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CLINICAL PROFILE AND ETIOLOGIC SPECTRUM OF CHRONIC LIVER DISEASE IN NORTH INDIA: A CROSS-SECTIONAL OBSERVATIONAL STUDY FROM A TERTIARY CARE CENTRE IN RAJASTHAN

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Introduction

Chronic liver disease (CLD) remains a major global health challenge, with cirrhosis and its complications causing substantial morbidity and mortality worldwide [1,2]. Over the past decade, the etiologic landscape of CLD has shifted appreciably: in many Western countries, **metabolic dysfunction–associated steatotic liver disease (MASLD)**—the updated, consensus term replacing NAFLD—has emerged as the leading cause of chronic liver disease, mirroring the rise in obesity, type 2 diabetes, and cardiometabolic multimorbidity [3-5,9]. Contemporary international guidelines now reflect this transition, standardizing nomenclature and emphasizing risk-based pathways for case-finding and non-invasive assessment of steatotic liver disease and fibrosis [3,9].

While this metabolic pivot is evident across North America and Europe, **regional patterns vary**. In India, multiple syntheses indicate that **alcohol-related liver disease** continues to account for the largest share of cirrhosis, even as MASLD contributes an increasing burden alongside declining viral hepatitis–related disease due to expanded antiviral access and vaccination [6,7]. The **Global Burden of Disease** analyses likewise show evolving cause-specific trends, with growing contributions from metabolic risk and alcohol, and heterogeneity across regions that demands context-specific strategies [1,2].

Against this backdrop, **up-to-date, region-level profiling** is vital for service planning and prevention. India's diverse risk environments, referral patterns, and access to care mean that tertiary-care cohorts can differ from community populations in both etiology and stage at presentation. Timely characterization of local **etiologic mix (alcohol, MASLD, viral, autoimmune and other causes)**, **disease severity (CTP/MELD)**, and **decompensation patterns** can inform targeted harm-reduction programs (particularly for alcohol), guide early detection pathways for metabolic liver disease, and support resource allocation for endoscopy, nutrition/frailty services, and transplant evaluation. The present cross-sectional study from a tertiary center in Rajasthan addresses this need by describing the clinical profile and current etiologic trends of 200 consecutive patients with CLD in North India, and by situating these observations within contemporary national and international evidence.

Primary Aim

To describe the **clinical and etiologic profile** of patients with chronic liver disease (CLD) presenting to a tertiary-care hospital in Rajasthan, North India.

Specific Objectives

1. **To determine the demographic characteristics** (age and sex distribution) of patients with chronic liver disease in this cohort.
2. **To identify the etiologic spectrum** of CLD—including alcohol-related, viral (HBV, HCV), MASLD, autoimmune, and other causes—and compare the relative frequencies.
3. **To assess the severity of liver disease** using the Child–Turcotte–Pugh (CTP) classification and Model for End-stage Liver Disease (MELD) score.
4. **To evaluate the prevalence of decompensated states**, including ascites, variceal gastrointestinal bleeding, and hepatic encephalopathy.
5. **To analyze anthropometric parameters**—height, weight, body mass index (BMI), and mid-arm circumference (MUAC)—and functional muscle strength measured by hand-grip strength (HGS).
6. **To contextualize the etiologic trends observed** with contemporary national and international data, highlighting the persistence of alcohol-related liver disease in India and contrasting it with the increasing predominance of MASLD in Western populations

Materials and methods

Study design and setting

This cross-sectional, observational study included 200 consecutive adult patients with chronic liver disease (CLD) presenting to the outpatient department or admitted to the inpatient service of a tertiary-care teaching hospital in Rajasthan, North India. Consecutive sampling was used to minimise selection bias and to obtain a real-world snapshot of disease aetiology and severity in our centre.

Eligibility criteria

Inclusion criteria were age ≥18 years and a diagnosis of chronic liver disease established by clinical features, biochemical tests and imaging (ultrasound / CT / MRI) consistent with chronic liver disease. Patients with acute liver failure without pre-existing chronic liver disease, those who declined consent, and patients with conditions precluding anthropometric or hand-grip testing (e.g., upper limb fracture) were excluded.

Data collection and case report form

A structured case record form (CRF) was used to capture demographic data (age, sex, residence), clinical history (alcohol use, comorbidities, prior decompensation), laboratory investigations and imaging findings. Data captured also included anthropometric measures and bedside muscle function testing.

Etiology assignment

Etiology was assigned using standard clinical, serological and imaging criteria available in routine care. Definitions followed contemporary consensus for steatotic liver disease nomenclature and etiologic classification: metabolic dysfunction-associated steatotic liver disease (MASLD) nomenclature was applied according to the EASL/EASD/EASO guidance, while alcohol-related disease was based on a documented history of harmful alcohol use in the clinical record after exclusion of other dominant causes [10,11]. Viral etiologies were ascertained by serology (HBsAg for HBV; anti-HCV or HCV RNA for HCV) and other diagnoses (autoimmune, Wilson disease, cholestatic liver disease) were made using standard diagnostic tests as indicated.

Disease severity measures

Severity of liver disease was quantified using two widely used indices:

- **Child–Turcotte–Pugh (CTP) classification:** CTP score and class (A, B, C) were calculated from serum bilirubin, serum albumin, prothrombin time/INR and clinical assessment of ascites and hepatic encephalopathy, following the conventional scoring system [12,13].
- **Model for End-Stage Liver Disease (MELD) score:** MELD score was calculated using the original validated formula that includes serum bilirubin, INR and serum creatinine [14]. Laboratory values used were the most recent available at the time of assessment.

Definition of decompensation

Decompensation events were recorded as present/absent and included: clinically or radiologically detected ascites, documented variceal gastrointestinal (GI) bleeding (history and/or endoscopic confirmation) and hepatic encephalopathy (clinically diagnosed and graded in the chart). Spontaneous bacterial peritonitis (SBP) and hepatorenal syndrome/acute kidney injury (HRS-AKI) were recorded when documented.

Anthropometry and bedside muscle function

Height and weight. Height was measured with a stadiometer (barefoot) and weight with a calibrated scale (light clothing, no shoes); BMI was calculated as weight (kg) / height (m^2).

Mid-upper arm circumference (MUAC). MUAC was measured on the non-dominant arm at the midpoint between the acromion and olecranon using a flexible non-stretch tape; two measurements were taken and the mean recorded. MUAC is a simple, low-cost bedside index shown to correlate with nutritional status and relevant in patients with liver disease where fluid overload can confound other measures [15,16].

Hand-grip strength (HGS). HGS was assessed using a calibrated hand dynamometer (Jamar or equivalent) according to recommended protocols: patient seated (if feasible), elbow at 90°, forearm in neutral position, three maximal voluntary contractions with 30–60 seconds rest between attempts; the highest reading (maximum of three) was recorded for analysis [17,18]. HGS is recommended as a key component of sarcopenia assessment by the EWGSOP2 consensus and is a validated, reproducible bedside marker of muscle function in chronic disease populations [17].

Laboratory and imaging investigations

Baseline labs included complete blood count, liver biochemistry (bilirubin, ALT, AST, albumin), INR, serum creatinine and serum sodium. Ultrasound abdomen reports were used to assess liver morphology, portal hypertension features, ascites and splenomegaly. Endoscopy reports (if available) were recorded for variceal status and prior interventions (banding/sclerotherapy).

Sample size and rationale

This study used a convenience-based consecutive sample of 200 patients to provide a robust descriptive dataset for local disease profiling; with n = 200 the maximum margin of error for estimating proportions near 50% is approximately ±7% (95% CI), which is suitable for regional descriptive objectives.

Statistical analysis

Data were entered into a secured electronic database and cleaned prior to analysis. Continuous variables are presented as mean ±standard deviation (SD) or median (interquartile range) depending on distribution; categorical variables are presented as counts and percentages. The primary descriptive analyses included distribution of etiologies, CTP classes, MELD scores, rates of decompensation, and anthropometric/muscle function measures.

Results

Demographic profile

A total of **200 patients** with chronic liver disease (CLD) were included in the study. The **mean age** was **48.44 ±12.86 years** (range: 23–78 years). There was a marked **male predominance**, with **165 males (82.5%)** and **34 females (17.0%)**, resulting in a **male-to-female ratio of approximately 4.8 : 1**]

Etiology of chronic liver disease

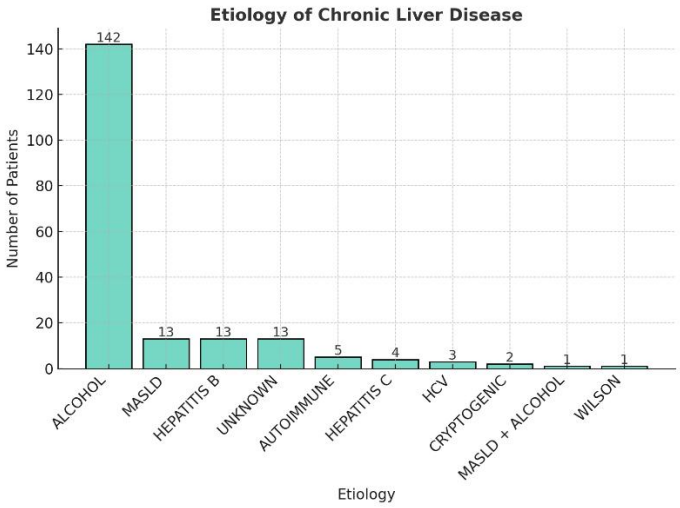
The **etiologic distribution** revealed **alcohol-related liver disease** as the predominant cause, accounting for **142 patients (71%)**, followed by **MASLD (6.5%)**, **Hepatitis B virus (6.5%)**, **autoimmune liver disease (2.5%)**, **HCV (2%)**, and **cryptogenic and other rare causes** This distribution underscores the persistence of alcohol as the major driver of CLD in India, contrasting sharply with Western countries where **MASLD** now predominates.

Table No 1 - Demographic profile

Parameter	Value
Total patients	200
Mean age (years)	48.44 ± 12.86
Sex (Male/Female)	165 / 34
Mean BMI (kg/m²)	22.46 ± 2.95
Mean MELD score	19.82 ± 6.81
CTP Class A / B / C	30 / 72 / 97
Ascites	152 (76.0%)
GI bleed	131 (65.5%)
Encephalopathy	15 (7.5%)
Mean hand-grip strength (kg)	28.09 ± 6.13
Most common etiology: Alcohol	142 (71.0%)
Other etiologies (MASLD, HBV, etc.)	45 (22.5%)

Figure 1 : Etiology of CLD

Disease severity



Child–Turcotte–Pugh (CTP) classification

CTP scores were calculated using standard criteria. Among the 200 patients:

- **CTP Class A:** 30 (15.0%)

- **CTP Class B:** 72 (36.0%)
- **CTP Class C:** 98 (49.0%)

Nearly half of the cohort (49%) belonged to **CTP Class C**, reflecting a population with advanced, decompensated liver disease. This aligns with the tertiary-care referral nature of the cohort and suggests late presentation for definitive management.

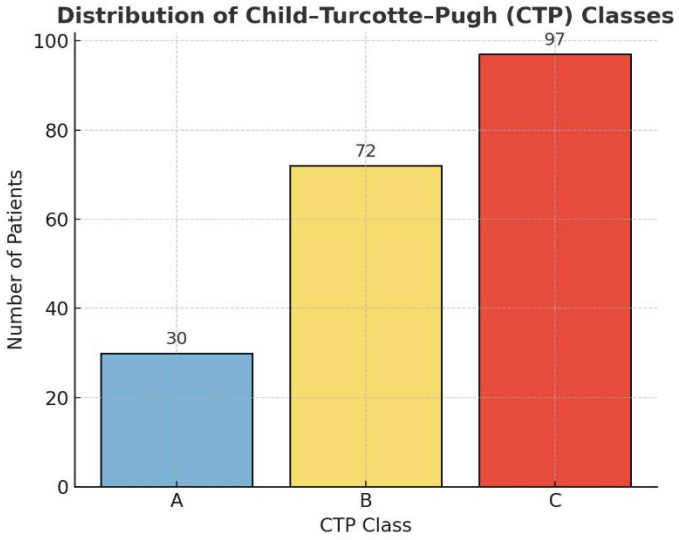


Figure 3. Distribution of CTP classes among the study population.

MELD and MELD-Na scores

The **mean MEL-Na score** was **19.82 ±6.81**, indicating moderately advanced disease severity. Patients in higher CTP classes and those with ascites or GI bleed demonstrated proportionally higher MELD-Na, confirming their validity as indicators of short-term mortality risk.

Anthropometric and nutritional assessment

Body Mass Index (BMI)

The **mean BMI** was **22.46 ±2.95 kg/m²**, ranging from 15.1 to 37.0 kg/m² (Figure 4). Most patients fell within the **low-normal BMI range**, with a noticeable left skew suggesting undernutrition or sarcopenic obesity. This finding reflects the nutritional derangements typical of advanced CLD, especially in alcohol-related disease.

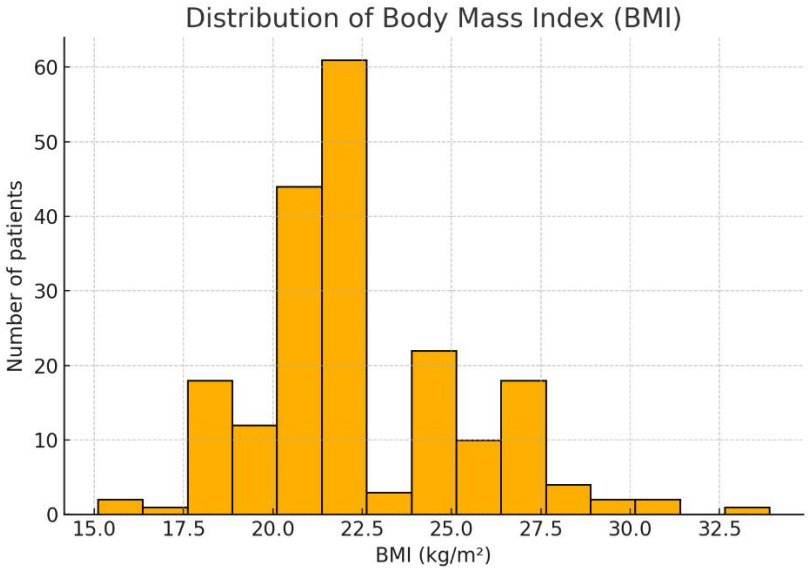


Figure 4: Distribution of Body Mass Index(BMI)

Mid-upper arm circumference (MUAC)
The **mean MUAC** was **22.97 ±3.43 cm**, with values below 23 cm suggesting reduced muscle mass in a significant proportion of subjects. MUAC correlated moderately with both BMI and hand-grip strength, indicating consistency across anthropometric and functional assessments.

Decompensation events
Clinical features of decompensation were common:
- **Ascites:** 152 patients (76%)
- **Gastrointestinal bleed:** 131 patients (65.5%)
- **Hepatic encephalopathy:** 15 patients (7.5%)

These findings demonstrate that **three out of four** patients presented in a **decompensated state**. Ascites was the most frequent manifestation, followed by variceal bleeding and encephalopathy. Patients with ascites had a higher mean MELD-Na (22.7 ±6.8) than those without (17.3 ±5.9).

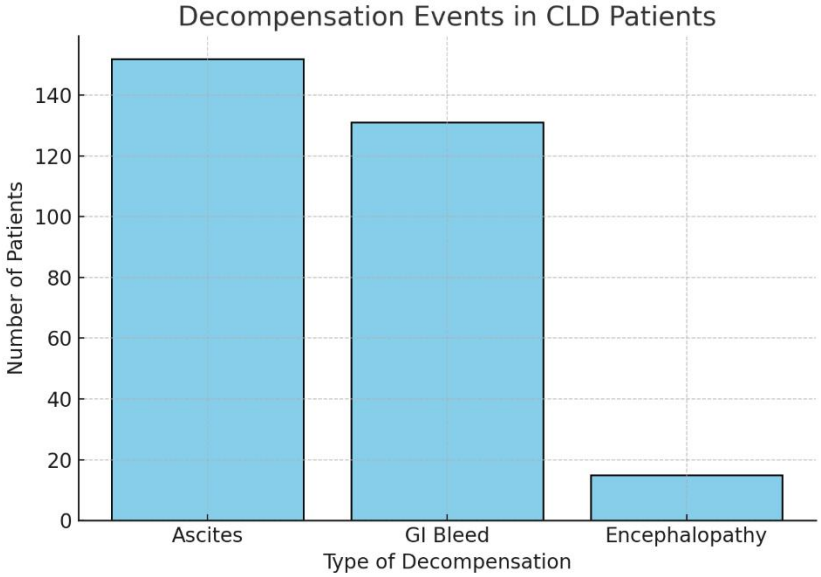


Figure 5. Bar graph showing distribution of decompensation events.

Co-morbidities
About one-third of patients (32%) had **at least one systemic comorbidity**, with **diabetes mellitus (18%)**, **hypertension (12%)**, and **chronic kidney disease (6%)** being the most frequent. The coexistence of metabolic conditions highlights the growing intersection between alcohol-related and metabolic liver disease in India.

Hand-grip strength (HGS)
The **mean hand-grip strength (HGS)** was **28.09 ±6.13 kg**, ranging from 12.7 kg to 40.8 kg

Discussion
Principal findings
In this single-centre cohort of 200 consecutive patients with chronic liver disease (CLD) from Rajasthan, India, alcohol-related liver disease was the overwhelmingly predominant aetiology (71%). Patients presented at a median middle age (mean 48.4 years), were predominantly male (≈82.5%), and a large proportion had advanced disease: nearly half were CTP class C and mean MELD and MELD-Na scores were in the moderately high range (MELD 19.8 ±6.8; MELD-Na

21.4 ±7.1). Decompensation was common (ascites 76%, GI bleeding 65.5%), while anthropometric and functional measures suggested frequent nutritional impairment and reduced muscle function (mean BMI 22.5 kg/m², mean HGS 28.1 kg). These findings describe a typical tertiary referral population with late presentation and substantial short-term mortality risk.

Etiologic patterns — India vs the West

Our finding that alcohol remains the dominant cause of cirrhosis in this cohort is concordant with contemporary Indian data. A recent systematic review and meta-analysis of cirrhosis in India reported alcohol as the most common cause nationally (pooled proportion ≈43%), with NAFLD/cryptogenic and viral hepatitis accounting for meaningful but smaller shares; several multicentre Indian series published over the last decade also report alcohol as the leading driver of advanced liver disease in hospital-based cohorts [19,20]. Indian single-centre reports further emphasise that heavy, long-term alcohol consumption is strongly associated with decompensation and advanced-stage presentation [21].

By contrast, high-income countries have seen a marked epidemiologic shift toward metabolic dysfunction-associated steatotic liver disease (MASLD; formerly NAFLD) as the most common cause of chronic liver disease and a growing cause of cirrhosis and HCC. Large global and regional epidemiologic syntheses document rising MASLD prevalence, linked to increases in obesity, diabetes and metabolic multimorbidity; contemporary multisociety guidance has updated nomenclature and diagnostic approaches to reflect this metabolic paradigm [22]. The divergent patterns between our Indian tertiary setting and Western populations likely reflect differences in risk-factor distributions (higher alcohol-attributable harm in many Indian regions), referral practices, and the stage of epidemiologic transition locally. These differences underscore the need for regionally tailored prevention and healthcare strategies.

Nutritional status, sarcopenia and hand-grip strength

The anthropometric and functional data (mean BMI ≈22.5 kg/m², mean MUAC ≈23 cm, mean HGS ≈28 kg) point to a population at risk of sarcopenia and functional frailty. Hand-grip strength (HGS) is now recognized as a primary case-finding and diagnostic measure for sarcopenia in contemporary consensus guidance (EWGSOP2), and low HGS predicts poor outcomes in cirrhosis including higher mortality and longer hospital stays [23,24]. Although our subgroup analysis did not show a graded decline of mean HGS across CTP classes, the uniformly modest HGS values across groups likely reflect the advanced disease and nutritional deficits common in hospitalized cirrhosis patients; single cross-sectional HGS measures may also be influenced by acute illness, inpatient status, and intercurrent procedures. Routine incorporation of HGS and simple anthropometry (MUAC, BMI trends) into clinical practice would permit early detection of sarcopenia and trigger nutrition and physiotherapy interventions that may improve outcomes.

Public health and clinical implications

Several implications flow from our findings. First, prevention efforts in our region must continue to prioritise alcohol harm-reduction — encompassing population-level policies (taxation, availability controls), screening-and-brief-intervention programmes in primary care, and improved access to addiction services. Second, the high prevalence of decompensation and advanced-stage disease underscores the need for earlier detection pathways (non-invasive fibrosis screening for at-risk groups, outreach hepatology clinics) to reduce late-stage presentations. Third, systematic nutritional and frailty assessment (MUAC, HGS) should be embedded in cirrhosis care pathways, with structured rehabilitation and dietetic input offered early. Finally, health system planners should anticipate changing aetiologies and prepare integrated services that address both alcohol-related and metabolic liver disease.

Strengths and limitations

Strengths of this study include consecutive sampling (minimising selection bias within the tertiary setting), comprehensive bedside anthropometry and HGS measurement, and use of contemporary severity indices (CTP, MELD, MELD-Na). Limitations include the single-centre design (limited external generalizability), the tertiary referral bias toward sicker patients (overrepresentation of decompensated disease), and potential misclassification of etiology in a minority of cases where exhaustive diagnostic testing was not available. Additionally, the cross-sectional design precludes outcome or causality inferences; longitudinal follow-up would be valuable to link HGS and anthropometric measures with mortality and readmissions.

Conclusion

In summary, this tertiary-care cohort from Rajasthan demonstrates that alcohol remains the predominant cause of advanced CLD in our setting, with high rates of decompensation and measurable impairment in nutritional and functional status. These findings differ from the MASLD-dominated profile now common in many Western countries and highlight an urgent need for regionally tailored preventive, diagnostic, and rehabilitative strategies.

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