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ANALYSIS OF LFT AND COAGULATION PROFILE IN LIVER CIRRHOSIS PATIENTS

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Abstract

Background: *Cirrhosis impairs the liver's synthetic and excretory functions, disrupting both biochemical and coagulation homeostasis. This study evaluated liver function tests (LFTs) and coagulation parameters in patients with cirrhosis and examined their interrelationship.*

Methods: *In this cross-sectional study, 68 adults with clinically or laboratory-confirmed cirrhosis were recruited from the pathology and gastroenterology departments of two tertiary-care hospitals in Lahore, Pakistan, using non-probability purposive sampling. Serum bilirubin, ALT, AST, ALP, albumin, total protein, prothrombin time (PT), INR, activated partial thromboplastin time (APTT), and platelet count were measured. Data were analysed in SPSS v27 using descriptive statistics, Spearman correlation, and linear regression (significance set at $p < 0.05$).*

Results: *The cohort (58.8% female; mean age 53.7 ± 13.2 years) showed elevated AST (96.3 ± 30.4 U/L), ALT (78.0 ± 26.6 U/L) and ALP (225.4 ± 68.1 U/L), with an AST/ALT ratio of 1.40, alongside raised total bilirubin (4.15 ± 1.40 mg/dL) and low albumin (2.66 ± 0.41 g/dL). Coagulation indices were prolonged (PT 21.9 ± 3.5 sec; INR 1.88 ± 0.33; APTT 48.8 ± 6.0 sec) and platelet counts were reduced (99.9 ± 23.5 ×10 ^{9}/L). Non-alcoholic fatty liver disease (36.8%) and alcoholic liver disease (33.8%) were the leading etiologies. Total and direct bilirubin were strongly correlated ($r = 0.909$, $p < 0.01$); PT showed a weak positive correlation with albumin ($r = 0.286$, $p < 0.05$). No biochemical marker independently predicted INR on regression analysis (all $p > 0.05$).*

Conclusion: *Cirrhosis was associated with concurrent hepatocellular injury, impaired synthetic function, and coagulopathy, reinforcing the value of combined LFT and coagulation panels in disease assessment, though no single marker reliably predicted INR in this cohort.*

*The findings suggest that while bilirubin and liver enzymes strongly indicate liver damage, coagulation markers alone are insufficient predictors of cirrhosis severity, **emphasizing the need for** multidimensional clinical assessment.*

INTRODUCTION

Cirrhosis is the end-stage consequence of chronic liver injury, in which progressive fibrosis and regenerative nodularity replace normal hepatic architecture, eventually compromising both metabolic and synthetic liver functions (Ginés et

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al., 2022). Globally, viral hepatitis B and C, alcohol-related liver disease, and non-alcoholic fatty liver disease (NAFLD) are the dominant drivers of cirrhosis, with NAFLD-related disease rising sharply alongside obesity and metabolic syndrome (Younossi, Wong, Anstee, & Henry, 2023; Khare, Liu, Chilambe, & Khare, 2025).

Because the liver synthesises clotting factors II, V, VII, IX and X, hepatic dysfunction commonly produces a coagulopathy reflected in prolonged PT, INR and APTT, compounded by thrombocytopenia secondary to hypersplenism and portal hypertension (Trebicka & Garcia-Tsao, 2025). Liver function tests, including bilirubin, ALT, AST, ALP and albumin, complement coagulation studies by characterising the degree of hepatocellular injury and synthetic failure (Lala, Zubair, & Minter, 2023). Despite their routine clinical use, the relationship between specific LFT abnormalities and coagulation derangement in local cirrhotic populations is incompletely characterised. This study therefore profiled LFTs and coagulation parameters in cirrhotic patients in Lahore and examined the correlation between hepatic biochemical markers and coagulation indices, to support their combined use in assessing disease severity and bleeding risk.

Objectives

1. To characterize patterns of bilirubin and liver enzyme (ALT, AST, ALP) abnormalities in cirrhosis patients.
2. To assess liver synthetic function using PT, APTT, INR, and serum albumin.
3. To examine the relationship between coagulation profile abnormalities and cirrhosis severity.

Research Questions

1. What patterns of bilirubin and liver enzyme (ALT, AST, ALP) abnormalities are observed in cirrhosis patients?
2. How do serum albumin and coagulation indices (PT, APTT, INR) reflect liver synthetic function in this population?
3. Is there a significant correlation between coagulation parameters (PT, INR, platelet count) and cirrhosis severity?

Recent studies highlight a growing global burden of liver diseases, particularly those associated with alcohol use. Alcohol Use Disorder has been increasing significantly, contributing to a parallel rise in Alcohol-Related Liver Disease, especially among younger populations. Despite this trend, many patients do not receive adequate treatment for underlying alcohol dependence, limiting effective disease management and recovery outcomes.

Research further emphasizes that advanced liver conditions often require complex interventions such as Liver Transplantation, though eligibility remains controversial due to relapse risks and insufficient prior therapy. This underscores the need for integrated treatment approaches combining pharmacological, behavioral, and multidisciplinary care strategies.

Epidemiological evidence also indicates a steady increase in liver disease-related mortality. Common contributors include Viral Hepatitis, Non-Alcoholic Fatty Liver Disease, and alcohol-related conditions. Additionally, abnormal liver function tests are prevalent in primary care, affecting up to 20% of the population, often requiring further clinical evaluation to determine etiology.

Overall, the literature supports the need for early diagnosis, comprehensive assessment, and integrated management approaches to reduce disease burden and improve patient outcomes.

2. Materials and Methods

2.1 Study design and setting

This cross-sectional, observational study was conducted over six months (January–June 2026) in the pathology and gastroenterology departments of Sheikh Zayed Hospital and Mayo Hospital, two tertiary-care teaching hospitals in Lahore, Pakistan.

2.2 Participants

Sixty-eight adults (18–78 years) with clinically or laboratory-confirmed cirrhosis were enrolled using non-probability purposive sampling; the sample size was calculated from an estimated prevalence of abnormal LFT/coagulation findings with 90% confidence and a 10% margin of error. Patients with haematological disorders (e.g., haemophilia, leukaemia), those on anticoagulant therapy, individuals with chronic kidney disease, and patients with malignancy or other severe systemic illness were excluded to limit confounding of coagulation parameters.

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2.3 Laboratory assessment

Venous blood (3–5 mL) was drawn under aseptic conditions and processed on automated biochemical and haematological analysers. Parameters assessed were total and direct serum bilirubin, ALT, AST, ALP, albumin, total protein, PT, INR, APTT and platelet count.

2.4 Statistical analysis

Data were analysed in SPSS v27 (IBM Corp.). Continuous variables were summarised as mean ± standard deviation; categorical variables as frequencies and percentages. Normality was assessed with Kolmogorov–Smirnov and Shapiro–Wilk tests. Spearman's rho was used to evaluate correlations between LFT and coagulation parameters, and linear regression assessed whether biochemical markers predicted INR. Statistical significance was set at $p < 0.05$. Ethical approval was obtained from the collaborating hospitals' Institutional Review Boards, and written informed consent was secured from all participants.

Results

4.1 Descriptive Statistics of Clinical Parameters

Table 4.1: ***Descriptive Statistics of Liver Function and Coagulation Parameters (N = 68)***

Variable	Min	Max	M	SD
ALT (U/L)	35	120	78.03	26.61
AST (U/L)	41	149	96.31	30.38
ALP (U/L)	120	350	225.35	68.07
Total Bilirubin (mg/dL)	1.62	6.36	4.15	1.40
Direct Bilirubin (mg/dL)	0.79	4.33	2.36	0.94
Albumin (g/dL)	2.01	3.38	2.66	0.41
Total Protein (g/dL)	5.03	6.76	5.89	0.46
PT (sec)	16.1	28.0	21.87	3.49
INR	1.31	2.45	1.88	0.33
APTT (sec)	39.0	59.6	48.79	6.05
Platelet Count ($\times 10^9$/L)	64	140	99.93	23.54
AST/ALT Ratio	0.39	3.56	1.40	0.69
Indirect Bilirubin	0.58	3.60	1.79	0.67
Age (years)	30	75	53.72	13.20

The descriptive findings indicate **elevated liver enzymes (AST, ALT, ALP)** and **increased bilirubin levels**, confirming hepatocellular damage and cholestasis in cirrhosis patients. Additionally, **low albumin levels and prolonged PT/INR** reflect impaired synthetic liver function, directly addressing **Objective 1 and 2**.

4.2 Demographic Characteristics

Table 4.2: *Gender Distribution of Participants*

Gender	Frequency	Percentage
Male	28	41.2%
Female	40	58.8%

The sample consisted predominantly of females (58.8%), which should be considered when interpreting biochemical variations.

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4.3 Etiological Profile of Cirrhosis

Table 4.3: ***Etiology of Liver Cirrhosis***

Etiology	Frequency	Percentage
Non-Alcoholic Fatty Liver Disease	25	36.8%
Alcoholic Liver Disease	23	33.8%
Hepatitis B	11	16.2%
Hepatitis C	9	13.2%

NAFLD emerged as the leading cause, followed closely by alcoholic liver disease, highlighting the **dual metabolic and lifestyle burden** of cirrhosis.

4.4 Normality Assessment

Table 4.4: ***Tests of Normality (Shapiro–Wilk)***

Variable	Statistic	p
Age	.942	.003
ALT	.941	.003
AST	.956	.017
ALP	.949	.007
Total Bilirubin	.934	.001
Albumin	.938	.002
PT	.951	.009
INR	.962	.035
Platelet Count	.936	.002

Most variables violated normality assumptions ($p < .05$), justifying the use of **non-parametric analysis (Spearman's rho)**. This strengthens the methodological rigor of the study.

4.5 Correlation Analysis

Table 4.5: ***Spearman Correlation Between Liver Function and Coagulation Parameters***

Variables	1	2	3	4
1. Total Bilirubin	—			
2. Direct Bilirubin	.91**	—		
3. Albumin	.11	.22	—	
4. PT	-.06	-.19	.29*	—

Note. *p < .05, **p < .01

A **very strong positive correlation** between total and direct bilirubin ($r = .91$, $p < .01$) confirms consistent bilirubin elevation patterns **(RQ1)**. A **significant positive relationship between PT and albumin ($r = .29$, $p < .05$)** indicates interaction between liver synthetic and coagulation functions **(RQ2)**. Other correlations were weak, suggesting **multifactorial disease progression** rather than simple linear associations **(RQ3)**.

4.6 Regression Analysis

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Table 4.6: ***ANOVA for Regression Model Predicting INR***

Source	SS	df	MS	F	p
Regression	0.466	5	0.093	0.86	.515
Residual	6.732	62	0.109		

The regression model was **not statistically significant (p = .515)**, indicating that selected predictors do not collectively explain variation in INR.

Table 4.7: ***Regression Coefficients Predicting INR***

Predictor	B	SE	β	t	p
Total Bilirubin	.033	.029	.143	1.15	.253
Albumin	.040	.101	.051	0.40	.690
AST/ALT Ratio	.024	.061	.050	0.39	.697
Platelet Count	.000	.002	.017	0.13	.895
ALP	-.001	.001	-.187	-1.48	.143

No individual predictor significantly influenced INR ($p > .05$), suggesting that coagulation dysfunction in cirrhosis is complex and not driven by single biochemical markers.

Discussion

This cohort demonstrated the classic biochemical signature of cirrhosis: elevated transaminases and ALP indicating hepatocellular injury and cholestasis, raised bilirubin reflecting impaired excretory capacity, and low albumin denoting diminished synthetic function, the latter consistent with the known association between hypoalbuminaemia, ascites, and poorer prognosis in cirrhosis (Trebicka & Garcia-Tsao, 2025). An AST/ALT ratio exceeding 1 is frequently described in advanced and alcohol-related liver injury, in keeping with the substantial proportion of alcoholic liver disease in this sample.

Prolongation of PT, INR and APTT, together with reduced platelet counts, supports impaired synthesis of vitamin K–dependent clotting factors and hypersplenism-related thrombocytopenia, both well-documented features of cirrhotic coagulopathy that heighten bleeding risk (Chen et al., 2023; Trebicka & Garcia-Tsao, 2025). The strong correlation between total and direct bilirubin reinforces their shared dependence on hepatocyte conjugating and excretory capacity. The weak positive correlation between PT and albumin, while statistically significant, was modest and should be interpreted cautiously; it does not establish that albumin meaningfully predicts coagulation status, and the absence of a significant regression model for INR suggests that no single biochemical marker captured the multifactorial basis of coagulopathy in this sample a finding that itself underscores the value of interpreting LFTs and coagulation indices as a composite panel rather than relying on isolated parameters.

The predominance of NAFLD and alcoholic liver disease as etiologies, ahead of viral hepatitis, mirrors a broader regional and global shift toward metabolic and alcohol-related drivers of chronic liver disease (Khare et al., 2025; Tapper & Parikh, 2023), with implications for preventive screening and lifestyle-focused intervention.

This study has limitations. The single-timepoint cross-sectional design and modest sample size (n = 68) drawn from two tertiary centres in one city limit generalisability and preclude causal inference between coagulopathy and liver injury severity. Results may also be influenced by inter-laboratory variation in assay methods and patient clinical status at sampling. Larger, multi-centre, longitudinal studies incorporating severity scores such as Child–Pugh or MELD are warranted to validate these associations.

Conclusion

In this cohort of cirrhotic patients, elevated liver enzymes and bilirubin alongside reduced albumin confirmed concurrent hepatocellular injury and synthetic dysfunction, while prolonged PT, INR and APTT with thrombocytopenia confirmed clinically relevant coagulopathy. NAFLD and

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alcoholic liver disease emerged as leading etiologies, highlighting the growing contribution of metabolic and lifestyle factors to cirrhosis burden. Although no single biochemical marker predicted INR, the combined use of LFTs and coagulation profiles remains a practical, accessible tool for assessing liver damage severity and bleeding risk, supporting routine, periodic monitoring in cirrhotic patients.

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