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ORIGINAL ARTICLE

Correlation between computed tomography findings and liver function parameters in chronic liver disease

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ABSTRACT

Background: Chronic liver disease (CLD) leads to cirrhosis, hepatocellular carcinoma (HCC), and liver failure. Non-invasive assessment tools are crucial for the early detection and management of these complications of CLD.
Objective: To determine the correlation between computed tomography findings and liver function parameters in patients with chronic liver disease.
Methods: This retrospective study was conducted in the Department of Radiology, Lady Reading Hospital, Peshawar, Pakistan from December 2024 to January 2025 after obtaining the Institutional ethical approval (Ref. No. 569 MTI/LRH). Medical records of 276 patients with CLD from 1st October 2023 to 30th September 2024 were reviewed. Data were retrieved from the radiology department and laboratory information systems. Data of adult patients were included if they had undergone a contrast-enhanced computed tomography (CT) and liver function tests (LFTs) and hematological tests done within seven days of the CT scan. CT reports were assessed for cirrhosis, ascites, splenomegaly, portal vein thrombosis (PVT), and hepatocellular carcinoma. LFTs and hematological tests included ALT, AST, bilirubin, albumin, INR, and platelet count. Statistical analysis involved Pearson's correlation and multivariate logistic regression.
Results: Cirrhosis was present in 96.7%, ascites in 64.1%, splenomegaly in 47.8%, PVT in 40.2%, and HCC in 73.9%. There was a significant negative correlation ($r = -0.81$, $p<0.001$) between cirrhosis and serum albumin levels. The severity of ascites correlated positively ($r=0.70$, $p=0.008$) with serum bilirubin levels and INR ($r=0.60$, $p=0.010$), indicating worsening of hepatic function. Splenomegaly was significantly correlated with reduced platelet counts ($r = -0.75$, $p< 0.001$).
Conclusion: CT findings in chronic liver disease showed significant correlations with liver function parameters. Cirrhosis correlated with low albumin, ascites severity with elevated bilirubin and INR, and splenomegaly with thrombocytopenia. These findings highlight the complementary role of CT imaging and biochemical markers in assessing disease severity and hepatic dysfunction.

Key Words: Chronic Liver Disease, Cirrhosis, Liver Function Tests, Tomography X-Ray Computed.

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INTRODUCTION

Chronic liver disease (CLD) is a condition where the liver experiences long-term inflammation and scarring. This can lead to cirrhosis, hepatocellular carcinoma (HCC), and eventually liver failure.[1] Early diagnosis and treatment are crucial to managing these complications.[2] CLD presents a substantial challenge in Pakistan, contributing to the high rates of morbidity and mortality, with a high prevalence of Hepatitis B (HBV) and Hepatitis C (HCV), estimated to be around 2.5% and 4.8% respectively, major risk factors for CLD in the country.[3] CLD is an ailment contributing to high rates of illness and death. This

highlights the need for effective, non-invasive methods to assess the disease and detect complications immediately.

Modern imaging methods such as Computed tomography (CT) scans, Magnetic resonance imaging, and ultrasound play a vital role in evaluating changes in the liver. They help detect complications such as portal hypertension, ascites, splenomegaly, and portal vein thrombosis (PVT).[4] These tools provide valuable insights into the severity of CLD.[5] Liver function tests (LFTs) and hematological tests offer important information on liver damage. They measure key markers like alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin, albumin, international normalized ratio (INR), and platelet count. These tests help evaluate liver function and detect impairments.[6] Although widely used, the direct link between imaging and biochemical markers is poorly understood. This limits their full potential in non-invasive disease staging.[7]

Previous research has primarily examined either radiological assessments or biochemical markers in isolation.[8] While individual associations between cirrhosis and hypoalbuminemia or portal hypertension and thrombocytopenia have been reported,[9] comprehensive correlation analyses remain limited. Many studies lack standardized imaging criteria and robust statistical validation, restricting clinical applicability.[10]

The rationale is to enhance non-invasive staging and early detection of liver dysfunction by integrating radiological and biochemical data, which may improve diagnostic accuracy, help in timely intervention, and provide support in evidence-based management in clinical settings.

The objective of this study was to determine the correlation between CT findings and liver function parameters in patients with chronic liver disease and to determine the predictors of liver dysfunction.

METHODS

This retrospective study was conducted in the Department of Radiology, Lady Reading Hospital, Peshawar, Pakistan. Data from the hospital medical records of 276 patients diagnosed with CLD from 1st October 2023 to 30th September 2024 were included. Data of patients older than 20 years, diagnosed with CLD based on clinical history, persistent biochemical abnormalities (such as deranged LFTs for at least six months), and/or features suggestive of chronic liver injury, including liver surface nodularity, segmental volume redistribution, or signs of portal hypertension, on contrast-enhanced CT and corresponding LFTs and other hematological tests done within seven days of CT scan were included. Since it was retrospective, a waiver of informed consent was obtained from the Ethical Committee of the hospital. Patient confidentiality was maintained by anonymizing all imaging and laboratory records.

CT scan reports were excluded if they were incomplete, lacked documentation of key hepatic features (e.g., cirrhosis, ascites, splenomegaly, portal vein status, or liver masses), or if there was poor image quality (mentioned in the report) that limited diagnostic interpretation. Reports of patients with concurrent hematological disorders (written in the history available in the records) or those undergoing chemotherapy were also excluded, as these conditions could confound the interpretation of platelet count and INR values. Additionally, any cases with missing or delayed (>7 days) LFT data were excluded.

Radiological data were extracted from archived contrast-enhanced multiphasic CT scan reports in the hospital's Picture Archiving and Communication System (PACS). These reports were generated by board-certified radiologists during routine clinical care, key hepatic features including cirrhosis, ascites (graded as mild, moderate, or severe), splenomegaly (spleen >12 cm craniocaudal length), PVT, and HCC, diagnosed using LI-RADS criteria. All CT scans followed departmental protocols using a 160-slice multidetector. Liver-related laboratory parameters were retrieved from the hospital's laboratory information system, including ALT, AST, total and direct bilirubin, albumin, INR, and platelet count.

For consistency in assessment and analysis, the following operational definitions were applied while extracting data from past medical records and CT scan reports. Cirrhosis was identified based on documented liver surface nodularity, segmental volume redistribution, and the heterogeneous parenchymal texture. Ascites was graded as mild, moderate, or severe depending on the reported volume and distribution of free peritoneal fluid on CT. Splenomegaly was defined as a spleen measuring more than 12 cm in craniocaudal length. PVT was noted as partial or complete occlusion of the portal vein lumen. HCC was diagnosed according to LI-

RADS criteria, which included lesion size, number, arterial phase hyperenhancement, washout, and presence of vascular invasion. In terms of biochemical parameters, ALT and AST levels were used to assess hepatocellular injury, total and direct bilirubin for excretory function, albumin and INR for synthetic liver function, and platelet count as a surrogate marker for portal hypertension due to hypersplenism.

Ethical Approval

This study was conducted from December 2024 to January 2025 after taking approval from the Institutional Review Board (IRB) of Lady Reading Hospital, Peshawar, Pakistan (Ref. No. 569 MTI/LRH) on 31st December 2024.

Statistical Analysis

Data analysis was carried out using SPSS (version 26). Frequencies and percentages were calculated for categorical variables. Mean ± SD for quantitative variables. The correlation between computed tomography findings and liver function parameters was assessed using Pearson's correlation coefficient. A multivariate logistic regression model was employed to identify factors independently associated with liver dysfunction. The outcomes are presented regarding odds ratios (OR) with confidence intervals.

RESULTS

The study included 276 patients, with a male predominance (64.1%). The mean age was 56.2± 11.4 years. The prevalence of CLD was higher in males compared to females. Among the study population, cirrhosis was the most frequently observed CT scan finding (96.7%), followed by HCC (73.9%) and ascites (64.1%). PVT was present in 40.2% of patients, highlighting the extent of vascular complications in CLD (Table: 1)

Among the patients diagnosed with HCC, 60.3% of the lesions were classified as LR-5, indicating definite HCC. A total of 17.6% of lesions were LR-4 (probably HCC), while 7.4% were LR-3, representing an intermediate probability of HCC. Tumor in vein (LR-TIV) was observed in 14.7% of patients, reflecting vascular invasion, which is associated with a worse prognosis. No lesions were categorized as LR-1 or LR-2, as all included cases had radiological or clinical suspicion of HCC at the time of imaging (Table 2).

Biochemical markers of liver function demonstrated significant alterations in CLD patients. Elevated ALT, AST, and bilirubin levels, along with decreased albumin and platelet counts, were observed, reflecting the hepatic dysfunction and the portal hypertension (Table: 3).

Table 1: Computed tomography findings of patients suffering from chronic liver disease

Radiological Findings	n (%)
Cirrhosis	267 (96.7%)
Ascites (Total)	177 (64.1%)
Mild Ascites	61 (34.5%)
Moderate Ascites	74 (41.8%)
Severe Ascites	42 (23.7%)
Splenomegaly	132 (47.8%)
Portal Vein Thrombosis	111 (40.2%)
Hepatocellular Carcinoma (Total)	204 (73.9%)
Unifocal HCC	84 (41.2%)
Multifocal HCC	120 (58.8%)
Angioinvasion	99 (48.5%)

HCC= Hepatocellular Carcinoma

Table 2: Frequency of focal hepatic lesions based on LI-RADS classification on CT Imaging

LI-RADS Category	n (%)
LR-3 (Intermediate probability)	15 (7.4%)
LR-4 (Probably HCC)	36 (17.6%)
LR-5 (Definitely HCC)	123 (60.3%)
LR-TIV (Tumor in vein)	30 (14.7%)

HCC=Hepatocellular Carcinoma

Table 3: Liver function-related laboratory parameters

Biochemical Parameter	mean±SD
ALT (IU/L)	47.6±16.8
AST (IU/L)	72.4± 22.1
Bilirubin (mg/dL)	2.9 ± 0.90
Direct Bilirubin (mg/dL)	1.0 ± 0.50
Albumin (g/dL)	2.8 ± 0.70
INR	1.5 ± 0.40
Platelet Count (×10⁹/L)	112 ± 50

ALT=alanine aminotransferase; AST=aspartate aminotransferase; INR=international normalized ratio.

Significant correlations were observed between CT scan findings and markers of liver function using the Pearson's correlation test, reinforcing the relationship between hepatic structural abnormalities and liver dysfunction. Cirrhosis showed a significant negative correlation with albumin ($p<0.001$), suggesting a progressive decline in hepatic synthetic function. Ascites severity correlated significantly with bilirubin ($p=0.008$) and INR ($p=0.010$), indicating worsening hepatic function and coagulopathy. PVT was

associated with increased total bilirubin (p<0.001) and INR (p=0.019), reflecting impaired liver function and increased risk of thrombosis. Splenomegaly had a negative correlation with platelet count (r= -0.75, p<0.001), indicative of hypersplenism due to portal hypertension (Table: 4).

Cirrhosis had the highest predictive value (OR 3.72, p<0.001), reinforcing its role in CLD progression. Severe ascites (OR 2.95, p<0.01) and splenomegaly (OR 3.10, p<0.001) also significantly contributed to worsening liver function (Table: 5).

Table 4: Correlation between CT findings and liver function parameters

Radiological Finding	LFTs	Rho[a] (r)	p value
Cirrhosis	Albumin	-0.81	<0.001*
Cirrhosis	AST/ALT	0.58	0.002*
Ascites Severity	Total Bilirubin	0.70	0.008*
Ascites Severity	INR	0.60	0.010*
Splenomegaly	Platelet Count	-0.75	<0.001*
PVT	INR	0.41	0.030*
PVT	Total Bilirubin	0.72	<0.001*
HCC	INR	0.47	0.019*
HCC	AST/AL	0.65	<0.001*
HCC	Total Bilirubin	0.59	0.009*

*CT= computed tomography; ALT=alanine aminotransferase; AST=aspartate aminotransferase; LFTs=liver function test; INR=international normalized ratio; PVT=Portal Vein Thrombosis; HCC=Hepatocellular Carcinoma. Pearson's correlation was applied. *p< 0.05 was statistically significant.*

Table 5: Predictors of liver dysfunction

Variables	OR	95% CI	p value
Cirrhosis	3.72	2.30-6.02	<0.001*
Severe Ascites	2.95	1.90-4.67	0.009*
Splenomegaly	3.10	2.05-4.78	<0.001*

*Multivariate Regression Analysis was applied. OR=Odds Ratio; CI= Confidence Interval. *p< 0.05 was statistically significant.*

DISCUSSION

The present study explored the correlation between CT findings and liver function parameters in patients with chronic liver disease. By retrospectively analyzing imaging and laboratory data of 276 patients, we examined the association between key CT features—such as cirrhosis, ascites, and splenomegaly—and corresponding biochemical and hematological markers. The results demonstrated significant correlations, highlighting how imaging findings can reflect underlying hepatic dysfunction.

CLD is a major health issue worldwide, leading to serious complications like cirrhosis, HCC, and liver failure.[11] There is a strong need for effective, non-invasive tools to diagnose and manage CLD, as traditional methods like liver biopsy carry risks such as bleeding and sampling errors.[12] It highlights the value of imaging as a non-invasive way to assess liver function and disease progression. Our results confirmed previous findings and suggest that combining radiological and biochemical data can improve CLD diagnosis, staging, and early treatment. Our findings are consistent with previous studies that have demonstrated associations between imaging features and liver function markers in CLD. However, this study adds value by applying standardized CT imaging criteria and multivariate statistical analysis, supporting the integration of radiological and biochemical parameters in non-invasive disease assessment and staging.[12] There was significant correlation between cirrhosis and albumin levels. This matches findings from other studies that show liver function declines as fibrosis progresses. It supports albumin's role as a key marker of liver function in liver diseases.[13] Our study further supports the use of albumin as a prognostic biomarker for CLD. It also suggests that albumin could be integrated into disease staging systems, offering a reliable and non-invasive alternative to liver biopsy

In addition to cirrhosis, we found that the severity of ascites was positively associated with elevated bilirubin and INR levels, suggesting a decline in both excretory and synthetic liver function. This observation aligns with recent research by Wang et al. who demonstrated that increasing ascites volume on imaging correlates with worsening liver function parameters, particularly serum bilirubin and coagulation profiles such as INR.[14] The significant correlation between ascites and bilirubin and INR supports the known relationship between liver dysfunction, impaired bilirubin metabolism, and coagulopathy. This happens because the liver struggles to process bilirubin and produce clotting factors properly. These results are important for clinical practice. Using bilirubin and INR levels as markers for ascites severity could help identify patients at risk for complications like spontaneous bacterial peritonitis or variceal bleeding.

PVT is a serious complication in CLD. It is one of the reasons for worse health outcomes and higher

mortality rates. In this study, we found a strong positive correlation between PVT and elevated serum bilirubin levels. This finding is consistent with previous studies, which have demonstrated that PVT in the setting of CLD is associated with impaired hepatic perfusion and biliary congestion, contributing to hyperbilirubinemia. For instance, Luca et al. reported that PVT in cirrhotic patients is significantly linked with worsened liver function, including elevated bilirubin and INR levels.[15] Similarly, studies by Francoz et al. also noted that patients with PVT often present with higher bilirubin levels and more advanced liver dysfunction, supporting our observations.[16] The way by which they cause worsening of liver functions, hepatic congestion, and portal hypertension, all of which worsen liver function. As a result, bilirubin levels rise, and issues with blood clotting become more prominent. The connection between PVT and biochemical markers emphasizes the importance of vascular complications in the progression of CLD.

The present study identified cirrhosis as the most significant imaging predictor of liver dysfunction, with strong correlations to hypoalbuminemia and elevated INR. This finding aligns with existing literature. For instance, Yeom et al. emphasized that morphological features of cirrhosis on CT—particularly surface nodularity and caudate lobe hypertrophy—strongly correlate with impaired synthetic function, as measured by albumin and prothrombin time.[17] Similarly, Peng et al. found that radiological cirrhosis was associated with significantly worse Child-Pugh and MELD scores, reinforcing its diagnostic and prognostic value.[18]

Severe ascites also emerged as a strong indicator of hepatic dysfunction, correlating with elevated bilirubin and INR in the present study. This mirrors the findings of Sharma, who demonstrated that ascites, especially when moderate to severe on imaging, was independently associated with reduced synthetic capacity and poorer survival.[19] Ascites reflects both portal hypertension and impaired hepatic synthesis, explaining its strong clinical relevance.

Splenomegaly, while somewhat less predictive than cirrhosis or ascites in the current study, was significantly associated with thrombocytopenia, a surrogate for portal hypertension and hypersplenism. This is consistent with work by Berzigotti et al, who found that spleen size, when assessed by imaging, correlated inversely with platelet count and directly with hepatic venous pressure gradient (HVPG), an invasive marker of portal hypertension.[20]

Taken together, our results reinforce and expand upon prior studies by demonstrating that CT-detected cirrhosis, ascites, and splenomegaly are not just structural findings but correlate meaningfully with biochemical and clinical markers of hepatic decompensation. Among these, cirrhosis consistently shows the highest predictive value, supporting its central role in non-invasive disease staging. These results highlight the clinical importance of these radiological features and their role in guiding treatment decisions.

CONCLUSION

Computed tomography findings in patients with chronic liver disease, such as cirrhosis, ascites, splenomegaly, PVT, and HCC, show significant correlations with parameters of liver function, highlighting how imaging findings can reflect underlying hepatic dysfunction. Cirrhosis correlated with low albumin, ascites severity with elevated bilirubin and INR, and splenomegaly with thrombocytopenia. These findings highlight the complementary role of CT imaging and liver function parameters in assessing disease severity and hepatic dysfunction.

Limitations of the study and future directions

It was a retrospective, single-center study, which may be subject to selection bias and limits the generalizability of the findings to broader populations. The timing of CT scans and laboratory tests, although restricted to within seven days, may still allow for clinical fluctuations in liver function that could affect correlation strength. Additionally, the study relied solely on imaging findings without histopathological confirmation, which may impact diagnostic accuracy for conditions such as cirrhosis or hepatocellular carcinoma. Key clinical details such as disease duration, Child-Pugh class, and liver disease etiology were not available, reducing the ability to adjust for confounders. Although HCC was categorized by type, no subgroup analysis was performed. Additionally, CT's full potential to detect complications like varices or collaterals was not explored.

Future studies should adopt a prospective, multicenter approach to validate these findings across diverse populations. Including detailed clinical data

and performing subgroup analyses, especially for different HCC types, would improve accuracy. CT imaging should also be used to its full diagnostic capacity to evaluate other CLD complications for better staging and management. Incorporating more precise imaging techniques, such as magnetic resonance imaging (MRI), and aligning laboratory tests more closely with imaging timing, may strengthen real-time clinical correlations.

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AUTHOR'S CONTRIBUTIONS:

- **MKK:** Conception of study, study design, data acquisition & analysis, manuscript drafting
- **SKSB**: Conception of study, data acquisition & analysis, manuscript drafting, critical review
- **SMSB:** Data collection, critical review, manuscript drafting
- **MZS:** Conception of study, study design, interpretation of data, critical review
- **MRS:** Data collection, critical review, manuscript drafting

All authors approved the final version to be published and agreed to be accountable for all aspects of the work, ensuring that any questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

CONFLICT OF INTEREST:

None

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