

Computed tomography-based morphologic changes and their association with liver function parameters in chronic liver disease

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ABSTRACT

Objective: To evaluate the association between computed tomography (CT) based morphologic features of chronic liver disease (CLD) and liver function parameters in patients with CLD.

Material and Methods: This hospital-based cross-sectional analytical study was conducted at Bahawal Victoria Hospital and Quaid-e-Azam Medical College, Bahawalpur, from January to December 2025. Consecutive adults with clinically suspected or established CLD who underwent multiphasic contrast-enhanced abdominal CT and had liver function tests available within 7 days were included. CT findings included cirrhosis, ascites (graded), splenomegaly, portal vein thrombosis, and hepatocellular carcinoma assessed using standardized criteria. Laboratory parameters included ALT, AST, bilirubin, albumin, INR, and platelet count. Associations between CT findings and laboratory parameters were assessed, and multivariable binary logistic regression was performed for liver dysfunction.

Results: Fifty participants were included; 28 (56.0%) were female and 41 (82.0%) met the liver dysfunction definition. Cirrhosis ($\rho = -0.347$, $p = 0.013$) and ascites grade ($\rho = -0.294$, $p = 0.038$) correlated with lower albumin, while splenomegaly correlated with lower platelet count ($\rho = -0.610$, $p < 0.001$). Other associations were not significant. Hepatocellular carcinoma showed complete separation (all cases met the dysfunction definition) and was excluded from the regression model. No CT feature remained independently associated and cirrhosis showed a borderline association (OR 5.17, $p = 0.070$).

Conclusion: Routine multiphasic CT findings showed meaningful associations with selected laboratory indicators of liver dysfunction and portal hypertension in CLD. Larger multicenter studies are needed to validate these findings.

Keywords: Ascites, Chronic liver disease, Computed tomography, Portal hypertension, Splenomegaly

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INTRODUCTION

Chronic liver disease (CLD) involves ongoing hepatic inflammation and progressive fibrosis. With time, it may progress to cirrhosis, portal hypertension, hepatocellular carcinoma (HCC), and liver failure. The global burden of cirrhosis is high and continues to increase in many regions. This rise relates to changing causes of disease and delayed identification of advanced stages [1,2]. In low- and middle-income countries, outcomes are further affected by limited specialist services and late presentation. Pakistan carries a major CLD burden because viral hepatitis is endemic. Recent national and provincial findings show a large pool of hepatitis

C virus (HCV) infection. Modelling estimates suggest a viraemic HCV prevalence of about 4% around 2021, with clear provincial variation [3]. Repeated serosurveys also suggest sustained exposure to hepatitis B virus (HBV) and HCV in high-burden settings [4]. Meta-analyses of Pakistani studies report substantial variation across populations. This pattern reflects continued transmission and uneven screening coverage [5]. These factors contribute to frequent cirrhosis-related complications and high healthcare use. Early detection of decompensation and timely risk stratification remain essential to improve outcomes.

Non-invasive evaluation is now central to hepatology practice. Routine laboratory tests and composite scores are commonly used to estimate liver function and prognosis. Liver function tests (LFTs) provide information on hepatocellular injury and synthetic capacity. Key markers include alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin, albumin, and the international normalized ratio (INR). Platelet count is also clinically

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relevant. Thrombocytopenia often reflects portal hypertension through splenic sequestration and reduced thrombopoietin. Current consensus guidance supports combining non-invasive markers when estimating clinically significant portal hypertension and related risk [6].

Cross-sectional imaging adds complementary information by showing structural and hemodynamic effects of CLD. Computed tomography (CT) is widely used in routine care. It assesses liver contour and parenchymal heterogeneity, detects focal lesions, and identifies complications of portal hypertension. Common CT findings include ascites, splenomegaly, portosystemic collaterals, gastroesophageal varices, and portal vein thrombosis. Evidence also supports the use of CT-derived criteria for risk assessment. Models using ascites, varices, spontaneous portosystemic shunts, and splenomegaly have been assessed to classify clinically significant portal hypertension in CLD [7]. Quantitative measures are also being studied. CT-based spleen volume correlates with invasive portal pressure and has been used in predictive models with clinical indices [8]. Dual-energy CT measures have shown associations with functional reserve, including Child–Pugh class [9]. These findings suggest that imaging features may reflect pathophysiology that is also captured by biochemical abnormalities.

However, the relationship between routine CT findings and standard liver function parameters remains unclear. Many studies emphasize single outcomes or specialized imaging metrics that are not consistently available. Some work also uses variable imaging definitions and limited control of confounding factors. These issues reduce clinical applicability [8–10]. In Pakistan, CT is frequently used for abdominal symptoms and complication assessment. Clearer evidence is needed on how common CT findings relate to LFTs and hematologic indices. Such information could support non-invasive staging and earlier recognition of decompensation. It may also improve referral decisions and prioritization for surveillance.

Therefore, the present study was designed to evaluate the association between CT findings and liver function parameters in patients with chronic liver disease. In addition, it aimed to identify CT features associated with liver dysfunction using routinely available imaging features and laboratory measures. Such integrated radiologic–biochemical assessment may strengthen evidence-based management in

resource-constrained clinical settings and help guide timely interventions.

MATERIAL AND METHODS

This hospital-based cross-sectional analytical study was conducted in the Department of Radiology, Bahawal Victoria Hospital, and the Department of Pathology, Quaid-e-Azam Medical College, Bahawalpur, from January to December 2025. Consecutive adults (≥ 20 years) with clinically suspected or established chronic liver disease were included if they had persistent abnormalities in liver biochemistry (e.g., serum bilirubin, ALT, AST, albumin, and/or INR) for at least six months and/or imaging features suggestive of chronic liver injury. Eligible participants also underwent multiphasic contrast-enhanced CT and had laboratory tests available within 7 days of CT (preferably on the same day). The primary objective was to assess cross-sectional associations between CT morphologic findings and liver function parameters. The minimum sample size for the primary correlation analysis (expected $r = 0.41$, two-sided $\alpha = 0.05$, and power = 80%)¹¹ was 45 participants, calculated using Fisher's z-based sample size estimation for a correlation coefficient. A target sample size of 50 participants was selected after allowing for 10% inflation for possible incomplete data.

<https://www.medcalc.org/en/calc/sample-size-correlation-coefficient.php>

All CT examinations were performed using a standardized departmental multiphasic contrast protocol on a 64-slice multidetector CT scanner (Toshiba Medical Systems, Japan). Imaging findings were recorded on a structured proforma and included cirrhosis, ascites (graded as mild, moderate, or severe), splenomegaly, portal vein thrombosis (PVT), and hepatocellular carcinoma (HCC). Cirrhosis was documented when CT showed typical morphologic features such as an irregular or nodular liver surface, segmental volume redistribution, and heterogeneous parenchymal texture. Ascites was graded according to the volume and distribution of intraperitoneal free fluid. Mild ascites was defined as a small amount of perihepatic/perisplenic or pelvic free fluid; moderate ascites as fluid extending into the paracolic gutters; and severe ascites as large-volume, generalized intraperitoneal fluid with bowel floating. Splenomegaly was defined as a craniocaudal splenic length >12 cm. PVT was defined as partial or complete occlusion of the portal vein lumen. HCC was evaluated on multiphasic CT using LI-RADS

criteria. The assessment considered lesion size and number, arterial phase hyperenhancement, washout, and signs of vascular invasion. For quality assurance, scans were interpreted at consultant level and reported blinded to laboratory results.

Laboratory data were obtained from the hospital laboratory information system. It included alanine aminotransferase (ALT), aspartate aminotransferase (AST), total and direct bilirubin, serum albumin, international normalized ratio (INR), and platelet count. Serum biochemistry (including ALT, AST, bilirubin, and albumin) was analyzed on the Cobas c303 analyzer (Roche Diagnostics, Germany), while platelet counts were measured using the Swelab Alfa Plus automated hematology analyzer (Boule Diagnostics, Sweden). ALT and AST were used as indicators of hepatocellular injury. Bilirubin reflected excretory function. Albumin and INR reflected hepatic synthetic function. Platelet count was included because thrombocytopenia is commonly used in local practice as an indirect marker of portal hypertension and hypersplenism. For analysis, liver dysfunction was operationalized using pragmatic thresholds based on commonly used indicators of hepatic synthetic function in clinical practice, such as serum albumin <3.5 g/dL and/or INR >1.5 , with bilirubin interpreted alongside the clinical context [12,13]. Because albumin and INR may be influenced by non-hepatic factors (e.g., nutrition/inflammation, anticoagulant use, or vitamin K status), these measures were interpreted alongside clinical history. Patients were excluded if CT image quality was inadequate for confident assessment, if key imaging variables could not be evaluated, if laboratory results were unavailable within the defined timeframe, or if conditions likely to confound INR or platelet count were present, including known hematological disorders or ongoing chemotherapy. Patients receiving anticoagulant therapy were also excluded due to potential INR confounding.

Ethical approval was obtained from the Institutional Review Board before recruitment (IRB QAMC 009, 2025). Study records were de-identified to maintain confidentiality. Data analysis was performed using SPSS (version 26). Categorical variables included sex, cirrhosis (yes/ no), ascites (yes/ no), ascites grade (none/ mild/ moderate/ severe), splenomegaly (yes/ no), portal vein thrombosis (yes/ no), hepatocellular carcinoma (yes/ no), and liver dysfunction (yes/ no; defined as serum albumin <3.5 g/dL and/or INR >1.5); these were summarized as frequencies and percentages. Continuous variables

included age, ALT, AST, total bilirubin, direct bilirubin, serum albumin, INR, and platelet count; these were summarized as mean $\pm$ standard deviation (or median with interquartile range when non-normally distributed). Spearman's rank correlation coefficient (ρ) was used because CT variables were binary/ordinal and a non-parametric method was appropriate in this sample. Spearman correlation was performed between CT variables (cirrhosis, ascites grade, splenomegaly, portal vein thrombosis, HCC) and laboratory variables (albumin, INR, total bilirubin, direct bilirubin, platelet count, ALT, AST). A multivariable binary logistic regression model using the enter method was used to evaluate CT findings associated with liver dysfunction. The dependent variable was liver dysfunction (yes/ no). Independent variables were cirrhosis, ascites grade (per 1-level increase), splenomegaly, and portal vein thrombosis; HCC was excluded because complete separation occurred. Results were reported as odds ratios with 95% confidence intervals, and $p < 0.05$ was considered statistically significant.

RESULTS

Fifty adults with clinically suspected or established chronic liver disease were included. The mean age was 48.88 ± 7.91 years, and 28 (56.0%) were female. Liver dysfunction (albumin <3.5 g/dL and/or INR >1.5) was present in 41 (82.0%) participants. This high prevalence was largely driven by hypoalbuminemia, with INR elevations interpreted in the clinical context. Table-I shows the baseline characteristics of study participants. Cirrhosis was identified in 38 (76.0%) participants. Ascites was present in 27 (54.0%) and was graded as mild in 15 (30.0%), moderate in 5 (10.0%), and severe in 7 (14.0%). Splenomegaly was observed in 22 (44.0%), portal vein thrombosis in 5 (10.0%), and hepatocellular carcinoma in 7 (14.0%) as shown in Table-II.

Spearman's rank correlation analysis (Table-III) demonstrated statistically significant inverse associations between cirrhosis and serum albumin ($\rho = -0.347$, $p = 0.013$) and between ascites grade and serum albumin ($\rho = -0.294$, $p = 0.038$). Splenomegaly showed a strong inverse association with platelet count ($\rho = -0.610$, $p < 0.001$). Correlations of CT findings with INR, bilirubin, ALT, and AST were not statistically significant in this sample. In the unadjusted cross-tabulation, all participants with HCC met the study definition of liver dysfunction (complete separation); therefore, HCC was not entered into the multivariable model. In the

multivariable binary logistic regression model for liver dysfunction (Table-IV), cirrhosis showed an adjusted odds ratio (aOR) of 5.17 (95% CI: 0.87-30.63; $p = 0.070$), indicating a positive but not statistically significant association after adjustment. Ascites grade (per 1-level increase) had an aOR of 2.18

(95% CI: 0.64-7.45; $p = 0.215$), splenomegaly had an aOR of 2.52 (95% CI: 0.47-13.49; $p = 0.281$), and portal vein thrombosis had an aOR of 0.61 (95% CI: 0.04-8.73; $p = 0.718$). Wide confidence intervals suggest imprecision related to the small sample size and low frequency of some CT findings.

Table-I: Participant characteristics and laboratory profile (n=50).

Variable	Value
Age (years), mean $\pm$ SD	48.88 $\pm$ 7.91
Sex, n (%)	
Female	28 (56%)
Male	22 (44%)
ALT (IU/L), mean $\pm$ SD	45.47 $\pm$ 10.38
AST (IU/L), mean $\pm$ SD	74.49 $\pm$ 16.45
Total bilirubin (mg/dL), mean $\pm$ SD	2.82 $\pm$ 1.38
Direct bilirubin (mg/dL), mean $\pm$ SD	1.59 $\pm$ 0.86
Albumin (g/dL), mean $\pm$ SD	3.15 $\pm$ 0.68
INR, mean $\pm$ SD	1.40 $\pm$ 0.26
Platelets ($\times 10^9/L$), mean $\pm$ SD	138.16 $\pm$ 62.40

Table-II: CT findings on multiphasic contrast-enhanced CT (n=50).

CT finding	n (%)
Cirrhosis	38 (76.0)
Ascites (any)	27 (54.0)
Splenomegaly	22 (44.0)
Portal vein thrombosis	5 (10.0)
HCC	7 (14.0)
Ascites grade: None	23 (46.0)
Ascites grade: Mild	15 (30.0)
Ascites grade: Moderate	5 (10.0)
Ascites grade: Severe	7 (14.0)

Table-III: Spearman correlation of CT findings with laboratory parameters (rho with p-value).

CT finding	Albumin	INR	Total bilirubin	Direct bilirubin	Platelets	ALT	AST
Cirrhosis	-0.347 ($p=0.013$)	0.261 ($p=0.067$)	0.146 ($p=0.312$)	0.110 ($p=0.446$)	-0.159 ($p=0.270$)	-0.146 ($p=0.312$)	-0.190 ($p=0.187$)
Ascites Grade	-0.294 ($p=0.038$)	0.213 ($p=0.137$)	0.223 ($p=0.119$)	0.227 ($p=0.113$)	-0.024 ($p=0.866$)	0.004 ($p=0.977$)	-0.245 ($p=0.086$)
Splenomegaly	-0.112 ($p=0.440$)	0.207 ($p=0.150$)	-0.004 ($p=0.977$)	-0.008 ($p=0.954$)	-0.610 ($p<0.001$)	0.110 ($p=0.446$)	0.194 ($p=0.177$)
Portal vein thrombosis	-0.009 ($p=0.949$)	-0.007 ($p=0.962$)	-0.081 ($p=0.577$)	0.012 ($p=0.937$)	-0.171 ($p=0.235$)	-0.016 ($p=0.911$)	-0.014 ($p=0.924$)
HCC	-0.238 ($p=0.097$)	-0.010 ($p=0.945$)	-0.004 ($p=0.978$)	-0.042 ($p=0.772$)	-0.170 ($p=0.239$)	-0.020 ($p=0.891$)	-0.006 ($p=0.967$)

Table-IV: Multivariable logistic regression for liver dysfunction (n=50).

Predictor	OR (95% CI)	P
Cirrhosis	5.17 (0.87–30.63)	0.070
Ascites grade (per 1-level increase)	2.18 (0.64–7.45)	0.215
Splenomegaly	2.52 (0.47–13.49)	0.281
Portal vein thrombosis	0.61 (0.04–8.73)	0.718

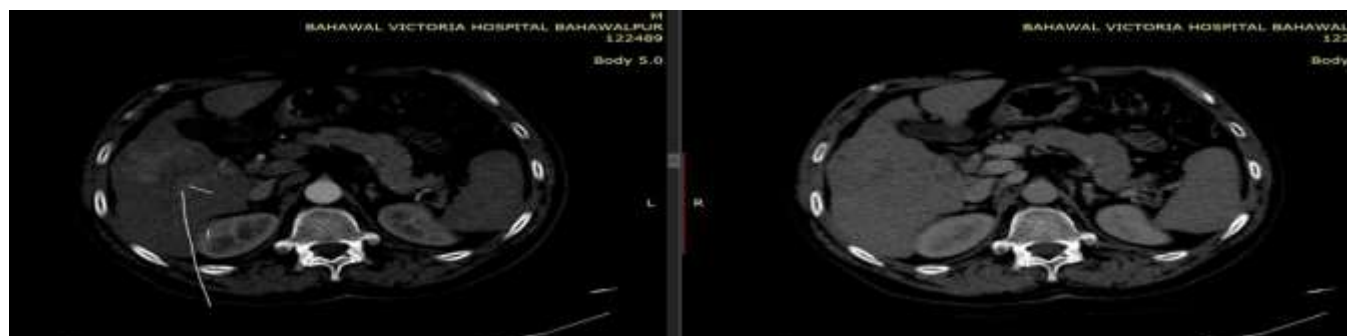


Figure-I: An arterially enhancing lesion in the right lobe of cirrhotic liver with Porto venous wash out and right portal vein invasion, consistent with Hepatocellular Carcinoma (HCC).

DISCUSSION

In this study, adults with chronic liver disease who underwent multiphasic contrast-enhanced CT, routine CT features of portal hypertension and advanced liver injury were associated with impaired hepatic function on laboratory testing. Ascites and splenomegaly showed the clearest links with worse synthetic and hematologic profiles. Cirrhosis and ascites were associated with lower albumin. Splenomegaly showed the strongest inverse association with platelet count. Hepatocellular carcinoma and portal vein thrombosis were less common. They were not independently associated with liver dysfunction in the adjusted model, likely due to low case numbers.

The association between CT signs of portal hypertension and hepatic dysfunction is expected. Progressive architectural distortion increases intrahepatic resistance and portal pressure. This drives splanchnic vasodilation and neurohumoral activation, which promote sodium and water retention and reduce effective arterial blood volume. These processes underpin decompensation and worsen outcomes. Current consensus and contemporary guidance place portal hypertension at the center of decompensation risk in advanced chronic liver disease [12].

Ascites is a defining event in decompensated cirrhosis. It often reflects clinically significant portal hypertension and systemic circulatory dysfunction. Modern guidance consistently frames new or worsening ascites as a major prognostic milestone that should prompt closer follow-up and active management [13,14]. In our cohort, ascites on CT behaved as a practical marker of more advanced functional compromise. This supports the role of imaging as a severity indicator, particularly when the clinical history is incomplete at the time of reporting. Similar approaches using cross-sectional imaging to infer portal hypertension severity have been reported, including CT-based criteria intended to help rule in or rule out clinically significant portal hypertension [7].

Splenomegaly is a classic manifestation of portal hypertension and hypersplenism and is commonly associated with thrombocytopenia. Recent CT literature supports splenic metrics as useful surrogates of portal pressure. CT-derived spleen volume has shown good diagnostic performance for severe portal hypertension at clinically relevant HVP thresholds [8]. CT liver and spleen volumetry has also been explored in selected populations, including patients with hepatocellular carcinoma,

where volumetric measures helped identify clinically significant portal hypertension [15]. In addition, automated CT-derived indices, such as the liver-to-spleen volume ratio, have been evaluated as prognostic markers in cohorts of compensated cirrhosis [16]. Platelet-based indices anchored to spleen measurements have also been associated with variceal risk in viral cirrhosis [17]. Our findings align with this evidence and support systematic reporting of splenic size as part of structured CT assessment in chronic liver disease. The EASL guideline on non-invasive tests also supports combining complementary markers for prognosis and risk stratification when feasible [6].

Cirrhosis on CT is defined by morphologic changes such as surface nodularity and segmental volume redistribution. These findings reflect chronicity and commonly coexist with portal hypertension. Studies from the region have also reported correlations between CT features and liver function parameters, supporting the clinical relevance of structured CT assessment in local practice [11].

Hepatocellular carcinoma was assessed using standardized multiphasic criteria. International guidance endorses multiphasic CT and MRI for non-invasive diagnosis and staging in at risk patients [18, 19]. Standardization through LI RADS has improved diagnostic consistency across centers and has supported clearer multidisciplinary communication [20]. In our cohort, HCC was more frequent among patients with advanced liver dysfunction. This finding is expected, given that most HCC develops in cirrhotic livers and that preserved hepatic reserve is a major determinant of treatment options [18,19]. However, HCC at diagnosis does not necessarily imply impaired synthetic function. Baseline liver reserve and tumor burden differ substantially across patients. This variability likely contributed to the absence of a consistent independent association with our study definition of dysfunction.

Portal vein thrombosis is a recognized complication of cirrhosis and reflects reduced portal flow, altered hemodynamics, and a rebalanced hemostatic system. Current guidance recommends individualized management based on acuity, extent, symptoms, bleeding risk, and transplant candidacy [21,22]. In our cohort, PVT occurred infrequently, which limited statistical power. This is consistent with the variable clinical impact reported in reviews, where outcomes depend on chronicity, degree of occlusion, mesenteric extension, and baseline liver reserve [23].

There is increasing interest in extracting more prognostic information from CT acquired for routine clinical indications. Beyond qualitative reporting, newer work has evaluated CT texture features and radiomics for risk stratification in portal hypertension and chronic liver disease [24]. Broader imaging reviews also highlight the growing role of advanced analytics and artificial intelligence to support biomarker discovery and severity assessment [10,25]. These approaches remain research-focused for many public sector hospitals, but they point to future tools that could augment routine reporting.

STRENGTHS AND LIMITATIONS

This study used a standardized multiphasic CT protocol and structured complication reporting. Scans were interpreted by blinded consultant radiologists, reducing reporting bias, and routine laboratory tests improved relevance for resource-limited settings. However, the single-center design and small sample limited assessment of uncommon findings such as PVT and HCC. The imbalanced outcome distribution may also have reduced the precision of logistic regression estimates. Liver dysfunction was defined pragmatically without Child–Pugh or MELD, and because CT and laboratory data were collected at one time point, causality cannot be inferred. Etiology, treatment effects, observer dependence in CT interpretation, and non-hepatic influences on albumin and INR may also have affected classification.

Contrast-enhanced CT is commonly used in CLD to assess symptoms and complications. Our findings support the value of routine features such as cirrhotic morphology, ascites, and splenomegaly, which may reflect portal hypertension and reduced functional reserve. These findings can help guide review, referral, variceal risk assessment, and closer follow-up or surveillance.

CONCLUSION

Routine multiphasic CT morphologic features of chronic liver disease, particularly cirrhotic morphology, ascites severity, and splenomegaly, showed meaningful associations with selected laboratory indicators of liver dysfunction and portal hypertension. However, no CT feature demonstrated an independent association with the binary liver dysfunction outcome in multivariable analysis. Systematic reporting of these features may complement laboratory assessment and help identify patients who may require closer follow-up and

specialist evaluation. Larger multicenter studies are needed to validate these findings.

CONFLICT OF INTEREST

None

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Declared none

AUTHORS CONTRIBUTION

Sara Nisar: Substantial contributions to study design, acquisition of data, reviewing manuscript critically for important intellectual content, final approval, accountable for all aspects of publication.

Sara Reza: Substantial contributions to acquisition of data and data analysis, manuscript drafting and reviewing it critical for important intellectual content, final approval, accountable for all aspects of publication.

Tanzeela Akram: Substantial contributions to analysis and interpretation of data, critical review, final approval, accountable for all aspects of publication.

Lubna Sarfaraz: Substantial contributions to concept, study design, critical review, final approval, accountable for all aspects of publication.

Hassan Ali: Substantial contributions to concept, study design, critical review, final approval, accountable for all aspects of publication.

REFERENCES

1. Tham EKJ, Tan DJH, Danpanichkul P, Ng CH, Syn N, Koh B, *et al.* The global burden of cirrhosis and other chronic liver diseases in 2021. *Liver Int.* 2025; 45(3): e70001. DOI: <https://doi.org/10.1111/liv.70001>.
2. Duo H, You J, Du S, Yu M, Wu S, Yue P, *et al.* Liver cirrhosis in 2021: Global Burden of Disease study. *PLoS One.* 2025; 20(7): e0328493. DOI: <https://doi.org/10.1371/journal.pone.0328493>.
3. Mooneyhan E, Qureshi H, Mahmood H, Tariq M, Maqbool NA, Anwar M, *et al.* Hepatitis C prevalence and elimination planning in Pakistan, a bottom-up approach accounting for provincial variation. *J Viral Hepat.* 2023; 30(4):345-54. DOI: <https://doi.org/10.1111/jvh.13802>.
4. Alamneh TS, Walker JG, Lim AG, Alam E, Hamid S, Foster GR, *et al.* Changes in the prevalence of hepatitis B and C viral infections in Sindh province, Pakistan: Findings from two sero-surveys in 2007 and 2019. *J Viral Hepat.* 2024; 31(11): 645-56. DOI: <https://doi.org/10.1111/jvh.13986>.
5. Al Kanaani Z, Mahmud S, Kouyoumjian SP, Abu-Raddad LJ. The epidemiology of hepatitis C virus in Pakistan: Systematic review and meta-analyses. *R Soc Open Sci.* 2018; 5(4): 180257. DOI: <https://doi.org/10.1098/rsos.180257>.

6. European Association for the Study of the Liver. EASL Clinical Practice Guidelines on non-invasive tests for evaluation of liver disease severity and prognosis - 2021 update. *J Hepatol.* 2021; 75(3): 659-89. DOI: <https://doi.org/10.1016/j.jhep.2021.05.025>
7. Heo S, Lee SS, Choi SH, Kim DW, Park HJ, Kim SY, *et al.* CT rule-in and rule-out criteria for clinically significant portal hypertension in chronic liver disease. *Radiology.* 2023; 309(1): e231208. DOI: <https://doi.org/10.1148/radiol.231208>
8. Chen X, Zhou J, Lin Y, Jia K, Tian J, Li F, *et al.* CT-derived spleen volume accurately diagnoses severe portal hypertension at HVP thresholds of 16 mmHg. *Sci Rep.* 2025; 15(1): 16850. DOI: <https://doi.org/10.1038/s41598-025-02143-3>
9. Hong SJ, Kim JE, Cho JM, Choi HC, Park MJ, Choi HY, *et al.* Dual energy CT-derived liver extracellular volume fraction for assessing liver functional reserve in patients with liver cirrhosis. *Medicina (Kaunas).* 2025; 61(9): 1561. DOI: <https://doi.org/10.3390/medicina61091561>
10. Dana J, Venkatasamy A, Saviano A, Lupberger J, Hoshida Y, Vilgrain V, *et al.* Conventional and artificial intelligence-based imaging for biomarker discovery in chronic liver disease. *Hepatol Int.* 2022; 16(3): 509-522. DOI: <https://doi.org/10.1007/s12072-022-10303-0>
11. Khalil MK, Bukhari SKS, Bukhari SMS, Shah MZ, Shah MR. Correlation between computed tomography findings and liver function parameters in chronic liver disease. *J Shalamar Med Dent Coll.* 2025; 6(1): 16-21. DOI: <https://doi.org/10.53685/jshmdc.v6i1.308>
12. Kaplan DE, Bosch J, Ripoll C, Thiele M, Fortune BE, Simonetto DA, *et al.* AASLD Practice Guidance on risk stratification and management of portal hypertension and varices in cirrhosis. *Hepatology.* 2024; 79(5): 1180-211. DOI: <https://doi.org/10.1097/hep.0000000000000647>
13. Biggins SW, Angeli P, Garcia-Tsao G, Ginès P, Ling SC, Nadim MK, *et al.* Diagnosis, evaluation, and management of ascites, spontaneous bacterial peritonitis and hepatorenal syndrome: 2021 practice guidance by the American Association for the Study of Liver Diseases. *Hepatology.* 2021; 74(2): 1014-48. DOI: <https://doi.org/10.1002/hep.31884>
14. Aithal GP, Palaniyappan N, China L, Harman DJ, Macken L, Ryan JM, *et al.* Guidelines on the management of ascites in cirrhosis. *Gut.* 2021; 70(1): 9-29. DOI: <https://doi.org/10.1136/gutjnl-2020-321790>
15. Romero-Cristóbal M, Gallego-Durán R, De la Mata M, López-Méndez E, Santos-Laso A, *et al.* CT-derived liver and spleen volume accurately diagnose clinically significant portal hypertension in patients with hepatocellular carcinoma. *JHEP Rep.* 2023; 5: 100645. DOI: <https://doi.org/10.1016/j.jhepr.2022.100645>
16. Kwon JH, Lee SS, Lee JM, Lee DH, Yoon JH, Kim JH, *et al.* Liver-to-spleen volume ratio automatically measured on CT predicts decompensation in patients with B viral compensated cirrhosis. *Korean J Radiol.* 2021; 22(12): 1985-95. DOI: <https://doi.org/10.3348/kjr.2021.0348>
17. Yu S, Chen W, Jiang Z. Platelet count/ spleen volume ratio has a good predictive value for esophageal varices in patients with hepatitis B liver cirrhosis. *PLoS One.* 2021; 16(12): e0260774. DOI: <https://doi.org/10.1371/journal.pone.0260774>
18. Singal AG, Llovet JM, Yarchoan M, Mehta N, Heimbach JK, Dawson LA, *et al.* AASLD Practice Guidance on prevention, diagnosis, and treatment of hepatocellular carcinoma. *Hepatology.* 2023; 78(6): 1922-65. DOI: <https://doi.org/10.1097/hep.0000000000000466>
19. European Association for the Study of the Liver. EASL Clinical Practice Guidelines on the management of hepatocellular carcinoma. *J Hepatol.* 2025; 82(2): 315-74. DOI: <https://doi.org/10.1016/j.jhep.2024.08.028>
20. Marks RM, Ryan A, Heba ER, Wolfson T, Sirlin CB. LI-RADS: past, present, and future. *AJR Am J Roentgenol.* 2021; 216(2): 295-307. DOI: <https://doi.org/10.2214/ajr.20.24272>
21. Northup PG, Garcia-Pagan JC, Garcia-Tsao G, Intagliata NM, Superina RA, Roberts LN, *et al.* Vascular liver disorders, portal vein thrombosis, and procedural bleeding in patients with liver disease: 2020 practice guidance by the American Association for the Study of Liver Diseases. *Hepatology.* 2021; 73(1): 366-413. DOI: <https://doi.org/10.1002/hep.31646>
22. Davis JPE, Lim JK, Francis FF, Ahn J. AGA Clinical Practice Update on management of portal vein thrombosis in patients with cirrhosis: Expert review. *Gastroenterology.* 2025; 168(2): 396-404.e1. DOI: <https://doi.org/10.1053/j.gastro.2024.10.038>
23. Prakash S, Bies J, Hassan M, Mares A, Didia SC. Portal vein thrombosis in cirrhosis: A literature review. *Front Med (Lausanne).* 2023; 10: 1134801. DOI: <https://doi.org/10.3389/fmed.2023.1134801>
24. Wan S, Wei Y, Zhang X, Yang C, Hu F, Song B. Computed tomography-based texture features for the risk stratification of portal hypertension and prediction of survival in patients with cirrhosis: a preliminary study. *Front Med (Lausanne).* 2022; 9: 863596. DOI: <https://doi.org/10.3389/fmed.2022.863596>
25. Morariu-Barb AM, Drugan T, Socaciu MA, Stefanescu H, Morariu AD, Lupsor-Platon M. CT-based radiomic models in biopsy-proven liver fibrosis staging: direct comparison of segmentation types and organ inclusion. *Diagnostics (Basel).* 2025; 15(21): 2671. DOI: <https://doi.org/10.3390/diagnostics15212671>