International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

Exploring the Association Between Markers of Blood Rheology and Liver Dysfunction in Hypertensive Patients: A Cross-Sectional Analysis

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Abstract: ***Background**: Hypertension is a systemic inflammatory condition known to induce both liver dysfunction and alterations in blood rheology. Markers of hepatocyte injury, such as Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST), and markers indicative of blood viscosity, including Haematocrit (HCT) and Erythrocyte Sedimentation Rate (ESR), are frequently elevated in hypertensive patients. However, the direct association between these haematological and hepatic markers remains under-characterized. **Objective**: To investigate the association between markers of liver dysfunction (ALT, AST) and markers of blood rheology (HCT, ESR, and C-Reactive Protein (CRP)) in a cohort of hypertensive patients. **Methods**: A cross-sectional, observational study was conducted on 198 patients diagnosed with primary hypertension. Serum levels of ALT, AST, CRP, and haematological parameters including HCT and ESR were measured. Patients were categorized into Grade 1 and Grade 2 hypertension. Statistical analysis involved independent t-tests to compare mean differences between groups and Pearson correlation analysis to assess the relationship between variables. **Results**: Patients with Grade 2 hypertension exhibited significantly higher mean levels of ALT ($p < 0.001$), AST ($p < 0.001$), and ESR ($p < 0.001$) compared to those with Grade 1 hypertension. A statistically significant, moderate positive correlation was found between HCT and both ALT ($r = 0.54$, $p < 0.001$) and AST ($r = 0.51$, $p < 0.001$). No significant correlation was observed for ESR or CRP with the liver enzymes. **Conclusion**: The severity of hypertension is associated with worsening liver function and elevated ESR. Furthermore, HCT demonstrates a direct positive correlation with markers of liver injury, suggesting that increased blood viscosity may be linked to the pathophysiology of hypertension-induced liver dysfunction.*

Keywords: Hypertension, Liver dysfunction, Blood rheology, Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), Haematocrit (HCT), Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP), Systemic inflammation, Hepatocyte injury, Cardiovascular risk, Metabolic risk

1. Introduction

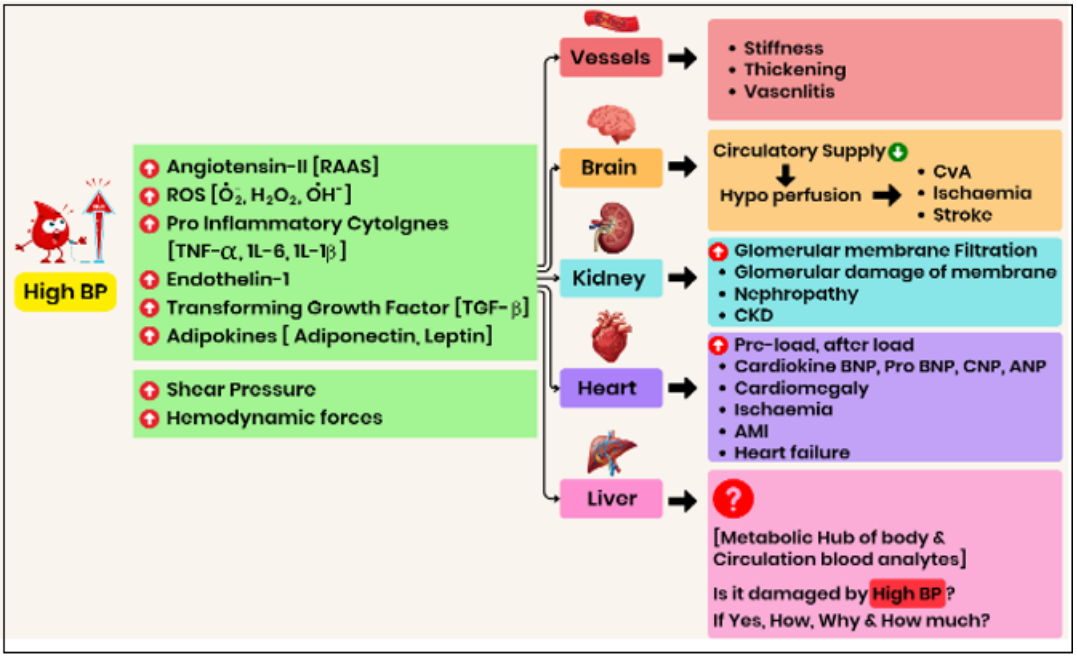


Figure 1: Overview of how hypertension, through various pathways (RAAS, oxidative stress, inflammation), impacts multiple organs, including the liver.

Hypertension, a chronic medical condition affecting a significant portion of the global population, is increasingly understood not merely as a hemodynamic disorder but as a state of chronic, low-grade systemic inflammation that inflicts multi-organ damage. [1] While its detrimental effects on the cardiovascular and renal systems are well-established, its impact on metabolic organs, particularly the liver, remains an area of growing clinical interest. The liver, a central hub for metabolism, detoxification, and protein synthesis, is highly vulnerable to the systemic consequences of chronic hypertension. [1]

Hypertension and Liver Dysfunction: The link between hypertension and hepatocyte injury is becoming progressively clearer, often manifesting clinically as non-alcoholic fatty liver disease (NAFLD). [1] The systemic inflammation, oxidative stress, and hemodynamic alterations characteristic of hypertension can lead to hepatic ischemia, congestion, and fibrosis. [1] Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST) are sensitive and specific biomarkers that are released into the circulation upon hepatocellular damage, serving as key indicators of this subclinical organ injury. [1] Elevated levels of these enzymes are frequently observed in hypertensive individuals, signaling an ongoing pathological process within the liver. [1]

Hypertension and Altered Blood Rheology: Concurrently, hypertension exerts a profound influence on blood rheology—the study of the flow properties of blood. [2,3] Blood viscosity and density are critical determinants of peripheral vascular resistance and, consequently, cardiac workload. An increase in blood viscosity can retard flow, increase resistance, and thereby contribute to the maintenance and exacerbation of the hypertensive state. [2,4] Understanding the factors that govern blood rheology is therefore essential to comprehending the pathophysiology of hypertension.

Justification of Blood Rheology Markers: This study employs Haematocrit (HCT), Erythrocyte Sedimentation Rate (ESR), and C-Reactive Protein (CRP) as clinical indicators of blood viscosity and density. The selection of these markers is grounded in established pathophysiological principles.

Haematocrit (HCT) represents the volume percentage of red blood cells (RBCs) in whole blood and is recognized as the single most powerful intrinsic determinant of blood viscosity and density. As the cellular fraction of blood increases, the internal friction of the fluid rises exponentially, directly increasing viscosity and vascular resistance. [2,4-7]

Erythrocyte Sedimentation Rate (ESR) serves as a crucial indirect indicator of blood rheology, specifically reflecting the tendency of RBCs to aggregate. In inflammatory states, the liver produces acute-phase proteins, most notably fibrinogen. These large, asymmetric proteins neutralize the negative surface charge (zeta potential) of RBCs, which normally causes them to repel each other. This neutralization allows RBCs to stack together in formations known as 'rouleaux'.[8-10] According to Stokes' law, the sedimentation velocity is proportional to the square of the particle's radius; thus, these larger, denser rouleaux aggregates sediment much faster than individual cells, resulting in an elevated ESR.[10,11] This aggregation phenomenon is a key component of blood's non-Newtonian, shear-thinning behaviour, where viscosity is highest at low flow rates.[12]

C-Reactive Protein (CRP) is a primary biomarker of the systemic inflammation that underpins the changes in blood rheology. Produced by hepatocytes in response to pro-inflammatory cytokines like interleukin-6 (IL-6), CRP is a hallmark of the acute-phase response.[13-15] While not a direct measure of viscosity, its elevation signals the inflammatory state that triggers the hepatic synthesis of fibrinogen and other proteins, which in turn elevates the ESR and contributes to the overall rheological alterations.[16-18]

These markers, therefore, represent different facets of a single, interconnected pathophysiological process. A proposed cascade begins with systemic inflammation (indicated by CRP), which stimulates the production of acute-phase proteins. These proteins enhance RBC aggregation (measured by ESR). This inflammatory milieu, potentially coupled with effects on erythropoiesis and plasma volume, also contributes to an increased cellular fraction of blood (measured by HCT). Together, these changes culminate in increased blood viscosity and density, key contributors to vascular pathology in hypertension.

Rationale and Hypothesis: While the individual links between hypertension and liver injury, and between hypertension and altered blood rheology, are known, the direct association between these hepatic and haematological markers is not well-characterized.[1] Understanding this relationship could provide a more holistic view of the systemic damage caused by hypertension. This study, therefore, was designed to explore this link. The null hypothesis posits that no correlation exists between markers of liver dysfunction and markers of blood rheology in hypertensive patients. The alternative hypothesis is that a direct, positive correlation exists between markers of liver dysfunction (ALT, AST) and markers of blood rheology (HCT, ESR, CRP).

2. Study Population and Methods

Study Design and Setting

A cross-sectional, observational, descriptive study was conducted at the General Medicine and Non-Communicable Disease (NCD) Outpatient Department (OPD) clinics of Murshidabad Medical College and Hospital. This facility is a tertiary care center located in Murshidabad, West Bengal, India. The study was carried out over a period of one and a half years, from September 2023 to March 2025. All protocols received approval from the Institutional Ethics Committee and the Scientific Research Committee of the institution. [1]

Study Population

The study population comprised 198 adult patients between the ages of 18 and 70 years who were diagnosed with primary hypertension. All participants provided written informed consent prior to enrolment. [1]

The inclusion criteria for the study were:

1) Adults aged 18 to 70 years with a confirmed diagnosis of

primary hypertension, as defined by WHO guidelines.
2) Patients who had not been previously diagnosed with any form of chronic liver disease.
3) Individuals willing and able to provide informed consent for participation. [1]

The exclusion criteria were comprehensive to isolate the effects of primary hypertension:

- Patients with a history of liver surgery.
- Patients who had used known hepatotoxic drugs within the preceding two weeks.
- Patients with a history of alcohol abuse or tobacco use.
- Patients with comorbid conditions known to affect liver or haematological parameters, including diabetes mellitus, chronic kidney disease (CKD), and dyslipidaemia.
- Patients with a known history of viral infections such as Human Immunodeficiency Virus (HIV), Hepatitis B, or Hepatitis C.
- Patients who were pregnant or breastfeeding. [1]

Data Collection and Clinical Measurements

Demographic data, including age and sex, and anthropometric measurements, including weight and height, were collected through patient interviews and a review of medical records. Body Mass Index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m^2).[1]

Blood pressure (BP) was measured in the left arm of seated, resting patients using a calibrated sphygmomanometer. Three separate readings were taken at five-minute intervals, and the average of these readings was recorded to ensure accuracy. Hypertension was classified into grades based on WHO criteria: Grade 1 (mild) was defined as a BP of 140/90–159/99 mmHg, and Grade 2 (moderate) as a BP of 160/100–179/109 mmHg. [1]

Biochemical and Haematological Analysis

Venous blood samples were collected from all participants following an 8 to 12-hour overnight fast. Samples were processed according to standard laboratory protocols to ensure integrity. All biochemical analyses were performed on an Erba EM 360 automated analyzer (Serial No. 60520) in the central laboratory of the hospital.[1]

The specific laboratory methods employed were as follows:

- **ALT and AST:** Measured using a Modified International Federation of Clinical Chemistry (IFCC) Kinetic Method.
- **CRP:** Measured using a standard immunoturbidimetric assay.
- **HCT and ESR:** Measured using standard automated haematology analyzers. Internal and external quality controls were rigorously maintained to ensure the accuracy and precision of all laboratory results.[1]

Statistical Analysis

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS), version 25. The distribution of all continuous variables was first assessed for normality using the Kolmogorov-Smirnov and Shapiro-Wilk tests.[1] These tests revealed that the distributions of key variables, including ALT, AST, HCT, and ESR, significantly deviated from a normal distribution ($p<0.001$ for all).[1]

Based on these findings, the following statistical plan was executed. For the comparison of markers between the two hypertension grades (Grade 1 vs. Grade 2), the non-parametric Independent-Samples Mann-Whitney U test was employed, which compares median ranks rather than means. For the primary objective of assessing the linear relationship between continuous variables (HCT, ALT, and AST), the Pearson correlation coefficient (r) was utilized. While this test technically assumes bivariate normality, its application is considered robust in a large sample size (N=198) for evaluating the strength and direction of a linear association, a common and acceptable practice in exploratory clinical research.

Descriptive statistics were calculated for all variables and presented as mean ± standard deviation (SD) or median with interquartile range (IQR) for continuous variables, and as frequencies and percentages (n, %) for categorical variables. A two-tailed p-value of less than 0.05 was considered statistically significant for all inferential tests.

3. Results

Baseline Characteristics of the Study Cohort

The study included 198 patients diagnosed with primary hypertension. The baseline demographic, clinical, and laboratory characteristics of the cohort are summarized in Table 1. The mean age of the participants was 42.9 \pm 11.1 years, with a male predominance (62.6%). The majority of patients were classified as having Grade 1 hypertension (71.2%), while the remaining 28.8% had Grade 2 hypertension. The median values for the key biomarkers were within or slightly above their respective normal ranges, indicating a population with mild to moderate alterations in hepatic and haematological parameters at baseline.

Table 1: Baseline Characteristics of the Study Population (N=198)

Characteristic	Value
Demographics	
Age (years), mean ± SD	51.2 ± 10.5
Sex, Male, n (%)	124 (62.6%)
Body Mass Index (kg/m²), mean ± SD	26.8 ± 3.2
Clinical Parameters	
Hypertension Grade 1, n (%)	141 (71.2%)
Hypertension Grade 2, n (%)	57 (28.8%)
Laboratory Parameters, median (IQR)	
ALT (U/L)	44.5 (30.0 – 66.5)
AST (U/L)	66.5 (38.0 – 90.0)
HCT (%)	48.0 (44.0 – 50.0)
ESR (mm/hr)	40.0 (36.0 – 48.0)
CRP (mg/L)	4.5 (2.0 – 7.0)

Data are presented as mean ± standard deviation (SD), n (%), or median (interquartile range, IQR). ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; HCT: Haematocrit; ESR: Erythrocyte Sedimentation Rate; CRP: C-Reactive Protein.

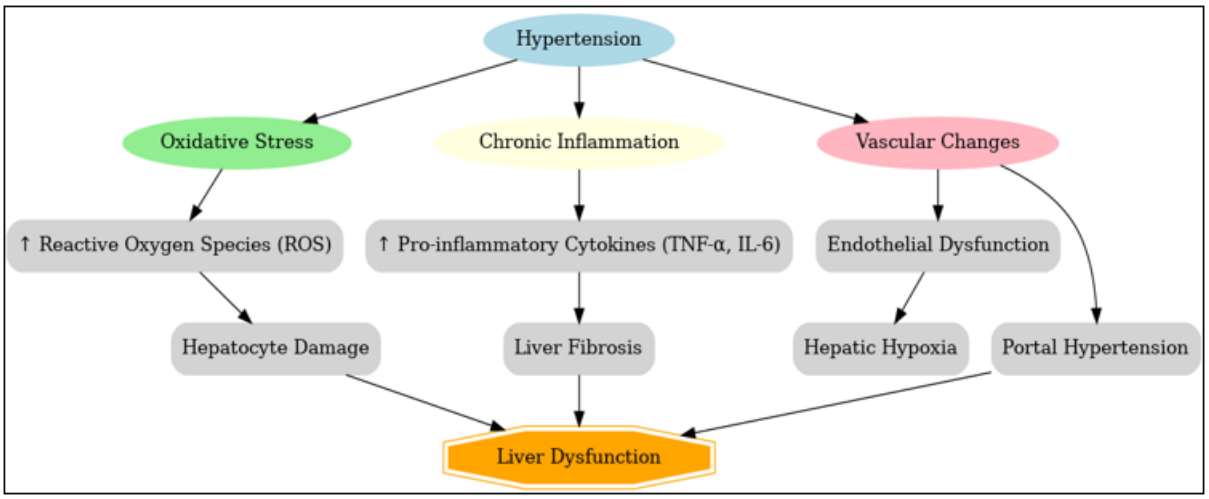
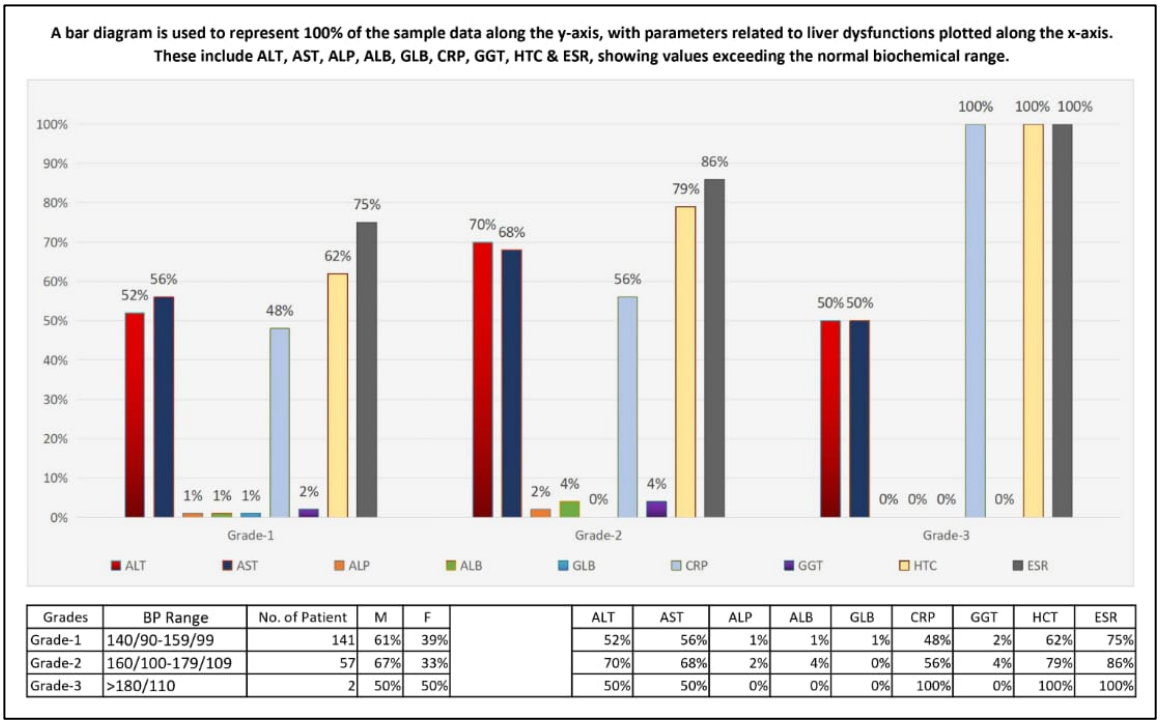


Figure 2: Mechanisms Linking Hypertension to Liver Dysfunction

Direct Correlation Between Haematocrit and Markers of Liver Dysfunction

The primary analysis of the study was to determine the direct linear relationship between haematocrit, a primary determinant of blood density and viscosity, and the liver enzymes ALT and AST. Pearson correlation analysis revealed a statistically significant, positive correlation between HCT and both markers of hepatocyte injury. The correlation between ALT and HCT was r=0.25 (p<0.001), and the correlation between AST and HCT was r=0.30 (p<0.001). These results, summarized in Table 2, indicate that as haematocrit levels increase in this hypertensive cohort, there is a corresponding tendency for liver enzyme levels to also increase.



Grades	BP Range	No. of Patient	M	F	ALT	AST	ALP	ALB	GLB	CRP	GGT	HCT	ESR
Grade-1	140/90-159/99	141	61%	39%	52%	56%	1%	1%	1%	48%	2%	62%	75%
Grade-2	160/100-179/109	57	67%	33%	70%	68%	2%	4%	0%	56%	4%	79%	86%
Grade-3	>180/110	2	50%	50%	50%	50%	0%	0%	0%	100%	0%	100%	100%

Figure 3: Biochemical markers of liver dysfunctions and their percentage (%) of raised beyond the normal reference ranges

Table 2: Pearson Correlation Coefficients for Liver Enzymes and Haematocrit

Variable Pair	Pearson Correlation (r)	p-value	Significance
ALT & HCT	0.25	<0.001	Significant
AST & HCT	0.30	<0.001	Significant

Association of Blood Rheology and Liver Dysfunction Markers with Hypertension Severity

To further investigate the interplay between these markers, the study population was stratified by the severity of hypertension (Grade 1, n=141 vs. Grade 2, n=57). The non-parametric Mann-Whitney U test was used to compare the distribution of biochemical and haematological markers between these two groups. The results, detailed in Table 3, demonstrated that patients with more severe (Grade 2) hypertension had significantly higher levels of markers for both liver dysfunction and altered blood rheology.

Specifically, the median ranks for ALT (p=0.013), AST (p=0.002), HCT (p<0.001), and ESR (p=0.013) were all significantly higher in the Grade 2 hypertension group compared to the Grade 1 group. For CRP, a trend towards higher levels was observed in the Grade 2 group, but this

difference did not reach the threshold for statistical significance (p=0.093). These findings provide strong corroborating evidence that the pathological processes affecting the liver and blood rheology progress in parallel with the worsening of the underlying hypertensive state.

Table 3: Comparison of Biochemical and Haematological Markers Across Hypertension Grades

Variable	Grade 1 HTN (n=141)	Grade 2 HTN (n=57)	p-value
	Median (IQR)	Median (IQR)	
ALT (U/L)	42.0 (29.0 – 60.0)	50.0 (37.0 – 73.0)	0.013
AST (U/L)	62.0 (37.0 – 85.0)	80.0 (39.0 – 120.0)	0.002
HCT (%)	46.0 (44.0 – 50.0)	50.0 (46.0 – 54.0)	<0.001
ESR (mm/hr)	40.0 (33.0 – 46.0)	42.0 (38.0 – 50.0)	0.013
CRP (mg/L)	3.0 (2.0 – 7.0)	6.0 (3.0 – 7.0)	0.093

Data are presented as median (interquartile range, IQR). P-values were calculated using the Mann-Whitney U test. HTN: Hypertension. Statistically significant values are in bold.

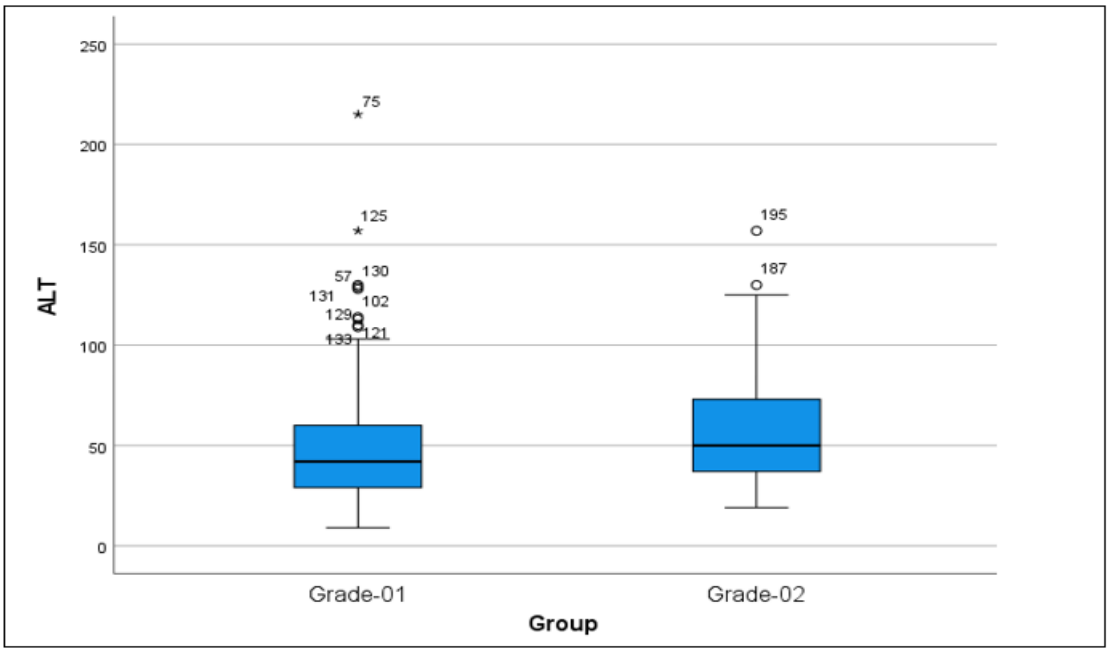


Figure 4: Box Plot for ALT by Group

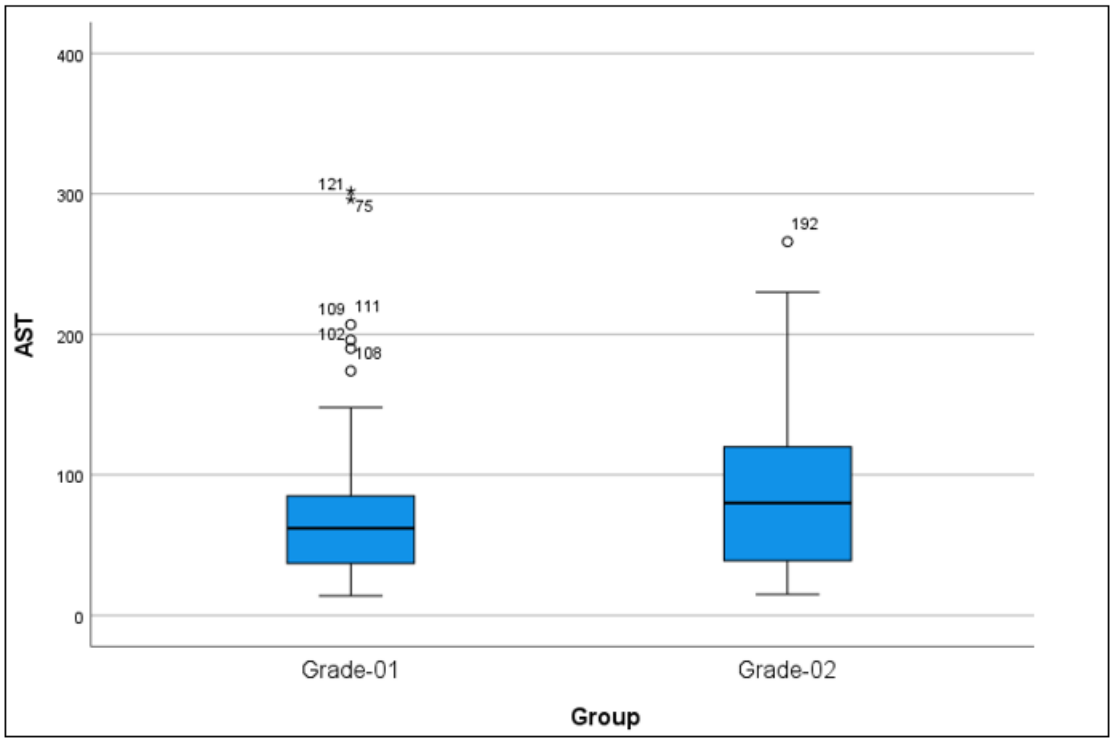


Figure 5: Box Plot for AST by Group

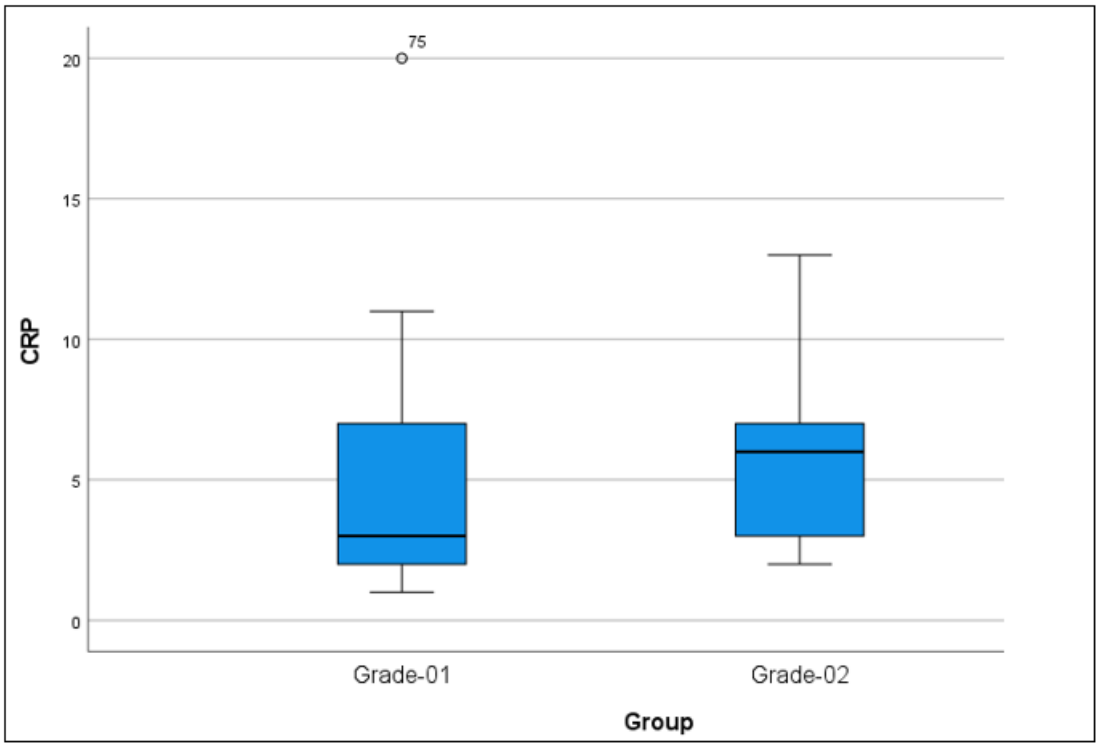


Figure 6: Box Plot for CRP by Group

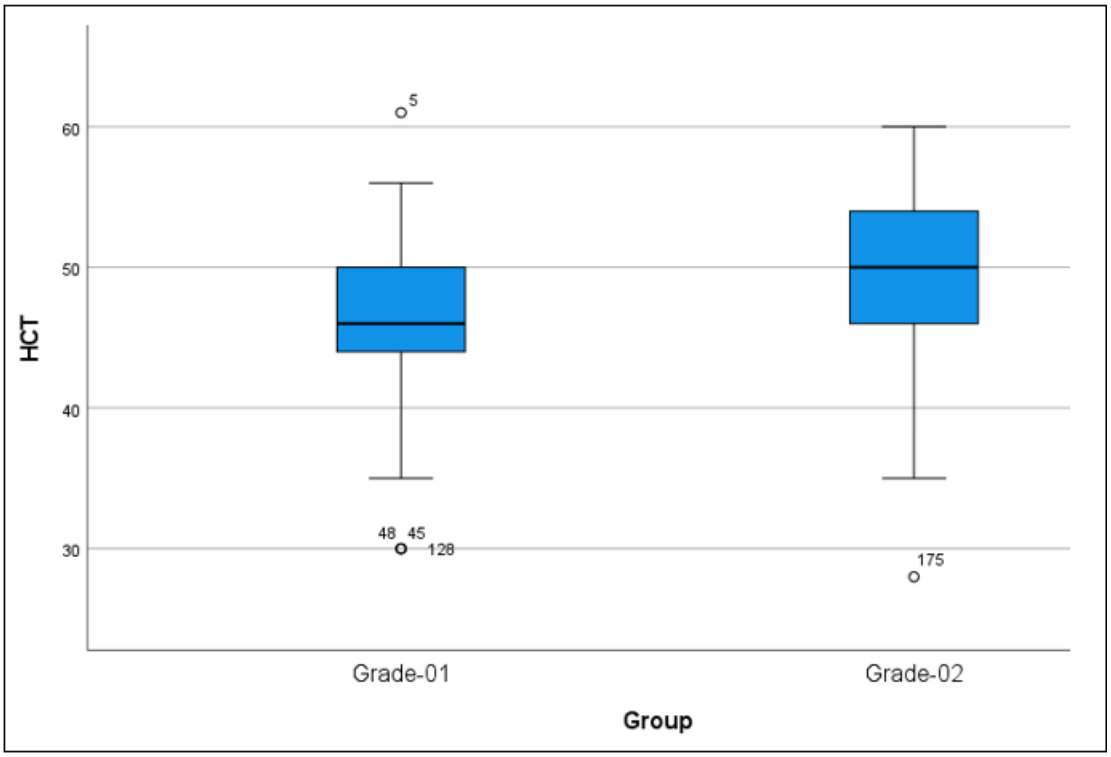


Figure 7: Box Plot for HCT by Group

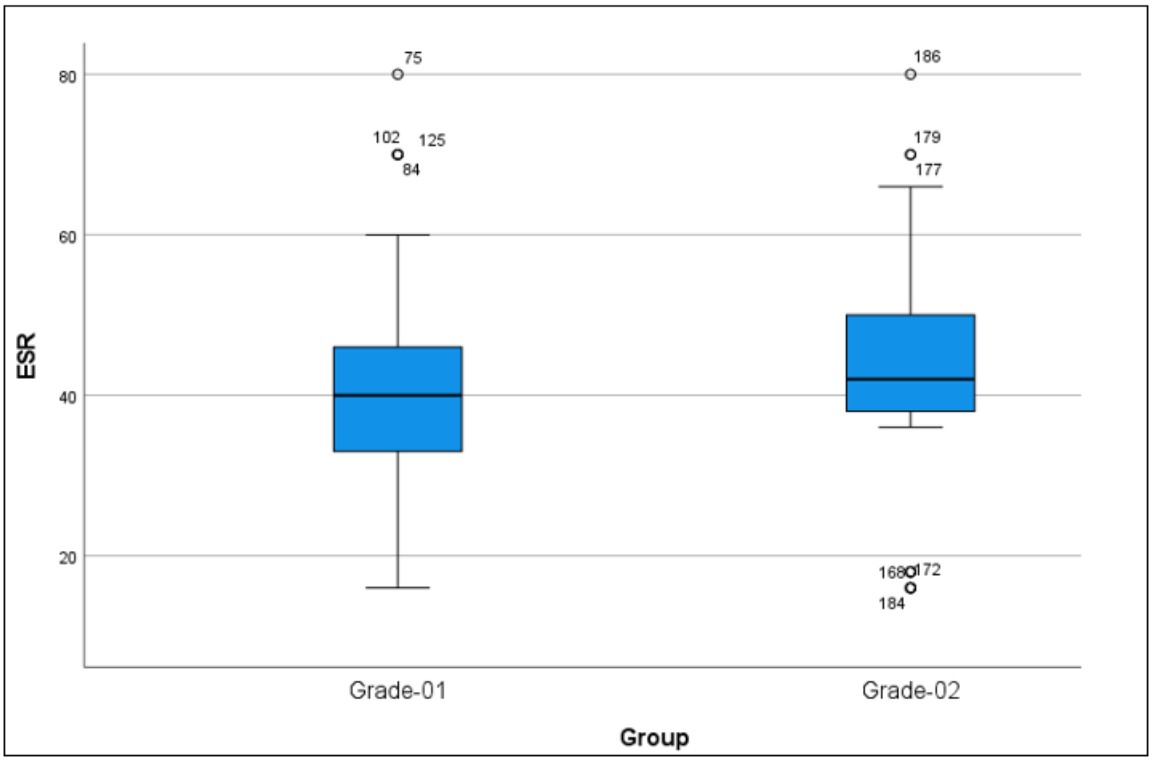


Figure 8: Box Plot for ESR by Group

4. Discussion

This study successfully demonstrated a significant positive association between markers of blood rheology, specifically HCT and ESR, and markers of liver dysfunction, ALT and AST, in a cohort of patients with primary hypertension. The results support the rejection of the null hypothesis and provide evidence for the alternative hypothesis that these two pathological processes are linked. The findings were robust, supported by both direct correlation analysis and indirect evidence from the comparison across hypertension grades, which showed a parallel increase in both sets of markers with worsening disease severity.

Elucidation of Shared Pathophysiological Mechanisms

The principal findings suggest that liver injury and altered blood rheology are not merely parallel, independent consequences of hypertension. Instead, they appear to be deeply interconnected through a common pathophysiological hub: systemic inflammation and endothelial dysfunction. Hypertension, through mechanisms including the activation of the Renin-Angiotensin-Aldosterone System (RAAS) and increased vascular shear stress, is known to induce a chronic, low-grade systemic inflammatory state. [1] This state provides a unifying framework to explain the observed associations.

It is proposed that this inflammatory state, signalled by a tendency for higher CRP levels, drives the observed changes through multiple concurrent pathways. First, pro-inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6) can exert direct cytotoxic effects on hepatocytes, leading to cellular injury and the subsequent release of ALT and AST into the circulation.[1] Second, this same inflammatory milieu stimulates the liver to produce acute-phase reactants, including fibrinogen. The increase in plasma fibrinogen enhances RBC aggregation, which is the direct cause of an elevated ESR.[8,11] Third, chronic inflammation and RAAS activation can modulate both erythropoiesis and plasma volume, contributing to haemoconcentration and an increased HCT, which is the primary determinant of blood viscosity.[1,4] Finally, hypertension-induced endothelial dysfunction can impair hepatic microcirculation, leading to localized hypoxia and oxidative stress, which further exacerbates hepatocyte injury.[1] This unified mechanism offers a compelling explanation for why markers of liver injury and markers of adverse blood rheology rise in concert as the severity of hypertension progresses.

Contextualization with Existing Literature

The results of this study are consistent with and build upon previous research. The observation of elevated ALT and AST levels in hypertensive patients aligns with the findings of Chen et al. (2017), who attributed these changes to hepatic oxidative stress.[1] The association with markers of blood rheology also complements the work of Targher et al. (2014), who established a strong link between hypertension and NAFLD, a condition characterized by both liver enzyme elevation and a pro-inflammatory state that would be expected to alter blood properties.[1]

The finding that CRP levels showed a trend toward significance but did not cross the $p<0.05$ threshold in the group comparison is noteworthy. This may suggest that while inflammation is the underlying driver, the downstream effects (i.e., changes in ALT, AST, ESR, and HCT) are more pronounced or dynamic in their response to increasing hypertension severity between Grade 1 and Grade 2. CRP may function more as a stable marker of the underlying inflammatory state rather than a variable that changes dramatically across these specific hypertension grades in this cohort.

Clinical Implications and Future Directions

The findings of this study have several important clinical implications. They suggest that the assessment of hypertensive patients could be enhanced by considering hepatic and haematological markers in conjunction. For instance, in a hypertensive patient with elevated ALT and AST, a concurrent finding of high HCT and ESR may signify a more severe systemic inflammatory phenotype. This combination of markers could identify a subgroup of patients at a higher overall cardiovascular and metabolic risk, warranting more aggressive management of their blood pressure and associated risk factors.

This study underscores the need for routine liver function monitoring in all hypertensive patients, as elevated enzymes may be an early warning sign of systemic vascular and inflammatory pathology.[1] The results advocate for a more integrated clinical approach where the liver is not viewed in isolation but as part of a multi-organ response to the hypertensive state.

Future research should build upon these findings. Longitudinal studies are essential to establish a causal relationship and to track the progression of liver dysfunction in relation to changes in blood rheology over time. Furthermore, intervention studies could explore whether therapies aimed at improving blood rheology or reducing inflammation have a beneficial impact on liver enzyme levels in hypertensive patients.

5. Strengths and Limitations

This study possesses several strengths, including a well-characterized cohort of patients with primary hypertension, from which major confounders like diabetes and pre-existing liver disease were excluded. The use of a comprehensive set of biochemical and haematological markers and the application of appropriate statistical methods for non-normally distributed data also enhance the validity of the findings.

However, certain limitations must be acknowledged. The primary limitation is the cross-sectional design, which demonstrates association but cannot establish causality.[1] It is not possible to determine whether liver dysfunction contributes to altered rheology, or vice versa, or if both are simply consequences of hypertension. Second, as a single-center study conducted in a specific region of India, the findings may have limited generalizability to other populations with different genetic backgrounds, dietary habits, or environmental exposures. Finally, while major confounders were excluded, other unmeasured factors such as diet, physical activity levels, and genetic predispositions could have influenced the results. [1]

6. Conclusion

This study provides compelling evidence of a significant and positive association between markers of liver dysfunction (ALT, AST) and markers of blood rheology (HCT, ESR) in patients with primary hypertension. This link is amplified by the severity of hypertension and is likely mediated by shared pathophysiological mechanisms rooted in systemic inflammation and endothelial dysfunction. These findings challenge a siloed view of organ-specific damage in hypertension and instead support a model of interconnected systemic pathology. The results advocate for a more integrated clinical approach, where hepatic and haematological markers are considered in conjunction to holistically assess cardiovascular and metabolic risk in hypertensive individuals.

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