syndrome or preeclampsia [14]. In this study, the obesity rate in patients with HELLP was 18%, and excessive BMI during pregnancy should raise concern. A family history of hypertension is also an important risk factor for HELLP syndrome. In this study, 19 patients had a family history of hypertension, including 13 in the partial HELLP group and 6 in the complete HELLP group. The family history of hypertension was consistent with reports in the literature [15]. Patients with HELLP syndrome may also present with other conditions. This study found that approximately 27% of patients with HELLP syndrome had gestational diabetes, with the rate of gestational diabetes in the partial and complete HELLP groups ranging from 26% to 27%, showing no significant difference. Both gestational diabetes mellitus and gestational hypertension can impair vascular endothelial cell function, leading to microvascular damage and organ complications, such as kidney and retinal damage. The interaction between these two conditions poses a significant threat to the health of both the mother and fetus [16,17]. Therefore, when preeclampsia is accompanied by gestational diabetes, early intervention and monitoring of disease progression are essential to prevent HELLP syndrome. This analysis found no differences between patients with partial and complete HELLP regarding general age, gestational body mass index, family history of hypertension, history of preeclampsia, or pregnancy complications such as gestational diabetes mellitus.

Typical symptoms of HELLP syndrome include general discomfort, right upper abdominal pain, and increased pulse pressure difference. However, these symptoms are non-specific [18]. Some pregnant women may experience nausea, vomiting, and other gastrointestinal symptoms, which are difficult to identify. Hypertension and proteinuria are not typical features of HELLP syndrome [19]. Therefore, diagnosis primarily relies on laboratory tests [18]. The examination indicators were divided into two categories: diagnostic and non-diagnostic indicators of HELLP syndrome. Based on the grouping definitions, the two groups exhibited differences in ALT, AST, LDH, TBil, and platelet counts, with the complete HELLP group presenting more severe values. However, the white blood cell count and D-dimer levels in the complete HELLP group were significantly higher than those in the partial HELLP group. This suggests that patients with preeclampsia and

concurrent inflammation or hypercoagulability are more likely to develop complete HELLP syndrome. The strong reserve capacity of the kidneys makes the increase in serum creatinine (Scr) less apparent in early renal injury; when Scr levels rise, it often indicates severe kidney damage [20]. While not a prerequisite for diagnosing HELLP syndrome, this study identified 11 patients with Scr >90 µmol/L, accounting for 11.4%. This highlights the importance of monitoring renal function during the diagnosis and treatment of HELLP syndrome to protect kidney health.

Pregnancy termination is recommended for patients with HELLP syndrome. Severe preeclampsia and HELLP syndrome are not absolute contraindications for vaginal delivery, and the method of delivery should be based on the patient's condition and preferences. In this study, most pregnant women underwent cesarean section for pregnancy termination, while two patients with milder conditions opted for vaginal delivery. Both newborns survived without asphyxiation. Following active treatment measures, both women were discharged within four to five days after delivery.

HELLP syndrome can result in significant adverse pregnancy outcomes and poses a threat to maternal health. Eclampsia represents the most severe stage in the progression of hypertensive disorders during pregnancy and is characterized by convulsions that occur in preeclampsia. In this study, five women with HELLP syndrome experienced convulsions, which led to fetal demise in two cases. Among these five patients, two had convulsions before admission to the hospital and were transported to the emergency room by ambulance. Three women had antenatal convulsions, while two developed postpartum eclampsia, indicating that even after delivery termination, there remains a possibility of disease exacerbation or the occurrence of eclamptic episodes. Therefore, clinicians should remain vigilant during clinical diagnosis and treatment. This phenomenon may be attributed to the increased vulnerability of the brain and kidneys to persistent vascular endothelial dysfunction after childbirth [21]. Notably, there were no maternal deaths among the HELLP syndrome cases collected in this study from 2016 to 2023, which can be attributed to the extensive experience in emergency and critical care among clinicians at our hospital and the support and cooperation of the multidisciplinary team.

Patients with HELLP syndrome are susceptible to fetal growth restriction, perinatal asphyxia, and other complications, including systemic small artery constriction, high placental vascular resistance, and placental tissue ischemia [13]. Thirteen perinatal deaths occurred during the study period. The mean maternal arterial pressure of the fetuses who died was 137 mmHg, compared to 122 mmHg for the surviving fetuses. In terms of gestational age, eight of the 13 pregnant women were between 28 and 34 weeks of gestation. During pregnancy, particularly between 28 and 34 weeks, patients with preeclampsia should closely monitor their blood pressure and receive appropriate medical treatment under supervision.

The strength of our study lies in evaluating the outcomes associated with complete and partial HELLP syndrome using data collected from a single tertiary care medical center, where consistent protocols were followed for managing HELLP syndrome. Future studies may involve collaboration with hospitals in other regions to expand the study to multiple obstetric medical centers, which would enhance the generalizability of the results.

5. Conclusions

Partial and complete HELLP syndromes differ primarily in laboratory indices, but they exhibit similarities in general characteristics, pregnancy-related complications, other laboratory tests, and neonatal demographic features. Both conditions can result in severe perinatal outcomes. Clinical diagnosis and treatment do not require the simultaneous presence of all three indicators—hemolysis, elevated liver enzymes, and low platelet count. Partial HELLP syndrome should be considered, and timely clinical treatment and intervention are essential.

Abbreviations

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; IVF-ET, *in vitro* fertilization-embryo transfer; ALT, alanine aminotransferase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; TBil, total bilirubin; PLT, platelet; Scr, serum creatinine; BUN, blood urea nitrogen; Fib, fibrinogen; FGR, fetal growth restriction.

Availability of Data and Materials

Data is provided within the manuscript. All data in this paper are from the medical database of Wuxi Maternal and Child Health Hospital. To protect patient privacy, the specific data involved in this study is not publicly available. All data are available upon reasonable request from the corresponding author.

Author Contributions

HG designed the research study and analyzed the data. JYC collected the data. HQW designed the research study. MHJ and YG provided help and advice on the data collection and ethics application. YLF designed the work, reviewed the article, and provided financial support. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study was performed in accordance with the Declaration of Helsinki and was reviewed and approved by the

Ethics committee of the Wuxi Maternal and Child Health Hospital (No. 2024-06-0507-14). This is a retrospective research paper, and the article does not show any clinical details or images that may infer the identity of patients. All participants provided informed consent.

Acknowledgment

Not applicable.

Funding

This study was supported by the following funding sources: Jiangsu Maternal and Child Health Research Project (F202135); Project of Women's Health Care Department under the Key Disciplines of Maternal and Child Health Care in Jiangsu Province (SFY3-FB2021).

Conflict of Interest

The authors declare no conflict of interest.

Declaration of AI and AI-assisted Technologies in the Writing Process

In preparation for this work, we used ChatGpt-3.5 to check spelling and grammar. After using this tool, we review and edit the content as needed and take full responsibility for the content of the publication.

References

[1] Weinstein L. Syndrome of hemolysis, elevated liver enzymes, and low platelet count: a severe consequence of hypertension in pregnancy. American Journal of Obstetrics and Gynecology. 1982; 142: 159–167. https://doi.org/10.1016/s0002-9378(16)32330-4.

[2] Petca A, Miron BC, Pacu I, Dumitrașcu MC, Mehedințu C, Șandru F, *et al*. HELLP Syndrome-Holistic Insight into Pathophysiology. Medicina (Kaunas, Lithuania). 2022; 58: 326. https://doi.org/10.3390/medicina58020326.

[3] Westbrook RH, Dusheiko G, Williamson C. Pregnancy and liver disease. Journal of Hepatology. 2016; 64: 933–945. https://doi.org/10.1016/j.jhep.2015.11.030.

[4] Li B, Yang H. Comparison of clinical features and pregnancy outcomes in early- and late-onset preeclampsia with HELLP syndrome: a 10-year retrospective study from a tertiary hospital and referral center in China. BMC Pregnancy and Childbirth. 2022; 22: 186. https://doi.org/10.1186/s12884-022-04466-9.

[5] Sibai BM. The HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): much ado about nothing? American Journal of Obstetrics and Gynecology. 1990; 162: 311–316. https://doi.org/10.1016/0002-9378(90)90376-i.

[6] Aydin S, Ersan F, Ark C, Arıoğlu Aydın C. Partial HELLP syndrome: maternal, perinatal, subsequent pregnancy and long-term maternal outcomes. The Journal of Obstetrics and Gynaecology Research. 2014; 40: 932–940. https://doi.org/10.1111/jog.12295.

[7] Abildgaard U, Heimdal K. Pathogenesis of the syndrome of hemolysis, elevated liver enzymes, and low platelet count (HELLP): a review. European Journal of Obstetrics, Gynecology, and Reproductive Biology. 2013; 166: 117–123. https://doi.org/10.1016/j.ejogrb.2012.09.026.

[8] Stojanovska V, Zenclussen AC. Innate and Adaptive Immune Responses in HELLP Syndrome. Frontiers in Immunology. 2020; 11: 667. https://doi.org/10.3389/fimmu.2020.00667.

[9] Sisti G, Faraci A, Silva J, Upadhyay R. Neutrophil-to-Lymphocyte Ratio, Platelet-to-Lymphocyte Ratio, and Routine Complete Blood Count Components in HELLP Syndrome: A Matched Case Control Study. Medicina (Kaunas, Lithuania). 2019; 55: 123. https://doi.org/10.3390/medicina55050123.

[10] İpek G, Tanaçan A, Ağaoğlu Z, Peker A, Şahin D. Can SIRI or other inflammatory indices predict HELLP syndrome in the first trimester? Journal of Reproductive Immunology. 2023; 159: 104126. https://doi.org/10.1016/j.jri.2023.104126.

[11] Folco EJ, Mawson TL, Vromman A, Bernardes-Souza B, Franck G, Persson O, *et al*. Neutrophil Extracellular Traps Induce Endothelial Cell Activation and Tissue Factor Production Through Interleukin-1α and Cathepsin G. Arteriosclerosis, Thrombosis, and Vascular Biology. 2018; 38: 1901–1912. https://doi.org/10.1161/ATVBAHA.118.311150.

[12] Wang LQ, Bone JN, Muraca GM, Razaz N, Joseph KS, Lisonkova S. Prepregnancy body mass index and other risk factors for early-onset and late-onset haemolysis, elevated liver enzymes and low platelets (HELLP) syndrome: a population-based retrospective cohort study in British Columbia, Canada. BMJ Open. 2024; 14: e079131. https://doi.org/10.1136/bmjopen-2023-079131.

[13] Poon LC, Shennan A, Hyett JA, Kapur A, Hadar E, Divakar H, *et al*. The International Federation of Gynecology and Obstetrics (FIGO) initiative on pre-eclampsia: A pragmatic guide for first-trimester screening and prevention. International Journal of Gynaecology and Obstetrics: the Official Organ of the International Federation of Gynaecology and Obstetrics. 2019; 145: 1–33. https://doi.org/10.1002/ijgo.12802.

[14] Rimboeck J, Gruber M, Weigl M, Huber P, Lunz D, Petermichl W. Obesity Correlates with Chronic Inflammation of the Innate Immune System in Preeclampsia and HELLP Syndrome during Pregnancy. Biomedicines. 2023; 11: 2851. https://doi.org/10.3390/biomedicines11102851.

[15] Bezerra PCFM, Leão MD, Queiroz JW, Melo EMD, Pereira FVM, Nóbrega MH, *et al*. Family history of hypertension as an important risk factor for the development of severe preeclampsia. Acta Obstetricia et Gynecologica Scandinavica. 2010; 89: 612–617. https://doi.org/10.3109/00016341003623720.

[16] Nunes JS, Ladeiras R, Machado L, Coelho D, Duarte C, Furtado JM. The Influence of Preeclampsia, Advanced Maternal Age and Maternal Obesity in Neonatal Outcomes Among Women with Gestational Diabetes. Revista Brasileira De Ginecologia E Obstetricia: Revista Da Federacao Brasileira Das Sociedades De Ginecologia E Obstetricia. 2020; 42: 607–613. https://doi.org/10.1055/s-0040-1710300.

[17] Dmitrenko OP, Karpova NS, Nurbekov MK, Papysheva OV. I/D Polymorphism Gene ACE and Risk of Preeclampsia in Women with Gestational Diabetes Mellitus. Disease Markers. 2020; 2020: 8875230. https://doi.org/10.1155/2020/8875230.

[18] Hypertensive Disorders in Pregnancy Subgroup, Chinese Society of Obstetrics and Gynecology, Chinese Medical Association. Diagnosis and treatment of hypertension and pre-eclampsia in pregnancy: a clinical practice guideline in China (2020). Zhonghua Fu Chan Ke Za Zhi. 2020; 55: 227–238. https://doi.org/10.3760/cma.j.cn112141-20200114-00039. (In Chinese)

[19] Rath W, Faridi A, Dudenhausen JW. HELLP syndrome. Journal of Perinatal Medicine. 2000; 28: 249–260. https://doi.org/10.1515/JPM.2000.033.

[20] Becker J, Friedman E. Renal function status. AJR. American Journal of Roentgenology. 2013; 200: 827–829. https://doi.org/10.2214/AJR.12.9872.

[21] Katsi V, Skalis G, Vamvakou G, Tousoulis D, Makris T. Postpartum Hypertension. Current Hypertension Reports. 2020; 22: 58. https://doi.org/10.1007/s11906-020-01058-w.