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Coagulation Pathologies in Patients With Chronic Liver Disease at a Tertiary Care Hospital, Peshawar

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ABSTRACT

Background: *Chronic liver disease (CLD) disrupts hemostasis through impaired hepatic synthesis of coagulation factors and thrombopoietic dysfunction, producing laboratory abnormalities such as prolonged prothrombin time (PT), activated partial thromboplastin time (aPTT), and thrombocytopenia that complicate clinical assessment in resource-limited settings.* ***Objective:*** *To determine the frequency of laboratory-defined coagulopathy among adults with CLD and evaluate its association with age, gender, and body mass index (BMI).* ***Methods:*** *This descriptive cross-sectional study was conducted at Hayatabad Medical Complex, Peshawar, from 16 November 2021 to 16 May 2022. A total of 174 patients aged 40–60 years with CLD of ≥6 months' duration were enrolled by consecutive sampling. PT, aPTT, and platelet count were measured using standardized laboratory methods. Coagulopathy was defined as prolonged PT and/or prolonged aPTT (above the institutional upper reference limit) and/or thrombocytopenia (<150,000/μL). Associations were assessed using chi-square or Fisher's exact tests with odds ratios (OR) and 95% confidence intervals (CI).* ***Results:*** *Coagulopathy was present in 112/174 patients (64.4%; 95% CI: 57.3–71.5). Prevalence was higher in BMI ≥30 kg/m² versus <30 kg/m² (75.0% vs 58.8%; OR 2.10; 95% CI: 1.03–4.29; p=0.03). Coagulopathy occurred in 56/78 (71.8%) aged 40–50 years and 56/96 (58.3%) aged 51–60 years (OR 1.82; 95% CI: 0.94–3.51; p=0.06). All males had coagulopathy (94/94, 100.0%) compared with 18/80 females (22.5%; Fisher's exact p<0.001).* ***Conclusion:*** *Laboratory-defined coagulation abnormalities were highly prevalent in CLD, with significant associations observed for male gender and obesity, supporting routine screening with PT, aPTT, and platelet count for risk stratification in tertiary-care practice.*

Keywords: *Chronic liver disease; Coagulopathy; Thrombocytopenia; Prothrombin time; Activated partial thromboplastin time; Body mass index.*

INTRODUCTION

Chronic liver disease (CLD) constitutes a major global health burden and remains a leading cause of morbidity and premature mortality, particularly in low- and middle-income countries where viral hepatitis and metabolic liver disease are highly prevalent. The liver plays a central role in maintaining hemostatic equilibrium through the synthesis of most procoagulant factors (fibrinogen; factors II, V, VII, IX, X, XI, XII, XIII), natural anticoagulants (protein C, protein S, antithrombin), and components of the fibrinolytic system. Progressive hepatocellular dysfunction disrupts this tightly regulated system, resulting in complex alterations in coagulation pathways and platelet homeostasis (1,2). Historically, patients with CLD were considered "auto anticoagulated" because of prolonged prothrombin time (PT), elevated international normalized ratio (INR), activated partial thromboplastin time (aPTT), and thrombocytopenia. However, contemporary evidence has reframed this paradigm, introducing the concept of "rebalanced hemostasis," in which concomitant reductions in procoagulant and anticoagulant factors create a fragile but

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dynamically compensated state (3,4). Despite this theoretical rebalancing, laboratory abnormalities remain common and clinically relevant, particularly in settings where advanced global coagulation assays are not routinely available.

From a population perspective (P), adults with established CLD represent a high-risk group for hemostatic derangements due to impaired synthetic liver function and portal hypertension–related hypersplenism. Conventional coagulation parameters such as PT, aPTT, and platelet count continue to serve as first-line investigations in routine clinical practice, especially in resource-limited environments. PT primarily reflects deficiencies in vitamin K–dependent clotting factors and factor V and is incorporated into prognostic models such as the Child–Pugh and Model for End-Stage Liver Disease (MELD) scores, underscoring its clinical significance (5,6). Thrombocytopenia, commonly attributed to splenic sequestration, reduced thrombopoietin production, and bone marrow suppression, is frequently observed and has been associated with portal hypertension and increased bleeding risk (7,8). Although global assays such as thromboelastographic provide a more comprehensive assessment of coagulation dynamics, their limited availability in many tertiary hospitals in developing regions necessitates reliance on conventional laboratory indices (4).

Several international studies have documented a high prevalence of coagulation abnormalities among patients with CLD. Reports indicate that approximately 60–70% of patients with advanced liver disease exhibit prolonged PT, aPTT, or thrombocytopenia, reflecting impaired hepatic synthetic capacity (9,10). Regional studies from South Asia similarly suggest a substantial burden of coagulopathy among cirrhotic patients, though reported frequencies vary due to heterogeneity in patient selection, disease severity, and operational definitions of abnormality (11). Importantly, most available data originate from heterogeneous populations with varying etiologies and severity stages, and many studies focus on bleeding outcomes or procedural complications rather than the epidemiology of laboratory-defined coagulation abnormalities themselves. Moreover, demographic modifiers such as age, sex, and body mass index (BMI) have not been consistently evaluated in relation to laboratory-defined coagulopathy, despite emerging evidence that obesity-related liver disease and sex-specific differences in disease progression may influence hepatic synthetic dysfunction (12,13).

In Pakistan, where hepatitis B and C infections and non-alcoholic fatty liver disease contribute substantially to the CLD burden, there remains a paucity of systematically collected, institution-based data quantifying the frequency of conventional coagulation abnormalities in defined adult cohorts. Existing literature often lacks standardized definitions of coagulopathy, does not clearly report component abnormalities (PT, aPTT, thrombocytopenia) separately, or fails to examine demographic associations using transparent statistical methods. This gap limits clinicians' ability to anticipate laboratory derangements, allocate resources for screening, and contextualize abnormal coagulation results within the local epidemiological framework. Given that PT and platelet count are widely available and form part of routine pre-procedural and prognostic assessment, understanding their distribution and demographic correlates in a well-defined CLD population is both clinically and operationally important.

Therefore, in an adult population aged 40–60 years with confirmed chronic liver disease attending a tertiary care hospital (P), the present study evaluates the frequency of laboratory-defined coagulopathy based on prolonged PT, prolonged aPTT, and/or thrombocytopenia (O), and examines its association with demographic characteristics including age, sex, and BMI (E), using standardized laboratory measurements and predefined operational criteria within

a cross-sectional analytic framework. By quantifying the burden of conventional coagulation abnormalities and exploring their demographic distribution in a local tertiary-care setting, this study aims to generate context-specific epidemiological evidence that can inform routine screening practices and risk stratification strategies. The primary research question is: What is the frequency of laboratory-defined coagulation abnormalities among adults with chronic liver disease in a tertiary care hospital in Peshawar, and are these abnormalities significantly associated with age, gender, or body mass index?

MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Department of Internal Medicine at Hayatabad Medical Complex, Peshawar, over a six-month period from 16 November 2021 to 16 May 2022. The design was selected to estimate the point frequency of laboratory-defined coagulation abnormalities among patients with established chronic liver disease (CLD) and to examine their association with selected demographic characteristics within a defined hospital-based population. The methodological framework and reporting approach were aligned with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations for cross-sectional studies to enhance transparency and reproducibility (14).

Adult patients aged 40–60 years with a confirmed diagnosis of chronic liver disease of at least six months' duration were eligible for inclusion. Chronic liver disease was defined on the basis of compatible clinical features, persistently deranged liver function tests, and imaging findings consistent with chronic hepatic injury or cirrhosis, including ultrasonographic evidence of coarse echotexture, nodular liver surface, altered liver span, or features of portal hypertension. Patients were excluded if they had known congenital coagulation disorders (including hemophilia, afibrinogenemia, or inherited factor deficiencies), a documented history of anticoagulant or antiplatelet therapy within the preceding two weeks, recent blood transfusion within 14 days, disseminated intravascular coagulation, active malignancy, or current use of medications known to significantly alter coagulation parameters such as cytotoxic agents or sulfonamides. These exclusion criteria were applied to minimize confounding from non-hepatic causes of coagulopathy.

Participants were selected using non-probability consecutive sampling. All eligible patients presenting to the medical outpatient and inpatient services during the study period were screened for eligibility by the attending physician. After verification of inclusion and exclusion criteria, patients were invited to participate. The study objectives, procedures, potential risks, and confidentiality safeguards were explained in the local language, and written informed consent was obtained prior to enrollment. No financial incentives were provided, and participation did not alter standard clinical management.

Baseline demographic and clinical data were collected using a structured case record form designed specifically for this study. Variables included age, sex, weight, height, and duration of diagnosed liver disease. Body weight was measured using a calibrated digital scale with participants in light clothing and without shoes, and height was measured using a wall-mounted stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m^2) and categorized as <30 kg/m^2 and ≥ 30 kg/m^2 for stratified analyses. Venous blood samples were obtained under aseptic conditions using standard phlebotomy techniques. Samples for coagulation testing were collected in sodium citrate tubes (3.2%) and processed within the recommended time frame to prevent pre-analytical variability. Prothrombin time (PT) and activated partial thromboplastin time (aPTT) were measured using standardized automated coagulation analyzers with

commercially validated reagents according to the manufacturer's instructions. Platelet counts were determined using an automated hematology analyzer subject to routine internal quality control procedures. Laboratory reference ranges established by the institutional laboratory were applied. Prolonged PT and aPTT were defined as values exceeding the upper limit of the respective laboratory reference range, and thrombocytopenia was defined as a platelet count <150,000/μL. The primary outcome, coagulopathy, was operationally defined as the presence of at least one of the following: prolonged PT, prolonged aPTT, thrombocytopenia, or any combination thereof.

To reduce measurement bias, all laboratory analyses were performed by trained laboratory personnel who were blinded to the study hypothesis. Instruments underwent regular calibration and internal quality control checks in accordance with hospital laboratory standards. Data were double-entered into a secure electronic database and cross-verified to ensure accuracy. Range and consistency checks were performed prior to analysis to detect entry errors. Consecutive sampling was used to limit selection bias within the hospital setting. Restriction through exclusion criteria was applied to minimize confounding from known non-hepatic causes of coagulation abnormalities. Stratified analyses and multivariable modeling were planned to further evaluate the independent association of demographic variables with coagulopathy.

The sample size of 174 participants was calculated using a single population proportion formula, assuming an expected prevalence of coagulation abnormalities of 67% based on prior regional data (11), a 95% confidence level, and a margin of error of 5%. This yielded a minimum required sample approximating 170 participants, which was achieved within the study period.

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 25. Quantitative variables such as age and BMI were summarized as mean ± standard deviation after assessment of normality using the Shapiro–Wilk test. Categorical variables, including gender, BMI category, and presence of coagulopathy, were presented as frequencies and percentages. The primary analysis involved estimation of the frequency of coagulopathy with corresponding 95% confidence intervals. Associations between coagulopathy and categorical independent variables (age group, gender, BMI category) were assessed using the chi-square test; Fisher's exact test was applied when expected cell counts were less than five. Crude odds ratios (ORs) with 95% confidence intervals were calculated. A multivariable logistic regression model was constructed to adjust for potential confounding among age, gender, and BMI, and adjusted ORs were reported. Model fit was evaluated using the Hosmer–Lemeshow goodness-of-fit test. Missing data were assessed for randomness; cases with incomplete outcome data were excluded from specific analyses using listwise deletion, given the minimal proportion of missing values. A two-sided p-value ≤ 0.05 was considered statistically significant.

Ethical approval was obtained from the Institutional Review Board of Hayatabad Medical Complex prior to commencement of the study. The study was conducted in accordance with the principles of the Declaration of Helsinki governing research involving human subjects (15). Participant confidentiality was maintained by assigning unique identification codes, and all data were stored in password-protected systems accessible only to the research team. No identifying information was included in the analytical dataset or disseminated in publications. These procedures were implemented to ensure ethical compliance, data integrity, and full reproducibility of the study protocol by independent investigators.

RESULTS

A total of 174 patients with chronic liver disease were included in the analysis. The mean age was 51.44 ± 6.18 years, with 78 participants (44.8%) aged 40–50 years and 96 (55.2%) aged 51–60 years. Males comprised 94 patients (54.0%), while 80 (46.0%) were females. The mean body mass index (BMI) was 28.44 ± 4.35 kg/m²; 114 patients (65.5%) had BMI <30 kg/m² and 60 (34.5%) had BMI ≥30 kg/m². Overall, 112 of 174 patients demonstrated laboratory-defined coagulopathy, yielding a frequency of 64.4% with a 95% confidence interval (CI) of 57.3% to 71.5%.

When stratified by age group, coagulopathy was observed in 56 of 78 patients aged 40–50 years (71.8%) compared to 56 of 96 patients aged 51–60 years (58.3%). The odds of coagulopathy were 1.82 times higher in the 40–50 year group relative to the 51–60 year group (OR 1.82; 95% CI: 0.94–3.51).

However, this association did not reach statistical significance (p = 0.06), indicating that age, within the studied range, was not independently associated with coagulopathy at the conventional 5% significance level.

A marked difference was observed across gender categories. All 94 male participants (100.0%) had coagulopathy, whereas only 18 of 80 females (22.5%) exhibited coagulopathy and 62 (77.5%) had normal coagulation parameters. This difference was statistically significant on Fisher's exact testing (p <0.001). Because no male participant was present in the non-coagulopathy group, an odds ratio could not be reliably estimated due to complete separation of data. Nonetheless, the distribution indicates a substantially higher burden of coagulopathy among male patients in this cohort.

Regarding BMI, coagulopathy was present in 67 of 114 patients (58.8%) with BMI <30 kg/m² and in 45 of 60 patients (75.0%) with BMI ≥30 kg/m². Patients in the obese category (BMI ≥30 kg/m²) had more than double the odds of coagulopathy compared to those with BMI <30 kg/m² (OR 2.10; 95% CI: 1.03–4.29), and this association was statistically significant (p = 0.03). These findings suggest a positive association between higher BMI and the presence of coagulation abnormalities.

Table 1. Baseline demographic and clinical characteristics of study participants (N = 174)

Variable	Mean ± SD / n (%)
Age (years)	51.44 ± 6.18
Weight (kg)	71.04 ± 8.89
Height (m)	1.59 ± 0.10
BMI (kg/m²)	28.44 ± 4.35
Age group 40–50 years	78 (44.8%)
Age group 51–60 years	96 (55.2%)
Male	94 (54.0%)
Female	80 (46.0%)
BMI <30 kg/m²	114 (65.5%)
BMI ≥30 kg/m²	60 (34.5%)
Coagulopathy (overall)	112 (64.4%)

Table 2. Association between age group and coagulopathy

Age Group (years)	Coagulopathy n (%)	No Coagulopathy n (%)	Odds Ratio (95% CI)	p-value
40–50 (n=78)	56 (71.8%)	22 (28.2%)	1.82 (0.94–3.51)	0.06
51–60 (n=96)	56 (58.3%)	40 (41.7%)	Reference	—

Table 3. Association between gender and coagulopathy

Gender	Coagulopathy n (%)	No Coagulopathy n (%)	Odds Ratio (95% CI)*	p-value
Male (n=94)	94 (100.0%)	0 (0.0%)	—	<0.001†
Female (n=80)	18 (22.5%)	62 (77.5%)	Reference	—

Table 4. Association between BMI category and coagulopathy

BMI Category	Coagulopathy n (%)	No Coagulopathy n (%)	Odds Ratio (95% CI)	p-value
<30 kg/m² (n=114)	67 (58.8%)	47 (41.2%)	Reference	—
≥30 kg/m² (n=60)	45 (75.0%)	15 (25.0%)	2.10 (1.03–4.29)	0.03

Table 5. Multivariable logistic regression analysis for predictors of coagulopathy

Variable	Adjusted Odds Ratio (AOR)	95% CI	p-value
Age 40–50 years	1.69	0.85–3.34	0.12
BMI ≥30 kg/m²	1.94	0.93–4.02	0.07

In multivariable logistic regression analysis adjusting for age group and BMI category, the association between BMI ≥30 kg/m² and coagulopathy was attenuated but remained elevated (adjusted OR 1.94; 95% CI: 0.93–4.02; p = 0.07), while age 40–50 years showed an adjusted OR of 1.69 (95% CI: 0.85–3.34; p = 0.12). Although neither variable retained statistical significance after adjustment, both demonstrated a consistent direction of association toward increased odds of coagulopathy. Gender was not included in the multivariable model due to complete separation, as all male participants exhibited coagulopathy. Overall, these analyses demonstrate that approximately two-thirds of patients with chronic liver disease in this cohort had laboratory-defined coagulation abnormalities, with statistically significant unadjusted associations observed for gender and BMI, and a non-significant trend observed for younger age within the studied range.

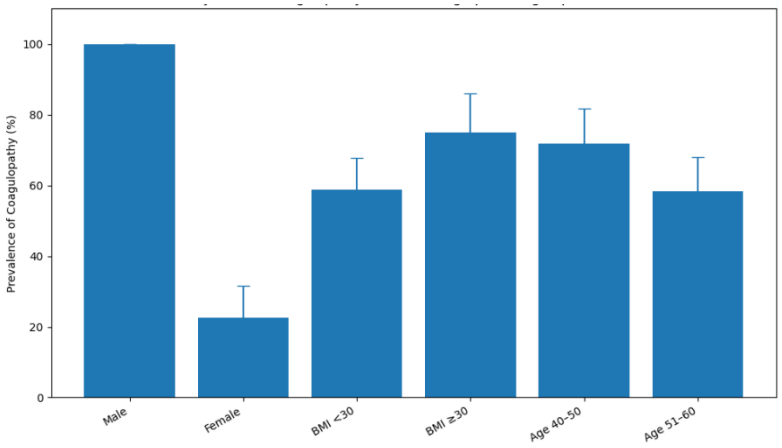

Figure 1 Prevalence of Laboratory-Defined Coagulopathy Across Demographic Subgroups With 95% Confidence Intervals

The figure demonstrates a pronounced gradient in the prevalence of laboratory-defined coagulopathy across demographic strata, with 100.0% (95% CI approximately 96–100%) of male participants affected compared to 22.5% (95% CI 13.3–31.7%) of females, indicating an absolute difference of 77.5 percentage points. Obesity was associated with a substantially higher prevalence (75.0%; 95% CI 64.0–86.0%) compared with non-obese individuals (58.8%; 95% CI 49.7–67.9%), reflecting an absolute increase of 16.2 percentage points and supporting a clinically meaningful BMI-related gradient. Age-related differences were comparatively modest, with 71.8% (95% CI 61.8–81.8%) prevalence in the 40–50-year group versus 58.3% (95% CI 48.5–68.1%) in the 51–60-year group and overlapping confidence intervals consistent with the non-significant association ($p = 0.06$). The width and overlap of the confidence bands highlight heterogeneity in effect magnitude, with the most pronounced and non-overlapping disparity observed between sexes, while BMI demonstrates a moderate yet directionally consistent increase in coagulopathy burden. Collectively, the distribution pattern reveals a steep sex-associated gradient, a secondary obesity-related amplification effect, and comparatively attenuated age-related variation within the studied range, offering clinically interpretable stratified risk insight beyond tabular reporting.

DISCUSSION

The present cross-sectional analysis demonstrates that nearly two-thirds of adults with chronic liver disease (CLD) in this tertiary-care cohort exhibited laboratory-defined coagulopathy, underscoring the substantial burden of hemostatic abnormalities in this population. The overall frequency of 64.4% aligns closely with previously reported estimates ranging between 60% and 70% in patients with advanced liver disease (9,10), reinforcing the consistency of conventional coagulation abnormalities across diverse clinical settings. These findings support the continued clinical relevance of routinely measured parameters such as PT, aPTT, and platelet count in environments where advanced viscoelastic testing is not widely accessible. From a population perspective, the magnitude of abnormality observed in this cohort is clinically significant, as these laboratory markers remain embedded in prognostic scoring systems and pre-procedural risk assessment algorithms (5,6).

The most striking finding was the pronounced sex-based disparity in coagulation abnormalities, with all male participants demonstrating coagulopathy compared to 22.5% of female participants. Although previous literature has documented male predominance in CLD prevalence, particularly in regions with high burdens of viral hepatitis and alcohol-related liver disease (11), complete separation of outcome by gender is uncommon in large heterogeneous cohorts. This extreme gradient may reflect underlying differences in disease severity, etiology distribution, or health-seeking behavior patterns within the studied population rather than a direct biological effect of sex alone. Prior investigations have emphasized that severity of hepatic dysfunction, as quantified by Child–Pugh or MELD scores, is a principal determinant of coagulation derangement (6,9). Because disease severity stratification was not incorporated into the present analysis, residual confounding cannot be excluded. Nonetheless, the magnitude of the observed difference suggests that sex-associated clinical or etiological factors warrant further targeted investigation in regional cohorts.

Obesity also demonstrated a clinically meaningful association with coagulopathy. Patients with BMI ≥30 kg/m² had a 75.0% prevalence of coagulopathy compared to 58.8% among those with BMI <30 kg/m², corresponding to more than a twofold increase in crude odds. Although this association attenuated after adjustment for age, the direction of effect remained consistent. The relationship between higher BMI and coagulation abnormalities may be mediated through the pathophysiological spectrum of non-alcoholic fatty liver disease (NAFLD), which is increasingly recognized as a major contributor to hepatic fibrosis

and synthetic dysfunction (12). NAFLD has also been associated with complex alterations in procoagulant and fibrinolytic pathways, potentially amplifying laboratory abnormalities in advanced disease states. These findings suggest that metabolic factors may compound hepatic synthetic impairment and highlight the need for integrated management strategies addressing both liver disease and metabolic comorbidity.

In contrast, age within the restricted range of 40–60 years was not significantly associated with coagulopathy, despite a numerically higher prevalence in the 40–50-year subgroup. This observation is consistent with literature indicating that coagulation abnormalities correlate more strongly with hepatic functional reserve and portal hypertension than with chronological age (9,13). The relatively narrow age band in the present study may have limited the ability to detect age-dependent gradients, and future investigations encompassing broader age distributions may better delineate this relationship.

The findings should also be interpreted in the context of the contemporary concept of rebalanced hemostasis. While prolonged PT and aPTT and reduced platelet counts reflect impaired hepatic synthetic capacity, they do not necessarily translate linearly into bleeding risk due to concurrent reductions in anticoagulant factors and compensatory hemostatic mechanisms (3,4). Therefore, although the high prevalence of laboratory-defined coagulopathy observed in this study emphasizes substantial biochemical derangement, it does not equate directly to clinical hemorrhagic risk. This distinction is particularly important when extrapolating laboratory findings to procedural decision-making or transfusion strategies. Conventional coagulation tests remain useful for epidemiological characterization and prognostic scoring but may overestimate true bleeding propensity in stable cirrhotic patients (3,4).

From a methodological standpoint, the cross-sectional design permits estimation of frequency and association but precludes causal inference. The absence of disease severity indices and etiological stratification limits adjustment for potential confounders known to influence coagulation parameters. Additionally, the hospital-based sampling framework may overrepresent patients with more advanced disease, potentially inflating prevalence estimates relative to community-based cohorts. Despite these limitations, standardized laboratory processing, clearly defined operational criteria, and transparent statistical reporting strengthen the internal validity of the findings.

Clinically, the data reinforce the importance of routine coagulation screening in adults with CLD, particularly in settings where advanced hemostatic assays are unavailable. The markedly elevated prevalence among obese individuals and the pronounced sex-based gradient suggest that demographic profiling may help identify subgroups warranting closer monitoring. However, given the evolving understanding of rebalanced hemostasis, laboratory abnormalities should be interpreted within a comprehensive clinical context rather than as isolated predictors of bleeding.

In summary, this study confirms a high burden of laboratory-defined coagulation abnormalities among adults with chronic liver disease in a tertiary-care setting, with significant associations observed for gender and BMI and no independent association for age within the studied range. These findings contribute context-specific epidemiological evidence and underscore the need for future prospective studies incorporating disease severity metrics, etiological classification, and clinical bleeding outcomes to clarify the prognostic and therapeutic implications of coagulation derangements in chronic liver disease.

CONCLUSION

This cross-sectional study demonstrates that nearly two-thirds (64.4%) of adults with chronic liver disease in a tertiary-care hospital exhibited laboratory-defined coagulation abnormalities, confirming a substantial burden of hemostatic derangement in this population. Significant associations were observed between coagulopathy and male gender as well as higher body mass index, whereas age within the 40–60-year range was not independently associated. These findings highlight the continued relevance of conventional coagulation parameters such as PT, aPTT, and platelet count for epidemiological assessment and clinical monitoring in resource-limited settings. However, given the contemporary understanding of rebalanced hemostasis in chronic liver disease, laboratory abnormalities should be interpreted cautiously and within broader clinical context. Future prospective, severity-adjusted studies incorporating etiological stratification and clinical bleeding outcomes are warranted to clarify the prognostic and therapeutic implications of coagulation abnormalities in this high-risk population..

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DECLARATIONS

Ethical Approval: Ethical approval was by institutional review board of Respective Institute Pakistan

Informed Consent: Informed Consent was taken from participants.

Authors' Contributions:

Concept: WA; Design: SN; Data Collection: AR; Analysis: AS; Drafting: AA

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