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ORIGINAL RESEARCH

Liver function tests in patients with alcohol use disorder: Findings from a tertiary care Psychiatry unit in Mumbai

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ABSTRACT

Alcohol-related liver disease (ARLD) spans a pathological spectrum from hepatic steatosis to fibrosis and cirrhosis. Liver function tests (LFTs) including aminotransferases, bilirubin, and albumin constitute the standard first-line biochemical evaluation in patients with suspected hepatic injury. However, a clinically important challenge is that routine LFT values can remain within normal limits even in the presence of significant underlying liver pathology. This phenomenon has particular relevance in patients with alcohol use disorder (AUD), among whom hepatic damage may be advanced at the time of clinical presentation. This was a retrospective chart analysis conducted at a tertiary care centre in Mumbai. Indoor case records of all patients admitted to the psychiatry ward between January 2020 and December 2021 with a diagnosis of alcohol use disorder and a minimum of 12 months of regular alcohol consumption were reviewed. Demographic characteristics, type and pattern of alcohol consumption, comorbid substance use, LFT parameters (total bilirubin, serum glutamic-oxaloacetic transaminase, serum glutamic-pyruvic transaminase, and clinical signs of hepatic dysfunction were systematically extracted. One hundred male patients with AUD were included (mean age 38.35 ± 11.1 years; range 18–61 years). Although 55% and 36% of patients had AST and ALT values exceeding the upper limit of the reference range, respectively, clinically significant elevations were present in only 7% (AST) and 5% (ALT) of cases. An AST:ALT ratio > 2, suggestive of alcoholic liver injury, was identified in only 19 patients (19%). Only 6 patients exhibited clinical signs of hepatic decompensation. The majority of patients thus had normal or only mildly elevated LFT values despite chronic alcohol use. A substantial proportion of patients with alcohol use disorder may exhibit normal or minimally deranged liver enzyme levels despite prolonged alcohol exposure. These findings underscore the inadequacy of LFTs as sole screening tools for alcohol-related liver disease and support the use of complementary non-invasive modalities including transient elastography (FibroScan), FIB-4 index, and APRI score in the clinical evaluation of this population.

Keywords: Alcohol use disorder; Liver function tests; Aminotransferases; AST:ALT ratio; Alcohol-related liver disease; FibroScan; Transient elastography; Hepatic fibrosis

INTRODUCTION

Alcohol consumption is one of the principal causes of chronic liver disease globally and represents a major contributor to preventable morbidity and mortality worldwide [1]. Alcohol-related liver disease (ARLD) encompasses a well-characterised pathological spectrum, progressing from simple hepatic steatosis the earliest and most common manifestation through alcoholic hepatitis, hepatic fibrosis, and, at its most advanced stage, cirrhosis and its associated complications [1,2]. The rate of progression along this spectrum is determined by a complex interaction of factors including the duration and quantity of alcohol intake, genetic predisposition, sex, nutritional status, concurrent metabolic disorders, and the presence of co-existing hepatic insults [3].

The hepatotoxic mechanisms underpinning ARLD are multiple and interdependent. Chronic ethanol exposure promotes hepatocellular injury through oxidative stress, mitochondrial dysfunction, inflammatory cytokine cascades particularly involving tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) and the direct cytotoxicity of acetaldehyde, the principal reactive metabolite of ethanol oxidation [1,2]. These processes culminate in hepatocyte necrosis and the activation of hepatic stellate cells, driving progressive fibrogenesis and, ultimately, architectural distortion of the hepatic parenchyma [1].

From an epidemiological perspective, alcohol is estimated to account for approximately 50% of all cases of liver cirrhosis worldwide, imposing a substantial global burden of liver-related morbidity and mortality.[4] In developing countries, this burden is compounded by delayed diagnosis attributable to limited healthcare access, stigma associated with alcohol use, and the insidious clinical course of ARLD resulting in patients presenting at advanced and often irreversible stages of disease [5, 6].

Liver function tests (LFTs), encompassing aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), albumin, and prothrombin time, remain the most widely utilised and readily available initial biochemical tools for identifying and monitoring hepatic injury [7]. In the context of ARLD, AST and ALT are employed as the primary markers of hepatocellular damage. A characteristic AST:ALT ratio exceeding 2:1 has historically been considered a useful indicator of alcoholic liver injury, as ethanol-mediated mitochondrial dysfunction selectively elevates AST, while concurrent pyridoxal-5-phosphate depletion suppresses hepatic ALT synthesis, resulting in a pattern of disproportionate AST elevation relative to ALT [8,9].

A clinically significant and underappreciated limitation of LFTs, however, is their inability to exclude advanced liver disease. There is now substantial evidence that routine aminotransferase levels can remain entirely within normal reference limits even in patients with histologically or radiologically confirmed hepatic fibrosis or cirrhosis [10,11]. This diagnostic gap is particularly consequential in patients with AUD, in whom hepatic injury may be structural and progressive despite biochemically unremarkable enzyme levels. The failure to recognise this limitation may lead to false reassurance in the clinical setting and delayed use of more sensitive diagnostic modalities.

The present study was conducted to characterise the pattern and prevalence of LFT abnormalities in patients with AUD admitted to a tertiary care psychiatry unit in Mumbai, to examine the clinical correlates of biochemical findings, and to contribute institutional data to the growing body of evidence examining the diagnostic limitations of routine hepatic biochemistry in this population.

MATERIALS AND METHODS

Study Design and Ethical Approval: This was a retrospective chart analysis conducted at a tertiary care centre in Mumbai. Ethical clearance was obtained from the Institutional Ethics Committee prior to commencement of data extraction (IEC/94/21). Given the retrospective nature of the study and the use of de-identified archival data, individual patient consent was not required, in accordance with applicable institutional guidelines.

Study Population: Case records of all patients admitted to the psychiatry inpatient ward between January 2020 and December 2021 were systematically reviewed. Patients were eligible for inclusion if they: (i) carried a diagnosis of alcohol use disorder (AUD) established according to DSM-5 criteria; (ii) had a documented history of regular alcohol consumption for at least 12 consecutive months; and (iii) had complete LFT data available in their inpatient record.

Exclusion criteria: Patients were excluded if: (i) alcohol use disorder was not the primary or contributing admission diagnosis; (ii) LFT data were absent or incomplete; or (iii) an alternative primary hepatic diagnosis (e.g., viral hepatitis, autoimmune hepatitis) was documented that could independently account for LFT derangement.

Study variables: The following variables were systematically extracted from each eligible case record: sociodemographic data (age, sex); type and predominant form of alcohol consumed; comorbid substance use (tobacco, cannabis, others); LFT parameters including total bilirubin (mg/dL), AST/SGOT (IU/L), and ALT/SGPT (IU/L); the AST:ALT ratio; and the

presence of clinically documented signs and symptoms attributable to hepatic dysfunction (jaundice, melena, haematemesis, ascites, haemorrhoids).

Biochemical reference standards: An AST:ALT ratio > 2 was used as a supportive indicator of alcoholic aetiology of liver injury, in accordance with established biochemical criteria [8].

Statistical analysis: Descriptive statistics were used to summarise continuous variables (mean ± standard deviation [SD], range) and categorical variables (frequencies and percentages). Analysis was performed using IBM SPSS Statistics version 25.0.

RESULTS

Participant characteristics: A total of 100 male patients with AUD meeting the eligibility criteria were included in the study. The mean age was 38.35 ± 11.1 years (range: 18-61 years). All participants were male. The predominant type of alcohol consumed was country liquor (62 patients), followed by whisky (20 patients), taadi (10 patients), beer (9 patients), and other distilled spirits including rum and vodka (2 patients). Some patients reported concurrent use of more than one type of beverage; data were therefore recorded as overlapping (Table 1).

Table 1. Type of Alcohol Consumed by study participants

Alcohol Type	Number of patients (Overlapping Data)
Country Liquor	62
Whisky	20
Taadi	10
Beer	9
Others (Rum, Vodka, etc)	2

Note: Data was overlapping; as patients using more than one alcohol type

Comorbid Substance Use: Eighty patients (80%) reported concurrent use of at least one additional psychoactive substance. Nicotine use (in any form cigarettes, beedis, gutka, or smokeless tobacco) was the most common comorbidity, reported by 80 patients. Cannabis use was documented in 22 patients. Twenty patients reported no comorbid substance use. As this was an overlapping dataset, patients may be represented in more than one category (Table 2).

Table 2. Comorbid substance use by study participants

Comorbid Substance	Number of patients (Overlapping Data)
Nicotine	80
Cannabis	22
None	20

Note: Data was overlapping; as patients using more than one comorbid substance

Liver Function Test: Table 3 summarises the mean LFT values for the study population. LFT data were available for between 96 and 97 patients, depending on the parameter. The mean total bilirubin (0.97 ± 0.87 mg/dL), mean AST/SGOT (81.12 ± 86.24 IU/L), and mean ALT/SGPT (51.72 ± 51.50 IU/L) were all within or near the upper limits of normal reference ranges on a group basis; however, wide inter-individual variation was evident from the reported ranges, particularly for AST (15-562 IU/L) and ALT (11-300 IU/L).

Table 4 stratifies the proportion of patients exceeding the upper limit of the reference range versus those meeting the threshold for clinically significant elevation. While 55% of patients had AST values above the upper reference limit, only 7% had values meeting the threshold for clinical significance. Similarly, 36% had ALT values exceeding the reference range, but clinically significant ALT elevation was present in only 5% of patients. For bilirubin, 32 patients exceeded the upper limit of 1.0 mg/dL, but only 3 patients surpassed the clinically significant threshold of 2.7 mg/dL.

Importantly, an AST:ALT ratio > 2 the biochemical hallmark of alcoholic liver injury was present in only 19 of 100

patients (19%). Six patients (6%) presented with clinical signs attributable to hepatic decompensation: 3 with melena, 2 with haematemesis, and 1 patient each with jaundice, ascites, and haemorrhoids (Table 5).

Table 3. Liver Function Tests of study participants

Test (n)	Mean ± S.D.	Min. – Max.	Reference value
Total Bilirubin (97) {mg/dl}	0.97 ± 0.87	0.1 - 6.8	0-1mg/dl
SGOT/AST (96)* {IU/L}	81.12 ± 86.24	15 - 562	0-40 IU/L
SGPT/ALT (96)# {IU/L}	51.72 ± 51.50	11 -300	0-40 IU/L
SGOT:SGPT ratio (96)	1.69 ± 0.97	0.29- 6.45	-

*Serum Glutamic-Oxaloacetic Transaminase/AST: Aspartate aminotransferase, #Serum Glutamic-Pyruvic Transaminase/ALT: Alanine aminotransferase

Table 4. Proportion of patients exceeding reference and clinically significant thresholds for LFT parameters

Parameter	Normal Reference Range	Patients having above upper limits, n (%)	Clinically significant threshold	Patients with clinically significant values n (%)
Total Bilirubin	0-1 mg/dL	32 (32)	>2.7mg/dl	3 (3)
SGOT/AST	0-40 IU/L	55 (55)	>120 IU/L	7 (7)
SGPT/ALT	0-40 IU/L	36 (36)	>120 IU/L	5 (5)

Note: Clinically significant thresholds are defined as three times the upper limit of the reference range for aminotransferases, and > 2.7 mg/dL for total bilirubin, in accordance with standard hepatological practice [8]

Table 5. Clinical Signs and Symptoms of Hepatic Decompensation in Study Participants (n = 100)

Clinical manifestation	N
Melena	3
Haematemesis	2
Jaundice	1
Ascites	1
Haemorrhoids	1
Total with any clinical sing of hepatic dysfunction	6 (6%)

Note: Individual patients may have presented with more than one clinical manifestation.

DISCUSSION

The principal finding of this study is that the large majority of patients with AUD admitted to a tertiary care psychiatry unit had normal or only mildly abnormal LFT values, despite a documented history of prolonged regular alcohol consumption. Clinically significant LFT elevations were observed in fewer than 10% of patients for any individual parameter, and clinical signs of hepatic decompensation were present in only 6% of the cohort. These findings are consistent with, and add to, the growing body of evidence demonstrating that standard biochemical hepatic markers substantially underestimate the prevalence and severity of alcohol-related liver injury in patients with AUD.

Liver function tests have long served as the cornerstone of initial hepatic evaluation, and their role in identifying hepatocellular injury and monitoring disease progression is well established [7]. Ali et al. described the importance of interleukin-based and conventional biochemical markers in both diagnosing and longitudinally monitoring liver disease, highlighting the central role of AST, ALT, and bilirubin in clinical hepatology [12]. Shahid et al., examining the relationship between ultrasound grading of fatty liver disease and LFT values, found significant correlations between sonographic and biochemical measures of hepatic injury a finding that, while supporting LFT utility in some clinical contexts, does not exclude their limitations in advanced or fibrotic disease [13].

The pattern of aminotransferase elevation observed in this cohort is broadly consistent with the known biochemical signature of ARLD. The mean AST (81.12 IU/L) was disproportionately elevated relative to mean ALT (51.72 IU/L),

yielding a mean AST:ALT ratio above 1.5. However, an AST:ALT ratio exceeding the diagnostic threshold of 2:1 was observed in only 19% of patients a finding that corroborates data from Nyblom et al., who similarly reported AST elevation in 55% and ALT elevation in 36% of patients with chronic alcohol use, with the characteristic ratio present in a minority of cases [8]. This variability reflects the mechanistic complexity of hepatic enzyme release in ARLD: while ethanol-mediated mitochondrial injury promotes disproportionate release of mitochondrial AST, this pattern is not consistently expressed across individuals, and depletion of pyridoxal-5-phosphate the co-factor for ALT synthesis does not reduce ALT with equal magnitude in all patients [8,9]. Consequently, the AST:ALT ratio, while a useful corroborative marker, cannot be relied upon as a definitive diagnostic criterion for ARLD.

The observation that the majority of patients in this cohort had LFT values within or only marginally above the normal reference range is clinically important and challenges assumptions that underpin routine screening practice. Sullivan et al., in a retrospective cohort analysis of 78 patients with established alcoholic cirrhosis, demonstrated that approximately 90% of patients had normal ALT values and 15% had normal AST values despite histologically or radiologically confirmed advanced liver disease [14]. Critically, ALT levels showed no correlation with disease severity, complication rates, or mortality in that cohort findings that directly support the conclusion that aminotransferases cannot be used in isolation to assess the structural burden of ARLD[14]. Our findings from a psychiatric inpatient population in Mumbai reinforce these observations in a distinct clinical setting.

Further corroborating evidence comes from a recent large-scale Korean nationwide cohort study by Oh et al. involving more than 19,000 participants, which demonstrated that heavy alcohol consumption significantly increased the risk of liver disease including both alcoholic and non-alcoholic liver disease even among individuals who consistently maintained normal AST and ALT levels across repeated health assessments [15]. This landmark study fundamentally challenges the notion that normal liver enzyme values in heavy drinkers can be used as a reliable indicator of hepatic safety, and supports the need for risk stratification and supplementary investigation in this population.

Several mechanistic pathways explain the paradox of structural hepatic damage in the presence of normal biochemistry. First, aminotransferases primarily reflect active hepatocellular necrosis rather than the extent of fibrosis or architectural distortion [19]. In advanced fibrosis or cirrhosis, the progressive loss of functioning hepatocyte mass reduces the absolute quantity of enzyme available for release into the circulation. As hepatocyte numbers decline, enzyme release decreases a phenomenon that can result in paradoxically normal or declining aminotransferase levels even as the underlying disease advances [16,17]. Second, the liver possesses a substantial regenerative capacity that sustains metabolic function and biochemical normalcy until a critical proportion of parenchymal tissue has been compromised. Third, the gut–liver axis may modulate hepatic inflammation independently of aminotransferase-reflected injury: disruption of intestinal microbiota composition and increased intestinal permeability promote endotoxaemia and hepatic Kupffer cell activation, driving fibrogenesis without necessarily producing commensurate hepatocellular necrosis detectable by standard serum markers [18].

An additional dimension of clinical complexity in patients with AUD is the presence of comorbid conditions that may independently influence LFT interpretation. In this cohort, 80% of patients reported concurrent nicotine use and 22% reported cannabis use both of which have been associated with hepatic metabolic effects that may interact with alcohol-related injury pathways. Furthermore, the emerging literature on COVID-19-related hepatic involvement including elevated transaminases in patients with pre-existing hepatic conditions such as non-alcoholic fatty liver disease serves as a reminder that LFT values in complex, multi-morbid patients must always be interpreted within a full clinical and epidemiological context [19,20].

Dascal et al., in a Canadian cohort study examining LFT profiles in patients with chronic liver disease presenting with unconjugated hyperbilirubinaemia, demonstrated that patients with underlying chronic hepatic conditions including ARLD could present with largely unremarkable routine LFT profiles, with unconjugated hyperbilirubinaemia in the absence of elevated albumin or INR not necessarily signalling advanced fibrosis or necroinflammatory activity [21]. This further illustrates the heterogeneity of biochemical presentations in ALD and the inadequacy of any single LFT parameter as a reliable disease marker.

The present findings carry direct and actionable clinical implications. In routine practice, a normal LFT result in a patient with AUD may engender inappropriate reassurance and delay further hepatic evaluation. The available evidence including the data from this study strongly supports the integration of non-invasive hepatic fibrosis assessment tools as part of the standard evaluation of patients with AUD. Transient elastography (FibroScan) measures liver stiffness and provides validated, quantitative assessment of hepatic fibrosis with good sensitivity and specificity, making it particularly useful for screening high-risk populations such as chronic alcohol users [22,23]. Serum-based fibrosis indices the Fibrosis-4 (FIB-4) index [24], the AST-to-platelet ratio index (APRI) [25], and the enhanced liver fibrosis (ELF) panel [22] provide additional non-invasive tools for stratifying fibrosis risk in patients in whom imaging or elastography is not immediately available. Abdominal ultrasonography and, where indicated, cross-sectional imaging with computed tomography or magnetic resonance elastography can further characterise structural hepatic changes including steatosis, nodularity, splenomegaly, and portal hypertension.

The EASL Clinical Practice Guidelines on alcohol-related liver disease explicitly recommend a comprehensive approach to hepatic assessment in patients with AUD that incorporates clinical, biochemical, and imaging evaluation recognising that no single parameter is sufficient for reliable diagnosis or staging [22]. The findings of the present study argue for closer alignment between these guideline recommendations and clinical practice in the psychiatric inpatient setting, where patients with AUD may not routinely receive hepatological evaluation beyond standard LFTs.

CONCLUSION

This retrospective study demonstrates that the majority of patients admitted with alcohol use disorder to a tertiary care psychiatry unit in Mumbai exhibit normal or only mildly elevated liver enzyme levels despite prolonged alcohol use. Clinically significant biochemical derangement and clinical signs of hepatic decompensation were each present in fewer than 10% of the cohort. The AST:ALT ratio exceeded the diagnostic threshold in only 19% of patients. These findings confirm that standard liver function tests are insufficient as stand-alone tools for detecting or excluding alcohol-related liver disease. Clinicians evaluating patients with AUD should maintain a high degree of clinical suspicion irrespective of LFT results and should consider integrating non-invasive fibrosis assessment strategies including transient elastography, FIB-4, and APRI into routine clinical practice to improve early detection of hepatic injury and reduce the risk of delayed diagnosis in this vulnerable population.

Limitations: The findings of the present study must also be interpreted in light of certain limitations. The study utilized retrospective chart analysis and was limited to hospitalized patients with alcohol use disorder. Therefore, the results may not be fully generalizable to community populations or individuals with less severe alcohol use. Additionally, imaging studies and fibrosis markers were not systematically evaluated in all patients, which may have limited the ability to detect subclinical liver disease.

Despite these limitations, the study provides valuable insights into the limitations of liver function tests as screening tools for alcohol-related liver disease. The findings highlight the need for clinicians to adopt a more comprehensive approach when evaluating patients with chronic alcohol use.

Future research should focus on identifying more sensitive biomarkers for early detection of alcohol-related liver injury. Combining biochemical markers with imaging techniques and clinical risk assessment models may improve the accuracy of screening strategies and facilitate earlier intervention.

Declarations

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