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Original Research Article

Study of serum uric acid level in chronic liver disease and its association with Child-Turcotte-Pugh and model for end-stage liver disease score

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ABSTRACT

Background: Chronic liver disease (CLD) involves progressive hepatic injury, inflammation, and fibrosis, potentially leading to cirrhosis, liver failure, and increased mortality. Despite advancements, CLD remains a significant clinical challenge due to its varied nature and course. Serum uric acid (SUA), a by-product of purine metabolism, is emerging as a potential marker for CLD progression and prognosis. This study investigates the correlation between SUA levels and CLD severity, assessed by Child-Turcotte-Pugh (CTP) and model for end-stage liver disease (MELD) scores.
Methods: This cross-sectional study at GSVM medical college, Kanpur, included 54 CLD patients over 18 years. Exclusions were pregnancy, known gout, diabetes mellitus, recent surgery/trauma, chronic kidney disease, hypothyroidism, specific medications, and malignancies on chemotherapy. Data were collected via interviews, medical records, and lab tests (SUA, liver function). Liver disease severity was assessed using CTP and MELD scores. Statistical analyses examined relationships between SUA levels, CTP and MELD scores.
Results: SUA exhibit a positive correlation with the CTP score ($r=0.51$, $p<0.05$). Mean SUA levels were 4.20±0.45 mg/dL for CTP A, 6.46±2.18 mg/dl for CTP B, and 8.83±2.55 mg/dl for CTP C. An ANOVA test showed a significant association (F value: 11.78, $p<0.05$). A significant positive correlation was found between MELD scores and SUA levels ($r=0.438$, $p<0.05$).
Conclusions: The significant associations between SUA levels and CTP and MELD scores indicate that SUA could be a valuable biomarker in managing CLD. Incorporating SUA measurements may enhance prognostic accuracy and support personalized treatments. Further research is needed to validate these findings across diverse populations and clinical settings, potentially identifying new therapeutic targets.

Keywords: CLD, SUA, CTP score, MELD score, Liver cirrhosis, Liver function tests

INTRODUCTION

Chronic liver disease (CLD) represents a significant global health burden, characterized by progressive liver damage that can lead to cirrhosis, liver failure, and increased mortality. Among the various biochemical abnormalities associated with CLD, SUA levels have garnered increasing attention. SUA, the end product of purine metabolism, is primarily excreted by the kidneys but is also closely linked to hepatic function. In the context of CLD, impaired liver function can disrupt uric acid metabolism, potentially leading to hyperuricemia.[1,2]

This dysregulation may be influenced by various factors, including reduced urea synthesis, altered renal function, and increased lactic acid production. Elevated SUA levels have been associated with liver fibrosis, inflammation, and oxidative stress, and may also contribute to the development of complications such as hepatocellular carcinoma and portal hypertension.[3,4]

Despite growing interest in the relationship between SUA and CLD, the literature remains inconclusive, with conflicting findings regarding its clinical significance. Exploring the link between SUA levels and established prognostic tools like the CTP score and the MELD score could provide valuable insights into the role of SUA in the pathophysiology and prognosis of CLD.[5,6]

Therefor this study is carried out to investigate the correlation between SUA levels and CLD severity, assessed by CTP and MELD scores.

METHODS

Study design

This research was conducted as a cross-sectional study over a period of 16 months, from January 2023 to April 2024. The objective was to assess the SUA level in patients of CLD and Its association with the severity of the disease CTP and MELD score.

Setting

The study was conducted at Ganesh Shankar Vidyarthi memorial medical college, Kanpur, Uttar Pradesh, India. Data were collected from both outpatient department and inpatients wards of department of medicine.

Participants

The study included adult patients aged 18 years and above, of either sex, who were diagnosed with CLD. while exclusion criteria eliminated pregnant patients, those with gout, diabetes mellitus, recent surgery, trauma, chronic kidney disease, hypothyroidism, malignancies, or those on certain medications (e.g., allopurinol, febuxostat, thiazides, furosemide). Informed consent was obtained from all participants before enrollment.

Variables

The primary variables examined were biochemical markers, including complete blood count (CBC), liver function tests (LFT), kidney function tests (KFT), serum electrolytes (SE), prothrombin time-international normalized ratio (PT-INR), serum thyroid-stimulating hormone (TSH), glycated hemoglobin (HbA1c), and SUA levels. Clinical parameters included age, sex, body mass index (BMI), waist circumference, and severity of liver disease based on CTP class and MELD scores.

Data sources/measurement

Data were collected through structured patient interviews, medical records review, and laboratory tests. Biochemical parameters were measured from blood samples, while clinical evaluations were supported by imaging (ultrasonography) and endoscopic investigations. Standardized protocols were followed to ensure accuracy and consistency of data collection across participants.

Bias

Selection bias was minimized through the use of random sampling, ensuring each eligible patient had an equal chance of being selected. Confounding variables were controlled by adhering to strict inclusion and exclusion criteria. The use of standardized measurement protocols further reduced measurement bias.

Study size

The sample size of 54 patients was calculated based on the prevalence of CLD in India. With a 95% confidence interval and a 5% margin of error, the sample size was determined using the formula for prevalence studies.

Quantitative variables

Quantitative variables included biochemical markers such as SUA levels, LFT, and KFT, among others. Categorical variables such as sex, severity of ascites, and hepatic encephalopathy (HE) grades were analyzed alongside continuous variables like age, BMI, and MELD scores.

Statistical methods

Data were analyzed using the statistical package for social sciences (SPSS) v29.0. Descriptive statistics were computed for demographic and clinical variables. ANOVA test was used to assess associations between SUA with the CTP class. Pearson correlation analysis was done to assess correlation of SUA with CTP and MELD score. $P<0.05$ were considered statistically significant.

RESULTS

The distribution of age of the patient is from 18 to 85 years with maximum belongs to age group 41-50 years (31.5%). Mean age is 45.52 years with SD of 14.54 years (Table 1).

Table 1: Distribution of CLD patients according to age and etiology.

Variables	N	Percentages (%)
Age group (in years)		
18-30	9	16.7
31-40	11	20.4
41-50	17	31.5
51-60	10	18.5
61-70	5	9.3
>70	2	3.7
Mean±SD	45.52±14.54	
Etiology		
HCV related	6	11.1
HBV related	9	16.7
Alcoholic	39	72.2

Out of 54 patients 42 were males (77.8%) and 12 were females (22.2%) (Figure 1).

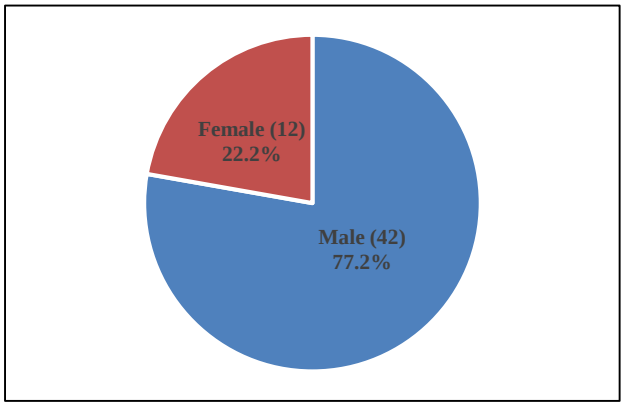

Figure 1: Gender distribution among CLD patients.

Etiological data reveals that 39 cases (72.2%) were alcohol related, 9 cases (16.7%) were HBV related and 6 cases (11.1%) were HCV related (Table 1).

Table 2: Distribution of patients according to severity of ascites and HE.

Variables	N	Percentages (%)
Grades of ascitis		
Mild	7	13.0
Moderate	35	64.8
Severe	12	22.2
HE		
Grade 0	36	66.7
Grade 1	2	3.7
Grade 2	8	14.8
Grade 3	7	13.0
Grade 4	1	1.9

Severity of ascites was also graded in the patients and maximum had moderate ascites (64.8%) followed by severe and mild, 22.2% and 13.0% respectively. Distribution of HE among cases revealed that max cases (36) were not in HE (grade-0) followed by 8 cases in HE grade-2, 7 patients in grade-3, 2 patients in grade-1 and only 1 patient in grade-4 (Table 2).

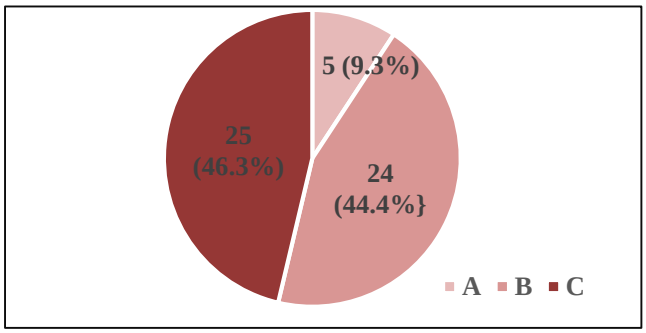

Figure 2: Distribution of patients as per the CTP class.

The 25 patients (46.3%) were in CTP class-C, which was maximum across the class followed by 24 patients (44.4%) in class- B and 5 patients (9.3%) in class-A (Figure 2).

Table 3: Association of CTP Class with SUA level among CLD patients.

CTP score	SUA	
	Mean	SD
A	4.20	0.45
B	6.46	2.18
C	8.83	2.55
ANOVA	F=11.78, p<0.05	

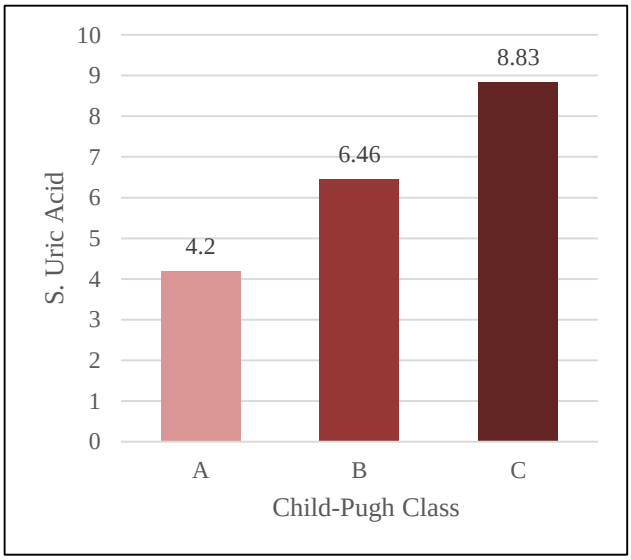

Figure 3: Correlation between SUA and Child-Pugh class.

The mean SUA levels were 4.20±0.45 mg/dl for CTP score A, 6.46±2.18 mg/dl for CTP score B, and 8.83±2.55 mg/dl for CTP score C.

An ANOVA test revealed a significant association, with an f value of 11.78 and a p value of less than 0.05 (Table 3 and Figure 3).

Table 4: Correlation of MELD score with SUA among CLD patients.

Pearson correlation	Between MELD score and SUA	
	R value	P value
Overall	0.438	<0.05
Within CTP=A	-0.411	0.491
Within CTP=B	0.243	0.252
Within CTP=C	0.112	0.593

The correlation between MELD score and SUA levels was examined. Overall, there was a significant positive correlation, with a Pearson correlation coefficient (R value) of 0.438 and a p value of less than 0.05 (Table 4).

Table 5: Correlation of SUA with LFT and other parameters among CLD patients.

Pearson correlation	CTP score	
	R value	P value
S. bilirubin	0.45	<0.05
S. albumin	-0.61	<0.05
PT-INR	0.59	<0.05
S. creatinine	0.13	0.344
MELD score	0.60	<0.05
SUA	0.51	<0.05
SGOT	0.14	0.302
SGPT	-0.14	0.304

Among the cases, CTP score shows significant correlations with various parameters, as determined by Pearson correlation analysis. The S. bilirubin levels exhibit a positive correlation with the CTP score (r=0.45, p<0.05), Conversely, S. albumin levels demonstrate a strong negative correlation (r=-0.61, p<0.05). PT-INR values also show a robust positive correlation with the CTP score (r=0.59, p<0.05) (Table 5).

SUA also exhibit a positive correlation with the CTP score (r=0.51, p<0.05), suggesting a link between uric acid levels and liver function impairment as assessed by the CTP score. However, parameters like S. Creatinine, SGOT, and SGPT show weaker correlations (r<0.15) with the CTP score, indicating less pronounced associations with liver disease severity in this context.

The ROC analysis was performed to evaluate the predictive ability of SUA levels for determining the CTP class among CLD patients. For predicting CTP class B, the area under the receiver operating characteristic curve (AUROC) was 0.896 with a 95% confidence interval (CI) of 0.78 to 1.00. The optimum cut-off value for uric acid was determined to be greater than 5.00. At this threshold, the sensitivity was 79.2% (95% CI: 68.4-90.0), and the specificity was 100% (95% CI: 100-100) (Figure 4).

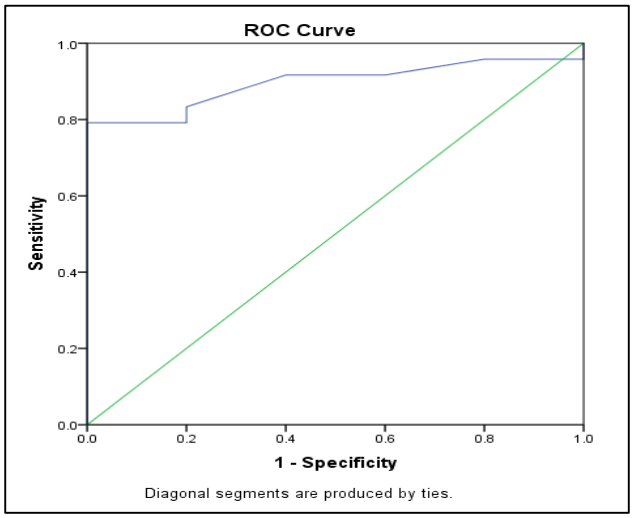

Figure 4: ROC analysis to predict CTP class B.

For predicting CTP class C, the AUROC was 0.807 with a 95% CI of 0.69 to 0.93. The optimum cut-off value for uric acid was greater than 6.05. This cut-off value provided a sensitivity of 96% (95% CI: 90.8-100) and a specificity of 62.1% (95% CI: 49.2-75.0) (Figure 5).

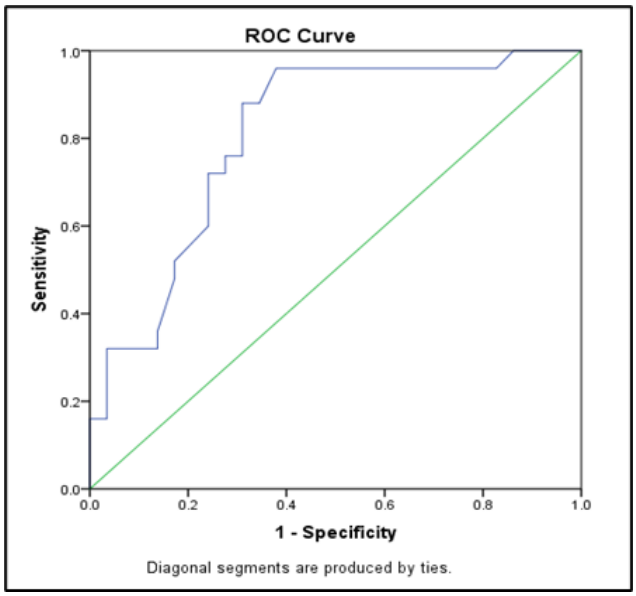

Figure 5: ROC analysis to predict CTP Class C.

DISCUSSION

In this study, we aimed to explore the relationship between SUA levels and the severity of CLD, assessed by the CTP and MELD scores.

Our study found that SUA levels significantly increased as CTP scores advanced from class A to class C, with mean SUA levels of 4.20±0.45 mg/dL for class A, 6.46±2.18 mg/dL for class B, and 8.83±2.55 mg/dl for class C. The statistical significance was confirmed by an ANOVA test (f=11.78, p<0.05), indicating that elevated SUA levels are associated with more severe liver dysfunction. This positive correlation aligns with the findings of Choudhary et al who similarly reported that SUA levels increased with worsening CTP class. Their research underscored that SUA is a reliable marker of liver impairment across different patient populations, suggesting that SUA could help identify patients at higher risk of liver disease complications.[7]

Our results also concur with those of Gupta et al who explored the association between SUA and liver disease severity in a similar cohort. They reported significantly higher mean SUA levels in patients with advanced liver disease, particularly in CTP classes B and C. Their findings reinforce the utility of SUA as a surrogate marker of liver function deterioration, especially in CLD contexts.[8]

Furthermore, Kumar et al studied the relationship between SUA levels and CTP scores in liver cirrhosis patients.

Their results showed that SUA levels increased with advancing CTP scores, peaking in CTP class C patients. Notably, they found significantly elevated SUA levels in patients with ascites and HE, characteristic of advanced liver disease. This mirrors our observation that higher SUA levels correlate with severe liver disease indicators, such as ascites and encephalopathy.[9]

In our study, we found a significant positive correlation between SUA levels and the MELD score ($r=0.438$, $p<0.05$), indicating that as liver disease severity increased, SUA levels also rose. This finding aligns with Prakash et al who reported a similar positive correlation between SUA and MELD scores in cirrhotic patients. They observed that higher MELD scores corresponded with elevated SUA levels, reinforcing SUA's potential as a biomarker for predicting disease progression and outcomes in CLD patients. Their study emphasized that SUA levels could provide additional prognostic information beyond traditional LFT.[10]

Similarly, Noklang et al assessed the correlation between SUA levels and liver function in chronic liver disease patients in South India. Their findings, consistent with ours, demonstrated that higher SUA levels were significantly associated with increased MELD scores. Noklang et al highlighted SUA's role in identifying patients at greater risk of complications, such as hepatocellular carcinoma and portal hypertension, further supporting the clinical relevance of SUA measurements in CLD management.[11]

Our study also revealed a significant negative correlation between SUA levels and serum albumin levels ($r=-0.36$, $p<0.05$), suggesting that as liver synthetic function deteriorates, SUA levels increase. This aligns with findings from Geetha et al who noted a similar inverse relationship between SUA and serum albumin levels. They concluded that higher SUA levels indicate more severe liver function impairment. Their study also reported a strong positive correlation between SUA and PT-INR, another liver dysfunction marker, which aligns with our observation that SUA correlates positively with PT-INR ($r=0.33$, $p<0.05$).[12]

Geetha et al conducted a ROC analysis to assess the predictive ability of SUA levels for distinguishing between different CTP classes. They found that SUA levels had a high area under the curve (AUC) for predicting CTP classes B and C, reinforcing SUA's potential as a reliable biomarker for identifying patients with advanced liver disease.

The results of our study are consistent with those of the cited studies, particularly regarding the positive correlation between SUA levels and liver disease severity measured by the CTP and MELD scores. The trends observed reinforce the conclusion that SUA levels can serve as valuable biomarkers for assessing liver function and predicting disease outcomes in CLD patients.

CONCLUSION

This study highlights the significant relationship between SUA levels and the severity of CLD as assessed by the CTP and MELD scores. The findings demonstrate that elevated SUA levels correlate positively with higher CTP and MELD scores, indicating greater liver function impairment. Specifically, SUA levels were notably higher in patients with advanced CLD, characterized by CTP class B and C, and higher MELD scores. The strong positive correlations between SUA levels and markers of liver dysfunction, such as serum bilirubin, PT-INR, and serum creatinine, further suggest that SUA could serve as a valuable biomarker for assessing the progression and severity of CLD. The ROC analysis confirmed the predictive ability of SUA levels, particularly for distinguishing between different CTP classes, with high sensitivity and specificity at identified cut-off points. These results underscore the potential clinical utility of incorporating SUA measurements into routine assessments of CLD patients. By enhancing prognostic accuracy, SUA could support more personalized treatment strategies and better management of patients with CLD. However, further studies involving larger and more diverse populations are warranted to validate these findings and explore the underlying mechanisms linking SUA with liver disease progression.

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Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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