

Association of Liver and Renal Function Parameters, Platelet Count and Lactate Dehydrogenase with Severity of Hypertensive Disorders of Pregnancy: A Prospective Observational Study

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ABSTRACT

Background: Hypertensive disorders of pregnancy are multisystem conditions in which hepatic injury, renal dysfunction, thrombocytopenia and tissue injury may accompany increasing clinical severity. Simple laboratory parameters may therefore provide useful information during maternal assessment.

Objectives: To evaluate the association of liver function parameters, renal function parameters, platelet count and lactate dehydrogenase (LDH) with severity of hypertensive disorders of pregnancy.

Methods: This prospective observational study included 50 pregnant women with hypertensive disorders of pregnancy. Women were classified as having mild (n=28) or severe disease (n=22) according to the severity category recorded clinically. Demographic, blood pressure and laboratory variables were analysed. Because several variables were non-normally distributed, between-group comparisons used the Mann-Whitney U test. Spearman rank correlation was used to examine the association between laboratory parameters and an ordinal severity code.

Results: Severe disease was associated with markedly higher median AST, ALT, ALP, total bilirubin, urea, creatinine, uric acid and LDH, together with lower albumin and platelet count (all $p < 0.001$). Median LDH increased from 213.0 (185.0-236.2) in mild disease to 764.0 (712.8-1072.5) in severe disease, while platelet count decreased from 205.0 (194.2-225.0) to 76.5 (65.8-84.8). Creatinine increased from 0.6 (0.6-0.7) to 1.6 (1.4-2.0). Each laboratory parameter showed a strong rank association with the recorded severity category, with absolute Spearman rho values ranging from 0.860 to 0.880 (all $p < 0.001$).

Conclusion: In this cohort, severe hypertensive disease was accompanied by a consistent biochemical pattern of hepatic injury, renal impairment, thrombocytopenia and elevated LDH. These routinely available parameters may support clinical assessment of disease severity, although larger studies with multivariable adjustment are required before defining independent prognostic thresholds.

Keywords: Hypertensive Disorders of Pregnancy, Preeclampsia, Liver Function Tests, Renal Function Tests, Platelet Count, Lactate Dehydrogenase, HELLP Syndrome.

INTRODUCTION

Hypertensive disorders of pregnancy remain an important cause of maternal and perinatal morbidity worldwide. Pre-eclampsia usually develops after 20 weeks of gestation and may progress from hypertension to a multisystem disorder involving the liver, kidneys, haematological system, central nervous

system and uteroplacental circulation.^[1,2] Contemporary guidance therefore places increasing emphasis on end-organ involvement rather than blood pressure alone when assessing disease severity.^[2,3]

The laboratory profile of hypertensive disease reflects its underlying endothelial and microvascular injury. Hepatic involvement may

appear as elevation of aminotransferases and bilirubin, while renal involvement can be accompanied by rising creatinine, urea and uric acid. Platelet consumption is particularly important in severe disease and in the haemolysis, elevated liver enzymes and low platelet count (HELLP) spectrum.^[4,5] Lactate dehydrogenase (LDH), although nonspecific, is a readily available marker of cellular injury and haemolysis and has been reported to rise with increasing severity of pre-eclampsia and eclampsia.^[6]

For tertiary-care obstetric units, the attraction of these tests lies in their availability. They can be repeated rapidly and interpreted alongside blood pressure, symptoms and gestational age. Yet the magnitude and pattern of laboratory abnormalities vary across patient populations, and a single parameter should not be treated as a stand-alone severity marker. The present study was undertaken to examine the association of routinely measured liver and renal function parameters, platelet count and LDH with clinically recorded severity of hypertensive disorders of pregnancy.

MATERIALS AND METHODS

Study Design and Setting

A prospective observational study was conducted in the Department of Obstetrics and Gynaecology at Chikkamagaluru Institute of medical Sciences, Chikkamagaluru, Karnataka for a period of one year from February 2025 to February 2026. The study included 50 pregnant women diagnosed with a hypertensive disorder of pregnancy.

Study Population and Clinical Classification

The analytical cohort comprised women recorded as having gestational hypertension, preeclampsia, severe preeclampsia, eclampsia or HELLP syndrome. The clinical record further classified disease severity as mild or severe. For the present analysis, this recorded severity category was retained without reclassification. Of the 50 women, 28 were in the mild category and 22 were in the severe category.

Variables Assessed

Maternal age, gestational age, parity, systolic and diastolic blood pressure, and the recorded

diagnostic category were analysed. Laboratory variables included aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), total bilirubin, albumin, urea, creatinine, uric acid, platelet count and LDH. No additional laboratory or outcome variables were introduced beyond those recorded for the study cohort.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of the participating institution prior to initiation. Eligible pregnant women were enrolled after obtaining informed consent. Participant confidentiality and anonymity were maintained throughout the study. Clinical assessment and laboratory investigations were undertaken as part of routine obstetric care, and no research-specific therapeutic intervention was introduced.

Statistical Analysis

Continuous variables were inspected for distributional characteristics. Because several laboratory measures showed non-normal distributions within severity groups, results are presented predominantly as median with interquartile range (IQR), and mild versus severe categories were compared using the Mann-Whitney U test. Categorical variables are shown as frequencies and percentages; Fisher exact test was used for nulliparity because of the sample size. For exploratory association analysis, severity was coded as 0 for mild and 1 for severe, and Spearman rank correlation coefficients were calculated for laboratory variables. A two-sided p-value <0.05 was considered statistically significant. Analyses were performed directly from the 50 participant records.

RESULTS

The study included 50 women with hypertensive disorders of pregnancy. Twenty-eight women (56.0%) were classified as having mild disease and 22 (44.0%) as severe disease (Figure 1). Diagnostic categories comprised gestational hypertension in 14 (28.0%), preeclampsia in 14 (28.0%), severe preeclampsia in 13 (26.0%), HELLP syndrome in 5 (10.0%) and eclampsia in 4 (8.0%) women (Figure 2).

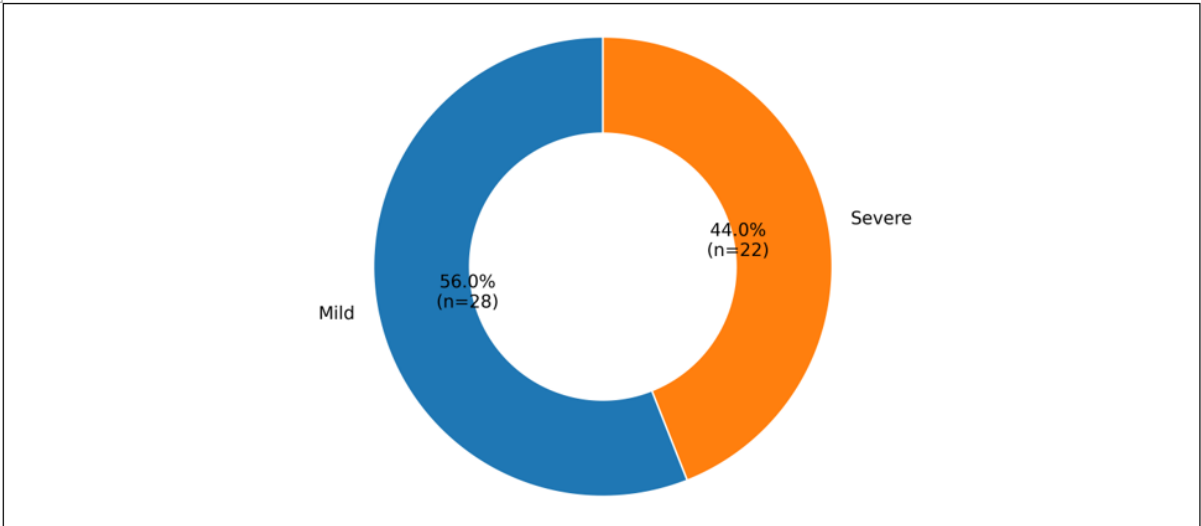


Figure 1. Distribution of mild and severe hypertensive disorders of pregnancy
Donut chart shows the proportion and number of women in each recorded severity category

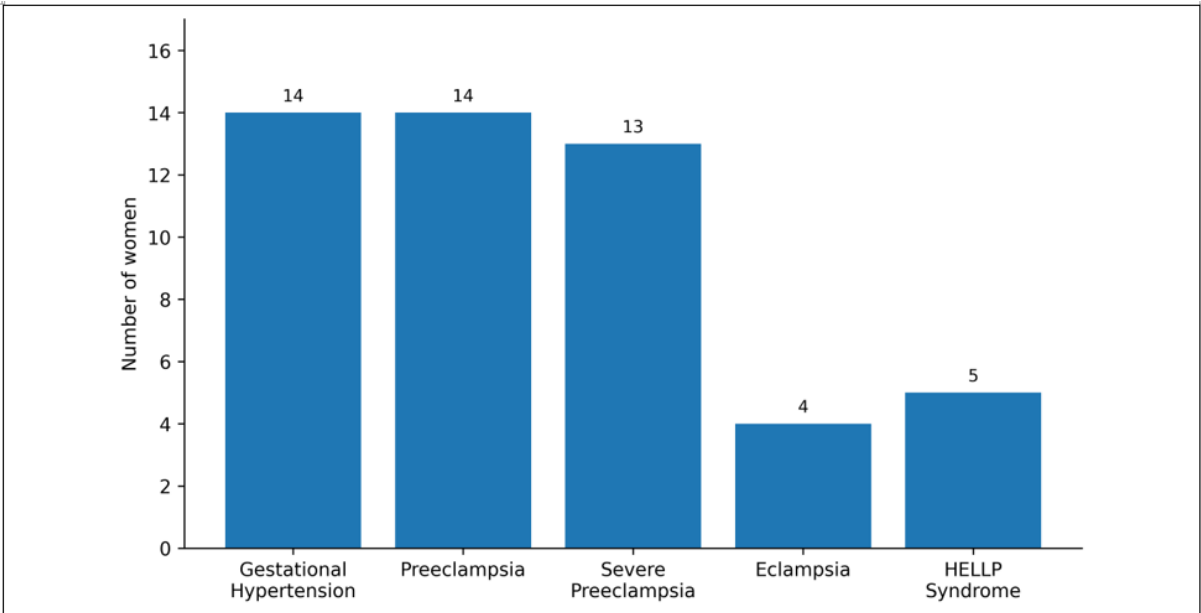


Figure 2. Distribution of clinical diagnostic categories in the study cohort
Bars represent the number of women in each recorded hypertensive disorder category

Variable	Mild (n=28)	Severe (n=22)	p-value
Age (years)	26.0 (23.8-29.0)	33.5 (31.0-35.8)	<0.001
Gestational age (weeks)	36.0 (35.0-38.0)	31.0 (30.0-32.0)	<0.001
Nulliparous, n (%)	17 (60.7)	8 (36.4)	0.154
Systolic blood pressure	144.0 (140.0-146.5)	165.0 (160.5-173.5)	<0.001
Diastolic blood pressure	92.0 (88.0-94.0)	111.0 (106.5-115.5)	<0.001

Table 1. Clinical characteristics according to disease severity (n=50)
Values are median (IQR) unless otherwise stated. Mann-Whitney U test for continuous variables; Fisher exact test for nulliparity

Women in the severe category were older and presented at an earlier gestational age than those in the mild category. Systolic and diastolic blood pressure were also substantially higher in severe disease (all p<0.001), whereas the proportion of nulliparous women did not differ significantly (Table 1).

Parameter	Mild (n=28)	Severe (n=22)	p-value
AST	27.0 (22.0-30.5)	106.0 (95.2-163.8)	<0.001
ALT	23.0 (18.0-26.5)	93.0 (82.5-146.5)	<0.001
ALP	99.0 (92.0-105.2)	173.5 (162.5-197.2)	<0.001
Total bilirubin	0.6 (0.5-0.7)	1.9 (1.6-2.5)	<0.001
Albumin	3.6 (3.5-3.7)	2.7 (2.4-2.8)	<0.001

Table 2. Liver-related laboratory parameters according to disease severity
Values are median (IQR). Mann-Whitney U test

All liver-related parameters differed significantly by severity (Table 2). Severe disease showed higher AST, ALT, ALP and total bilirubin, while albumin was lower. The separation was particularly marked for aminotransferases: median AST was 27.0

(22.0-30.5) in mild disease versus 106.0 (95.2-163.8) in severe disease, and median ALT was 23.0 (18.0-26.5) versus 93.0 (82.5-146.5), respectively (both $p<0.001$). Figure 3 illustrates the differences in AST, ALT and ALP.

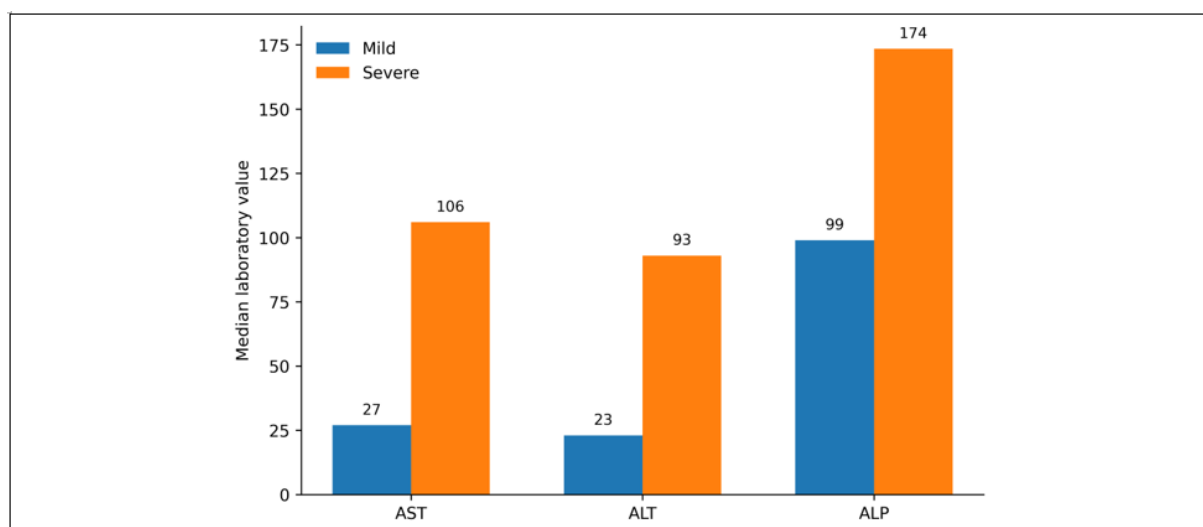


Figure 3. Median AST, ALT and ALP values according to severity category
Grouped bars display median laboratory values in mild and severe hypertensive disorders of pregnancy

Parameter	Mild (n=28)	Severe (n=22)	p-value
Urea	20.5 (18.0-22.2)	46.5 (42.2-55.8)	<0.001
Creatinine	0.6 (0.6-0.7)	1.6 (1.4-2.0)	<0.001
Uric acid	4.3 (3.9-4.8)	8.1 (7.6-8.9)	<0.001
Platelet count	205.0 (194.2-225.0)	76.5 (65.8-84.8)	<0.001
LDH	213.0 (185.0-236.2)	764.0 (712.8-1072.5)	<0.001

Table 3. Renal parameters, platelet count and LDH according to disease severity
Values are median (IQR). Mann-Whitney U test

Renal function markers and LDH increased consistently with severe disease, whereas platelet count fell sharply (Table 3). Median creatinine increased from 0.6 (0.6-0.7) in mild disease to 1.6 (1.4-2.0) in severe disease. LDH increased from 213.0 (185.0-

236.2) to 764.0 (712.8-1072.5), while platelet count decreased from 205.0 (194.2-225.0) to 76.5 (65.8-84.8); all comparisons had $p<0.001$. These opposing patterns are shown in Figure 4.

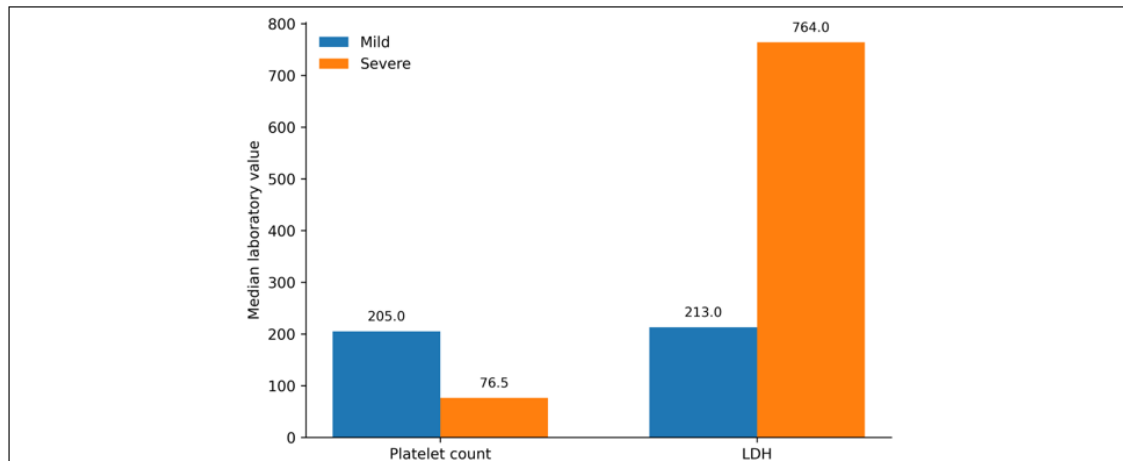


Figure 4. Median platelet count and LDH according to disease severity

Grouped bars show the marked fall in platelet count and rise in LDH among women with severe disease

Exploratory rank-correlation analysis demonstrated strong associations between each laboratory parameter and the recorded severity category (Table 4). Creatinine showed the largest positive coefficient ($\rho=0.880$), while albumin and platelet count showed strong inverse associations ($\rho=-0.863$ and -0.860 , respectively). All laboratory correlations were statistically significant at $p<0.001$.

DISCUSSION

The present study identified a coherent laboratory pattern across the severity spectrum of hypertensive disorders of pregnancy. Women classified with severe disease had substantially higher aminotransferases, bilirubin, urea, creatinine, uric acid and LDH, together with lower serum albumin and platelet count. The magnitude and consistency of these differences are clinically plausible because severe pre-eclampsia and related syndromes are characterised by diffuse endothelial dysfunction, vasoconstriction, microangiopathy and progressive involvement of multiple maternal organs.^[2,3]

Hepatic abnormalities were among the most conspicuous findings. AST and ALT were several-fold higher in the severe category, accompanied by higher ALP and bilirubin and lower albumin. Liver enzyme elevation in pre-eclampsia reflects hepatocellular injury and altered hepatic microcirculation, and becomes particularly relevant when disease approaches the HELLP spectrum.^[5] Recent cohort evidence also cautions against interpreting aminotransferase elevation in isolation. Greiner et al. found that AST or ALT levels at

least twice the upper limit of normal among patients with pre-eclampsia with severe features were not independently associated with adverse maternal outcomes, and the observed association with neonatal outcomes was attenuated after adjustment for gestational age at delivery.^[7] More recent meta-analytic evidence likewise demonstrates higher AST, ALT, ALP and bilirubin among women with pre-eclampsia compared with normotensive pregnancies.^[8] The current findings extend that pattern within an affected cohort by showing a clear separation between clinically recorded mild and severe disease.

Renal markers followed the same direction. Severe disease was associated with higher urea, creatinine and uric acid. Renal involvement in pre-eclampsia is linked to systemic endothelial injury, glomerular endotheliosis, altered angiogenic signalling and reduced filtration.^[9] In clinical practice, even a modest rise in serum creatinine during pregnancy can be important because normal gestation is usually accompanied by increased glomerular filtration. Uric acid also rose strongly with severity in this cohort. Large meta-analyses have confirmed that uric acid is generally higher in pre-eclampsia and tends to be higher in severe disease, eclampsia and HELLP syndrome.^[10] More recent case-control and meta-analytic evidence also supports an association between higher maternal uric acid and pre-eclampsia risk, while emphasising that observational association alone does not establish a causal or stand-alone prognostic role.^[11] It is therefore better interpreted as part of a broader biochemical and clinical profile.

The fall in platelet count and rise in LDH were especially marked. Platelet consumption results from endothelial activation and microangiopathic processes, while LDH may rise with tissue injury and haemolysis. These mechanisms are central to HELLP syndrome and severe pre-eclampsia disease.^[5,12] HELLP remains a particularly important severe phenotype because haemolysis, liver enzyme elevation and thrombocytopenia may evolve rapidly and require close maternal surveillance and timely obstetric management.^[13] Reddy Eleti and colleagues, in a 2023 Indian study, also reported progressively higher serum LDH across mild pre-eclampsia, severe pre-eclampsia and eclampsia, with higher levels associated with adverse fetomaternal outcomes.^[6] At the same time, thrombocytopenia in pregnancy has a broad differential diagnosis, and interpretation must remain clinical rather than numerical alone.^[14]

An important feature of this cohort is that severe disease was also associated with older maternal age and earlier gestational age. These differences matter. They indicate that the laboratory-severity relationships cannot automatically be interpreted as independent effects. With only 50 participants, multivariable modelling would be unstable and potentially misleading. The very strong rank correlations observed here should therefore be read as descriptive associations within this cohort rather than as validated prognostic coefficients or diagnostic thresholds.

The study has practical relevance to tertiary obstetric care, particularly where rapid access to advanced angiogenic biomarkers is limited. AST, ALT, bilirubin, creatinine, uric acid, platelet count and LDH are routinely obtainable in most secondary and tertiary hospitals and can help clinicians recognise a pattern of evolving end-organ involvement. Their value lies in combination with blood pressure, symptoms, gestational age and fetal assessment, not in replacing clinical judgement or established diagnostic criteria.^[2,3] Recent meta-analytic evidence continues to show an association between a history of pre-eclampsia and later chronic kidney disease and end-stage kidney disease, while the causal relationship and optimal intensity of long-term follow-up remain uncertain.^[15]

Several limitations should be acknowledged. The sample size was modest

and derived from a single tertiary-care cohort. The study did not include a normotensive comparison group, so its conclusions concern severity within hypertensive disease rather than the diagnostic ability of these tests to identify hypertensive disorders. Maternal and neonatal outcome variables were not available for analysis, preventing assessment of prognostic performance. The severity groups also differed in age and gestational age, and no adjusted analysis was attempted. Finally, the strong separation of several laboratory variables across the two groups may partly reflect the clinical diagnostic composition of the severe category, which included severe preeclampsia, eclampsia and HELLP syndrome.

CONCLUSION

Severe hypertensive disorders of pregnancy in this cohort were associated with a consistent pattern of hepatic dysfunction, renal impairment, reduced platelet count and elevated LDH. AST, ALT, bilirubin, urea, creatinine, uric acid and LDH were higher in severe disease, whereas albumin and platelet count were lower. These routinely available investigations can complement clinical assessment of maternal disease severity. Larger studies incorporating maternal and neonatal outcomes, gestational-age adjustment and multivariable modelling are needed before independent prognostic cut-offs can be proposed.

Source of Funding

Nil.

Conflict of Interest

None declared.

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