e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 75-82

Original Research Article

Role of Serum Uric Acid Levels as a Predictor for Assessing the Severity of Chronic Liver Disease

Rajubhai Padaya[1], Kautilya Chaturvedi[2], Heti Mistry[3], Shubhangi Deshpande[4]

[1,2]3rd year Resident doctor, Department of General Medicine, GMERS medical college & Hospital, Gotri, Vadodara, Gujarat

[3]Assistant Professor, Department of General Medicine, GMERS medical college & Hospital, Gotri, Vadodara, Gujarat

[4]Associate Professor, Department of General Medicine, GMERS medical college & Hospital, Gotri, Vadodara, Gujarat

Received: 25-07-2026 / Revised: 31-07-2026 / Accepted: 01-08-2026

Corresponding author: Dr. Heti Mistry

Conflict of interest: Nil

Abstract

Background: Chronic liver disease represents a major global health burden and is associated with significant morbidity and mortality. Disease progression from compensated liver disease to decompensated cirrhosis is often accompanied by metabolic disturbances, systemic inflammation, and impaired hepatic function. Several studies have suggested that elevated serum uric acid levels may correlate with nonalcoholic fatty liver disease, cirrhosis, and liver-related complications. However, the role of serum uric acid as a surrogate biomarker for determining disease severity in chronic liver disease remains insufficiently explored, particularly in hospital-based populations. The Child–Pugh classification remains widely used for assessing disease severity, but the addition of biochemical markers may improve clinical utility. Therefore, the present study aimed to assess serum uric acid levels in patients with chronic liver disease, analyze the association between serum uric acid levels and disease severity.

Methods: The present study was conducted as a hospital-based observational cross-sectional study in the Department of General Medicine at GMERS Medical College and Hospital, Gotri, Vadodara. The study duration extended from January 2024 to December 2025. A total of 150 patients diagnosed with chronic liver disease were included consecutively after fulfilling predefined inclusion and exclusion criteria. Patients aged 18 years and above, clinically, biochemically, and radiologically diagnosed with chronic liver disease, and willing to provide informed consent were included. Patients with acute liver failure, acute-on-chronic liver failure, malignancy, pregnancy, or those receiving medications affecting uric acid metabolism were excluded from the study. Data collection was performed using a structured proforma. Sociodemographic variables including age, sex, residence, socioeconomic status, and educational level were recorded. Detailed clinical history including alcohol intake, smoking habits, and presenting symptoms was obtained. A thorough clinical examination was performed, including measurement of vital parameters and assessment of signs of chronic liver disease. Laboratory investigations included complete blood count, liver function tests, coagulation profile, and serum uric acid measurement using standard enzymatic colorimetric methods. The severity of chronic liver disease was graded using the Child–Pugh classification. Data were entered and analyzed using SPSS version 20. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequency and percentage. Comparative analysis between Child–Pugh classes was performed using one-way ANOVA and chi-square tests. Receiver operating characteristic curve analysis was used to evaluate the diagnostic performance of serum uric acid in predicting disease severity. A p-value less than 0.05 was considered statistically significant.

Results: The mean age of the study population was 52.8 ± 10.4 years, with the majority of patients belonging to the 51–60-year age group (34.7%). Males constituted 68% of the study population, while females accounted for 32%. Alcohol consumption was reported in 46% of patients, while 36% were current smokers. The most common presenting feature was ascites (76%), followed by jaundice (58.7%), spider nevi (54.7%), pedal edema (49.3%), and hepatic encephalopathy (33.3%). Alcoholic liver disease was the most common etiology (46%), followed by NAFLD/NASH (27.3%), viral hepatitis (19.3%), and autoimmune causes (8.3%). The overall mean serum uric acid level was 5.75 ± 1.76 mg/dL. Ultrasonographic findings revealed nodular liver surface in 68% of patients, enlarged portal vein in 57.3%, and splenomegaly in 45.3%. Based on Child–Pugh classification, 32% of patients were categorized as Child A, 37.3% as Child B, and 30.7% as Child C. A significant association was observed between serum uric acid levels and Child–Pugh classification. Mean serum uric acid levels increased progressively from 4.18 ± 1.10 mg/dL in Child A to 5.86 ± 1.25 mg/dL in Child B and 7.12 ± 1.34 mg/dL in Child C ($p < 0.001$). Clinical severity indicators such as ascites, hepatic encephalopathy, and pedal edema showed significantly higher serum uric acid levels with increasing severity. Laboratory parameters including

bilirubin, liver enzymes, and INR also worsened with higher Child–Pugh classes. Hyperuricemia was significantly more common in severe disease groups. Receiver operating characteristic analysis suggested potential cut-off values of serum uric acid for predicting disease severity. These findings indicated that serum uric acid levels were significantly associated with the severity of chronic liver disease.
Conclusions: The present study demonstrated that serum uric acid levels were significantly associated with the severity of chronic liver disease. A progressive increase in serum uric acid levels was observed across Child–Pugh classes, indicating worsening hepatic dysfunction with increasing uric acid levels.
Keywords: Chronic Liver Disease, Child-Turcotte-Pugh, Estimated Glomerular Filtration Rate, Non-Alcoholic Fatty Liver Disease, Model For End-Stage Liver Disease, Serum Uric Acid.
DOI: 10.25258/ijcpr.18.8.13

Introduction

Chronic liver disease (CLD) represents a significant global health burden, encompassing a spectrum of progressive hepatic disorders that ultimately impair liver function and may culminate in cirrhosis, portal hypertension, hepatocellular carcinoma, and liver-related mortality. [1] The etiologies of CLD are diverse and include viral infections such as hepatitis B and C, chronic alcohol consumption, non-alcoholic fatty liver disease (NAFLD), autoimmune hepatitis, and metabolic or genetic liver disorders. [2] Regardless of the initiating factor, chronic hepatocellular injury often progresses through stages of fibrosis and architectural distortion, leading to cirrhosis, which is associated with multiple complications including variceal bleeding, ascites, hepatic encephalopathy, and renal dysfunction. [3]

Accurate assessment of the severity of CLD is critical for timely clinical decision-making, prognostication, prioritization for liver transplantation, and institution of preventive strategies aimed at reducing morbidity and mortality. Conventional methods of assessing CLD severity include histopathological evaluation through liver biopsy, radiological imaging, and the application of validated scoring systems such as Child-Turcotte-Pugh (CTP) classification and Model for End-Stage Disease (MELD) score. [4] Among the biochemical markers increasingly investigated in this context is serum uric acid (SUA), the final enzymatic product of purine metabolism catalyzed by xanthine oxidase. SUA levels are determined by the balance between production in the liver and dietary sources, versus renal and intestinal excretion. [5]

Hyperuricemia, defined as elevated SUA beyond the physiological range, has traditionally been associated with gout and urate nephrolithiasis; however, in recent decades, it has been increasingly recognized as a marker and mediator of systemic metabolic dysregulation. [6] Elevated SUA levels have been implicated in hypertension, cardiovascular disease, chronic kidney disease, metabolic syndrome, and insulin resistance. Given the central role of the liver in uric acid production and metabolism, disturbances in hepatic function inevitably affect SUA dynamics. Studies have suggested that both hypo- and hyperuricemia may be observed in CLD depending on the stage and associated metabolic derangements. [7,8] The mechanistic rationale behind exploring SUA as a predictor of CLD severity is multifactorial. On one hand, increased uric acid production reflects heightened oxidative stress and systemic inflammation—two pivotal mechanisms in the pathogenesis and progression of liver injury. [9] Xanthine oxidase, which generates uric acid, concurrently produces reactive oxygen species (ROS) that exacerbate hepatocellular damage and fibrogenesis. On the other hand, reduced SUA levels in advanced liver disease may reflect impaired hepatic synthesis, malnutrition, diminished muscle mass, or renal dysfunction secondary to hepatorenal physiology.

Therefore, SUA may function as a dynamic marker, with hyperuricemia predominating in early or metabolic-associated liver disease, while declining SUA levels are seen with decompensated cirrhosis. [10] From a clinical perspective, established scoring systems such as MELD and CTP remain the mainstay for assessing CLD severity and guiding liver transplantation candidacy. However, both systems have inherent limitations as in they are comparatively complicated, and CTP classifications and liver cirrhosis assessments often take into account Fibroscan findings, which are both expensive and not always available in resource poor settings. The addition of auxiliary biomarkers such as SUA to these scoring systems could potentially enhance their predictive accuracy and provide a more holistic picture of disease progression. Several studies have proposed composite indices combining SUA with other laboratory parameters to improve prognostic models. Such integration, if

validated, could revolutionize routine clinical evaluation and decision-making for CLD patients.

Materials and Methods

The present study was hospital-based observational cross-sectional design and conducted in the Department of General Medicine, GMERS Medical College and Hospital, Gotri, Vadodara. The total duration of the study was one year, conducted from January 2024 to December 2025. Patients aged ≥18 years, diagnosed with chronic liver disease (CLD) and attending the outpatient department or admitted to the General Medicine wards and provided informed consent for participation were included. All individuals were evaluated clinically, biochemically, and radiologically as per standard departmental protocol. Patients with acute liver failure or acute-on-chronic liver failure, malignancies or other systemic diseases known to significantly alter serum uric acid levels, Pregnant women, on medications affecting uric acid metabolism (e.g., uricosuric drugs, cytotoxic drugs) were excluded. A total of 150 patients fulfilling the eligibility criteria were enrolled consecutively during the study period by Using the standard formula $n = Z^2PQ / E^2$, where Z represents the standard normal deviate (1.96), P is the expected proportion (10%) [as described by Dhand and Khatkar (2014).], Q is (1 − P), and E is the allowable error (5%). A consecutive sampling technique was employed for participant recruitment. Data collection was carried out over a period of one year following the receipt of institutional ethics approval. All eligible patients presenting to the Department of General Medicine at GMERS Medical College and Hospital, Gotri, either through the outpatient department or inpatient wards, were screened for inclusion. After obtaining informed written consent, each participant was evaluated using a structured and pretested proforma. Sociodemographic details such as age, sex, residence, socioeconomic status, and educational background were recorded. A comprehensive history was elicited, which included the duration and nature of symptoms related to chronic liver disease, alcohol consumption, smoking habits, and other relevant lifestyle factors. Each patient then underwent a thorough clinical examination, during which vital parameters—including pulse rate, blood pressure, respiratory rate, temperature, and body mass index—were measured. Clinical signs associated with chronic liver disease, such as jaundice, ascites, pedal edema, hepatic encephalopathy, and spider nevi, were systematically assessed and documented. Subsequently, venous blood samples were collected under aseptic precautions for laboratory investigations, including complete blood count, liver function tests, coagulation profile, and measurement of serum uric acid using the standard enzymatic colorimetric method. Radiological evaluation in the form of abdominal ultrasonography was performed by qualified radiologists to assess liver echotexture, surface nodularity, portal vein diameter, and spleen size. Based on the combined clinical, laboratory, and radiological findings, the severity of chronic liver disease was graded using the Child–Pugh classification. All collected data were verified for accuracy, anonymized, and compiled for further statistical analysis. The collected data were compiled and analyzed using Statistical Package for the Social Sciences (SPSS) software, version 20.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages. Comparative analysis between different Child–Pugh classes was performed using the chi-square test for categorical variables and one-way ANOVA for continuous variables. Receiver Operating Characteristic (ROC) curve analysis was carried out to determine the diagnostic performance of serum uric acid in predicting severe chronic liver disease. A p-value of less than 0.05 was considered statistically significant for all tests.

Results

The age distribution of the 150 patients with chronic liver disease reveals that the majority were middle-aged to elderly adults. The largest proportion belonged to the 51–60-year age group (34.7%). Patients above 60 years constituted 22.6% of the cohort, while only a small fraction were younger than 40 years. The mean age of the study population was 52.8 ± 10.4 years. males constituted a significantly higher proportion (68%), while females accounted for 32%. Urban residents made up the majority of the study group (59.3%), whereas 40.7% were from rural areas. The largest group belonged to the lower middle class (29.3%), followed closely by the upper middle (25.3%) and upper lower class (24%). 27.3% of patients being illiterate and 22% having completed only primary education. Alcohol consumption was reported by 46% of patients, making it the most prevalent substance-related risk factor. Current smoking was observed in 36%. Ascites was the most common presenting feature, documented in 76% of patients, followed by jaundice (58.7%) and spider nevi (54.7%). Pedal edema was present in nearly half (49.3%) of the cohort, while hepatic encephalopathy was seen in 33.3%. Laboratory evaluation demonstrated anemia (mean hemoglobin 10.9 ± 2.1 g/dL) and thrombocytopenia (mean platelet count 1.4 ± 0.6 lakh/cc), consistent with hypersplenism and chronic disease. Liver function tests were notably deranged, with elevated bilirubin (3.8 ± 2.6 mg/dL), SGOT (98 ± 63.3 U/L), SGPT (78.4 ± 45.6 U/L), and ALP (182.9 ± 61.8 U/L). Hypoalbuminemia (2.8 ± 0.5 g/dL) and elevated

INR (1.8 ± 0.5) further indicated impaired synthetic function. Mean serum uric acid was 5.75 ± 1.76 mg/dL. Assessment of liver disease severity using the Child–Pugh Score showed that 32% of patients were categorized as Child A, 37.3% as Child B, and 30.7% as Child C. The etiological distribution of chronic liver disease showed that alcoholic liver disease was the most common cause, accounting for 69 patients (46.0%). Non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH) was identified in 41 patients (27.3%). Viral hepatitis was present in 29 patients (19.3%), while autoimmune and other etiologies were observed in 11 patients (8.3%). The corresponding mean serum uric acid levels for these etiologies were 6.30 ± 1.62 mg/dL for alcoholic liver disease, 5.75 ± 1.48 mg/dL for NAFLD/NASH, 4.90 ± 1.29 mg/dL for viral hepatitis, and 4.60 ± 1.18 mg/dL for autoimmune and other causes. No statistical significance test was reported for this table. Table 1 show that the association between clinical manifestations and Child–Pugh classification demonstrated several statistically significant findings. The presence of jaundice was significantly associated with the severity of chronic liver disease ($\chi^2 = 17.423$, $p < 0.001$). Jaundice was present in 37.5% of patients in Child–Pugh Class A, 64.3% in Class B, and 73.9% in Class C. The mean serum uric acid level among patients with jaundice was 6.39 ± 1.43 mg/dL. Ascites severity also showed a statistically significant association with Child–Pugh classification ($\chi^2 = 22.891$, $p = 0.002$). Among patients without ascites, 41.7% belonged to Child–Pugh Class A, 21.4% to Class B, and 8.7% to Class C. Mild ascites was observed in 37.5% of Child A patients, 46.4% of Child B patients, and 39.1% of Child C patients. Moderate ascites was seen in 16.7%, 21.4%, and 26.1% of patients in Child A, B, and C respectively, while severe ascites was present in 4.2% of Child A, 10.7% of Child B, and 26.1% of Child C patients. Mean serum uric acid levels increased across the ascites categories, ranging from 4.36 ± 1.01 mg/dL in patients without ascites to 7.08 ± 1.34 mg/dL in those with severe ascites. Hepatic encephalopathy was also significantly associated with Child–Pugh classification ($\chi^2 = 34.225$, $p < 0.001$). Among patients without encephalopathy, 91.7% belonged to Child–Pugh Class A, 67.9% to Class B, and 39.1% to Class C. Grade I–II encephalopathy was observed in 8.3% of Child A, 25.0% of Child B, and 43.5% of Child C patients. Grade III–IV encephalopathy was absent in Child A but was seen in 7.1% of Child B and 17.4% of Child C patients. Mean serum uric acid levels were 5.08 ± 1.22 mg/dL in patients without encephalopathy, 6.57 ± 1.29 mg/dL in Grade I–II, and 7.41 ± 1.36 mg/dL in Grade III–IV. Pedal edema also showed a statistically significant association with Child–Pugh classification ($\chi^2 = 28.456$, $p < 0.001$). It was present in 25.0% of patients in Child–Pugh Class A, 46.4% in Class B, and 78.3% in Class C. The mean serum uric acid level among patients with pedal edema was 6.41 ± 1.39 mg/dL. Similarly, spider nevi demonstrated a statistically significant relationship with Child–Pugh classification ($\chi^2 = 22.093$, $p < 0.001$). Spider nevi were present in 33.3% of patients in Child–Pugh Class A, 53.6% in Class B, and 78.3% in Class C. The mean serum uric acid level among patients with spider nevi was 6.29 ± 1.36 mg/dL. Table 2 show that Hemoglobin levels decreased significantly across groups ($p < 0.001$), suggesting increasing anemia in advanced disease due to hypersplenism, malnutrition, and chronic illness. Platelet counts displayed a strong decline from Child A to C (1.9 → 1.3 → 0.9 $\times 10^5/\mu L$), consistent with progressive portal hypertension and splenic sequestration. Markers of hepatocellular injury—SGOT, SGPT, and ALP—increased significantly with disease severity, indicating worsening hepatocyte dysfunction and cholestasis. Serum bilirubin rose sharply (1.9 mg/dL in Child A to 6.2 mg/dL in Child C), demonstrating impaired excretory function. Conversely, serum albumin, a key synthetic function indicator, showed a consistent decline (3.4 → 2.8 → 2.1 g/dL), significantly correlating with worse Child-Pugh class ($p < 0.001$). Serum globulin levels increased progressively due to chronic inflammation and impaired immunoglobulin clearance. Notably, INR showed one of the most dramatic changes, rising from 1.32 in Child A to 2.41 in Child C ($p < 0.001$), indicating severe compromise of hepatic synthesis of clotting factors. Table 3 and figure 1 show that Mean SUA levels progressively declined with worsening Child-Pugh class: 7.12 mg/dL in Child C, 5.86 mg/dL in Child B, and 4.18 mg/dL in Child A ($p < 0.001$), showing a significant positive association with severity of CLD.

Table 1: Association between clinical findings and Child-Pugh Score in the patients (n=150)

Clinical Finding	Child A (n=48)	Child B (n=56)	Child C (n=46)	Total (N=150)	Chi-Square Value	p-value	Serum uric acid
Jaundice Present	18 (37.5%)	36 (64.3%)	34 (73.9%)	88 (58.7%)	17.423	<0.001*	6.39 ± 1.43
Ascites							
• None	20 (41.7%)	12 (21.4%)	4 (8.7%)	36 (24.0%)	22.891	0.002*	4.36 ± 1.01
• Mild	18 (37.5%)	26 (46.4%)	18 (39.1%)	62 (41.3%)			5.54 ± 1.18
• Moderate	8 (16.7%)	12 (21.4%)	12 (26.1%)	32 (21.3%)			6.31 ± 1.26
• Severe	2 (4.2%)	6 (10.7%)	12 (26.1%)	20 (13.4%)			7.08 ± 1.34
Hepatic Encephalopathy							
• None	44 (91.7%)	38 (67.9%)	18 (39.1%)	100 (66.7%)	34.225	<0.001*	5.08 ± 1.22
• Grade I–II	4 (8.3%)	14 (25.0%)	20 (43.5%)	38 (25.3%)			6.57 ± 1.29
• Grade III–IV	0 (0%)	4 (7.1%)	8 (17.4%)	12 (8.0%)			7.41 ± 1.36
Pedal Edema	12 (25.0%)	26 (46.4%)	36 (78.3%)	74 (49.3%)	28.456	<0.001*	6.41 ± 1.39
Spider nevi	16 (33.3%)	30 (53.6%)	36 (78.3%)	82 (54.7%)	22.093	<0.001*	6.29 ± 1.36

Table 2: Association between laboratory parameters and Child-Pugh Score in the patients (n=150)

Laboratory Parameter	Child A (Mean ± SD) (n=48)	Child B (Mean ± SD) (n=56)	Child C (Mean ± SD) (n=46)	ANOVA / Chi-square	p-value
Hemoglobin (g/dL)	11.8 ± 1.9	10.6 ± 1.8	9.8 ± 1.7	F = 14.32	<0.001*
TLC (×10³/μL)	7.4 ± 2.1	7.7 ± 2.4	7.1 ± 2.2	F = 1.25	0.216
Platelets (×10⁵/μL)	1.9 ± 0.5	1.3 ± 0.5	0.9 ± 0.4	F = 39.48	<0.001*
Serum Bilirubin (mg/dL)	1.9 ± 0.8	3.8 ± 1.7	6.2 ± 2.3	F = 82.14	<0.001*
SGOT (U/L)	72 ± 41	94 ± 58	135 ± 68	F = 15.92	<0.001*
SGPT (U/L)	58 ± 30	78 ± 35	116 ± 50	F = 24.77	<0.001*
ALP (U/L)	148 ± 44	186 ± 52	232 ± 58	F = 29.33	<0.001*
Serum Albumin (g/dL)	3.4 ± 0.3	2.8 ± 0.4	2.1 ± 0.3	F = 128.6	<0.001*
Serum Globulin (g/dL)	3.1 ± 0.8	3.8 ± 0.9	4.4 ± 1.0	F = 22.48	<0.001*
INR	1.32 ± 0.16	1.82 ± 0.25	2.41 ± 0.32	F = 198.7	<0.001*

Table 3: Association between serum uric acid and Child-Pugh Score in the patients (n=150)

Serum Uric Acid Parameter	Child A (n = 48)	Child B (n = 56)	Child C (n = 46)	Statistical Test	p-value
Mean SUA (mg/dL)	4.18 ± 1.10	5.86 ± 1.25	7.12 ± 1.34	ANOVA F = 64.55	<0.001*
Hyperuricemia (>7 mg/dL in men, >6 mg/dL in women)	28 (58.3%)	18 (32.1%)	6 (13.0%)	$\chi^2 = 32.84$	<0.001*
Normal SUA levels	20 (41.7%)	38 (67.9%)	40 (87.0%)		

Discussion

The present study demonstrated that the majority of patients with chronic liver disease were middle-aged to elderly, with a mean age of 52.8 ± 10.4 years. The largest proportion belonged to the 51–60-year age group (34.7%), followed by 41–50 years (26.7%). These findings were consistent with Gupta et al. (2021), who reported that chronic liver disease predominantly affected middle-aged individuals, with progressive disease severity observed with advancing age. [5]

The present study also demonstrated higher serum uric acid levels in older patients, rising from 3.92 ± 0.88 mg/dL in younger patients to 6.74 ± 1.42 mg/dL in those above 60 years. Similar trends were observed by Liu et al. (2023), who reported that each 1 mg/dL increase in uric acid significantly increased the risk of chronic liver disease. [21] Male predominance was observed in the present study (68%), which was consistent with findings by Gupta et al. (2021), who reported 76% male participants. This may be attributed to higher alcohol consumption among males. [5] The majority of patients belonged to urban areas (59.3%), reflecting lifestyle-related risk factors. Educational and socioeconomic distributions were relatively uniform, with the lower middle class constituting 29.3%. Similar demographic patterns were observed by Oral et al. (2019), who reported that lifestyle factors and socioeconomic status influenced liver disease prevalence. [19] Additionally, substance use was common, with alcohol consumption in 46% and smoking in 36%

of patients. These findings aligned with Lombardi et al. (2016), who emphasized lifestyle factors as major contributors to liver disease progression. [17] The present study reported a mean serum uric acid level of 5.75 ± 1.76 mg/dL among patients with chronic liver disease. This finding was comparable to Gupta et al. (2021), who reported a mean uric acid level of 6.69 ± 2.92 mg/dL in chronic liver disease patients. [5] Similarly, Petta et al. (2011) reported an average uric acid level of 5.75 mg/dL among NAFLD patients, closely matching the findings of the present study. [13] These similarities support the hypothesis that uric acid elevation is common in chronic liver disease. The present study also demonstrated variation in uric acid levels across etiologies, with alcoholic liver disease showing the highest mean levels (6.30 ± 1.62 mg/dL), followed by NAFLD/NASH (5.75 ± 1.48 mg/dL), viral hepatitis (4.90 ± 1.29 mg/dL), and autoimmune causes (4.60 ± 1.18 mg/dL). Similar findings were observed by Sertoglu et al. (2014), who reported higher uric acid levels in NASH compared to simple steatosis. [16] Oral et al. (2019) also reported elevated uric acid levels among NAFLD patients compared to controls. [19] These findings indicated that uric acid levels vary depending on the underlying etiology of liver disease. The present study demonstrated a significant association between serum uric acid levels and the severity of chronic liver disease as assessed by Child–Pugh classification. Mean serum uric acid levels showed a progressive increase across Child–Pugh classes, with values of 4.18 ± 1.10 mg/dL in Child–Pugh Class A, 5.86 ± 1.25 mg/dL in Class B, and 7.12 ± 1.34 mg/dL in Class C, indicating a statistically significant trend ($p < 0.001$). This progressive increase suggested that worsening hepatic dysfunction was associated with higher serum uric acid levels. These findings supported the hypothesis that serum uric acid may serve as a marker of disease severity in chronic liver disease. Similar observations were reported by Gupta et al. (2021), who demonstrated a progressive increase in serum uric acid levels with advancing Child–Turcotte–Pugh class, with mean values of 4.03 mg/dL in Class A, 5.17 mg/dL in Class B, and 8.94 mg/dL in Class C. These comparable findings strengthened the evidence that elevated uric acid levels correlate with worsening liver disease severity. The present study also demonstrated a significant association between serum uric acid levels and clinical manifestations of chronic liver disease. Patients with jaundice had higher serum uric acid levels (6.39 ± 1.43 mg/dL), and increasing severity of ascites showed progressively elevated uric acid levels from 4.36 ± 1.01 mg/dL in patients without ascites to 7.08 ± 1.34 mg/dL in those with severe ascites. Similarly, hepatic encephalopathy severity correlated with increasing serum uric acid levels, with 5.08 ± 1.22 mg/dL in patients without encephalopathy, 6.57 ± 1.29 mg/dL in Grade I–II, and 7.41 ± 1.36 mg/dL in Grade III–IV encephalopathy. These findings indicated that serum uric acid levels increased in parallel with worsening clinical features of liver dysfunction. Comparable findings were reported by Afzali et al. (2010), who demonstrated that elevated serum uric acid levels were independently associated with cirrhosis and abnormal liver enzymes, suggesting that uric acid may reflect hepatic injury and disease progression. [12] In addition to clinical findings, the present study also demonstrated significant associations between ultrasonographic parameters and serum uric acid levels. Patients with nodular liver surface had higher uric acid levels (6.32 ± 1.41 mg/dL), while those with enlarged portal vein and splenomegaly had mean uric acid levels of 6.68 ± 1.33 mg/dL and 6.48 ± 1.31 mg/dL respectively. These findings indicated that serum uric acid levels increased in patients with features of portal hypertension and advanced cirrhosis. Similar observations were reported by Wang et al. (2015), who demonstrated that elevated serum uric acid to creatinine ratio was significantly associated with increased hepatic steatosis severity and disease progression. [10] The present study also demonstrated significant associations between serum uric acid and laboratory parameters of liver dysfunction. Progressive decline in hemoglobin and platelet count and increase in bilirubin, liver enzymes, and INR were observed with worsening Child–Pugh classification. These findings indicated that serum uric acid levels increased with worsening hepatic synthetic and excretory dysfunction. Similar findings were reported by Petta et al. (2011), who demonstrated that hyperuricemia was associated with higher NAFLD activity scores and liver fibrosis. [13] Huang et al. (2016) further reported that hyperuricemia was independently associated with severe lobular inflammation and advanced disease. These findings reinforced the association between elevated uric acid levels and worsening liver disease severity. [18] Several large cohort studies also support these findings. Ryu et al. (2011) reported that higher serum uric acid levels predicted incident fatty liver disease, with hazard ratios increasing progressively across uric acid quartiles. [14] Similarly, Sirota et al. (2013) demonstrated that individuals in the highest uric acid quartile had significantly higher odds of NAFLD and more severe disease. [15]

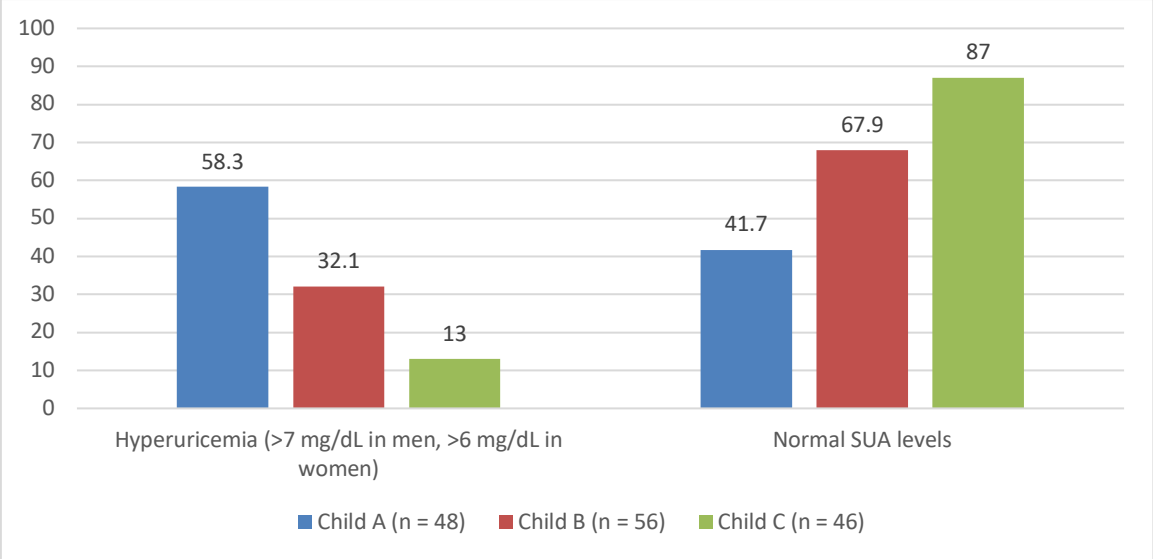

Figure 1: Association between serum uric acid and Child-Pugh Score in the patients (n=150)

The present study attempted to identify potential cut-off values of serum uric acid corresponding to increasing severity of chronic liver disease. A progressive rise in serum uric acid levels was observed across Child–Pugh classes, with mean values of 4.18 ± 1.10 mg/dL in Child-Pugh Class A, 5.86 ± 1.25 mg/dL in Class B, and 7.12 ± 1.34 mg/dL in Class C, demonstrating a statistically significant association ($p < 0.001$).

These findings suggested that serum uric acid levels increased proportionately with worsening liver dysfunction and could potentially serve as a surrogate marker for disease severity stratification. The identification of such threshold values is clinically relevant, as it may allow early recognition of patients at risk of advanced liver disease and facilitate timely intervention. The progressive increase in serum uric acid levels observed in the present study was comparable to findings reported by Gupta et al. (2021), who demonstrated rising uric acid levels with increasing Child-Turcotte-Pugh classification, with mean levels of 4.03 mg/dL in Child A, 5. [17] mg/dL in Child B, and 8.94 mg/dL in Child C. [5] these findings were similar to those of the present study, indicating that serum uric acid may reflect worsening hepatic function.

Furthermore, Afzali et al. (2010) reported that individuals in the highest serum uric acid tertile (>6 mg/dL) had significantly higher risk of cirrhosis-related hospitalization or death, suggesting that uric acid levels above 6 mg/dL may represent clinically meaningful thresholds for advanced liver disease.12 Similarly, Lee et al. (2010) demonstrated a graded increase in NAFLD incidence across uric acid quartiles, with incidence rising from 5.6% in the lowest quartile to 20.9% in the highest quartile, indicating that increasing uric acid levels are associated with progressive hepatic involvement. [11]

Several studies evaluating serum uric acid to creatinine ratios have also supported the concept of threshold values. Liu et al. (2023) reported that higher serum uric acid to creatinine ratio (6.14 ± 1.55) was significantly associated with moderate-to-severe metabolic dysfunction-associated fatty liver disease compared to mild disease (5.51 ± 1.19). [21] Similarly, Choi et al. (2023) demonstrated that increasing serum uric acid to creatinine ratio significantly correlated with mild and moderate-to-severe NAFLD, with odds ratios of 1.147 and 1.275 respectively. [20] These findings reinforced that rising uric acid levels are associated with disease progression and can help stratify severity.

The present study demonstrated that serum uric acid levels increased significantly with worsening chronic liver disease severity. Elevated uric acid was associated with clinical manifestations, laboratory abnormalities, and ultrasonographic findings. These findings supported previous studies and suggested that serum uric acid may serve as a simple, cost-effective biomarker for disease severity assessment.

Conclusion

The present study demonstrated that serum uric acid levels were significantly associated with the severity of chronic liver disease. A progressive increase in serum uric acid levels was observed across Child–Pugh classes, indicating worsening hepatic dysfunction with increasing uric acid levels. Clinical manifestations such as ascites, hepatic encephalopathy, jaundice, and pedal edema were associated with elevated serum uric acid levels. Similarly, laboratory parameters indicating

hepatic dysfunction showed worsening trends with increasing uric acid levels.

Ultrasonographic findings of advanced liver disease, including nodular liver surface, portal hypertension, and splenomegaly, were also associated with higher serum uric acid levels. These findings suggested that serum uric acid may reflect both functional and structural liver damage. The identification of potential cut-off values further supported the utility of serum uric acid as a surrogate biomarker for disease severity stratification.

References

1. Younossi ZM, Wong G, Anstee QM, Henry L. The global burden of liver disease. Clin Gastroenterol Hepatol. 2023;21(8):1978-91.
2. Paik JM, Golabi P, Younossi Y, Mishra A, Younossi ZM. Changes in the global burden of chronic liver diseases from 2012 to 2017: the growing impact of NAFLD. Hepatology. 2020;72(5):1605-16.
3. Paik JM, Kabbara K, Eberly KE, Younossi Y, Henry L, Younossi ZM. Global burden of NAFLD and chronic liver disease among adolescents and young adults. Hepatology. 2022;75(5):1204-17.
4. Asrani SK, Kouznetsova M, Ogola G, Taylor T, Masica A, Pope B, et al. Increasing health care burden of chronic liver disease compared with other chronic diseases, 2004–2013. Gastroenterology. 2018;155(3):719-29.
5. Gupta PK, Agarwal H, Singhal S, Manocha K. Study of association between serum uric acid levels and chronic liver disease. Eur J Mol Clin Med. 2021;8(4):1169-74.
6. Zhou Y, Wei F, Fan Y. High serum uric acid and risk of nonalcoholic fatty liver disease: a systematic review and meta-analysis. Clin Biochem. 2016;49(7-8):636-42.
7. Rao VV. Impact of uric acid on chronic liver disease: relationship with hepatic encephalopathy and liver function tests. J Contemp Clin Pract. 2019;5:90-4.
8. Hasan R, Roy PK, Khan MM, Ahmed F, Alam MR, Gain G. Relation of serum uric acid concentrations with etiology and severity in patients with cirrhosis of liver. Bangladesh Med J. 2021;50(1):46-51.
9. Mendoza YP, Tsouka S, Semmler G, Seubnooch P, Freiburghaus K, Mandorfer M, et al. Metabolic phenotyping of patients with advanced chronic liver disease for better characterization of cirrhosis regression. J Hepatol. 2024;81(6):983-94.
10. Wang F, Zheng X, Ni X, Li Y. Increased serum uric acid to creatinine ratio is associated with metabolic dysfunction-associated steatotic liver disease. Sci Rep. 2025;15(1):26941.
11. Lee JW, Cho YK, Ryan MC, Kim H, Lee SW, Chang E, et al. Serum uric acid as a predictor for development of nonalcoholic fatty liver disease in apparently healthy subjects: a 5-year retrospective cohort study. Gut Liver. 2010;4(3):378.
12. Afzali A, Weiss NS, Boyko EJ, Ioannou GN. Association between serum uric acid level and chronic liver disease in the United States. Hepatology. 2010;52(2):578-89.
13. Petta S, Cammà C, Cabibi D, Di Marco V, Craxì A. Hyperuricemia is associated with histological liver damage in patients with non-alcoholic fatty liver disease. Aliment Pharmacol Ther. 2011;34(7):757-66.
14. Ryu S, Chang Y, Kim SG, Cho J, Guallar E. Serum uric acid levels predict incident nonalcoholic fatty liver disease in healthy Korean men. Metabolism. 2011;60(6):860-6.
15. Sirota JC, McFann K, Targher G, Johnson RJ, Chonchol M, Jalal DI. Elevated serum uric acid levels are associated with non-alcoholic fatty liver disease independently of metabolic syndrome. Metabolism. 2013;62(3):392-9.
16. Sertoglu E, Ercin CN, Celebi G, Gurel H, Kayadibi H, Genc H, et al. Relationship of serum uric acid with non-alcoholic fatty liver disease. Clin Biochem. 2014;47(6):383-8.
17. Lombardi R, Pisano G, Fargion S. Role of serum uric acid and ferritin in development and progression of NAFLD. Int J Mol Sci. 2016;17(4):548.
18. Huang Q, Yu J, Zhang X, Liu S, Ge Y. Association of serum uric acid level with liver histology in biopsy proven non-alcoholic fatty liver disease. Biomed Rep. 2016;5(2):188-92.
19. Oral A, Sahin T, Turker F, Kocak E. Relationship between serum uric acid levels and nonalcoholic fatty liver disease in non-obese patients. Medicina. 2019;55(9):600.
20. Choi J, Joe H, Oh JE, Cho YJ, Shin HS, Heo NH. Correlation between NAFLD and serum uric acid-to-serum creatinine ratio. PLoS One. 2023;18(7):e0288666.
21. Liu Z, Wang Q, Huang H, Wang X, Xu C. Association between serum uric acid levels and long-term mortality of metabolic dysfunction-associated fatty liver disease. Diabetol Metab Syndr. 2023;15(1):27.