ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 05 | May - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

PROSPECTIVE CLINICAL STUDY OF ALCOHOLIC LIVER DISEASE AND ITS COMPLICATIONS IN THE TRIBAL BELT OF GUJARAT: A HOSPITAL-BASED OBSERVATIONAL STUDY

Internal Medicine

Dr. Smit Gajjar — 3rd Year Post-Graduate Resident, Department of General Medicine,Zydus Medical College and Hospital, Dahod, Gujarat, India

Dr. Smit Buddhadev* — 3rd Year Post-Graduate Resident, Department of General Medicine, Zydus Medical College and Hospital, Dahod, Gujarat, India*Corresponding Author

Dr. Ravindra Hadakshi — Professor, Department of General Medicine, Zydus Medical College and Hospital, Dahod, Gujarat, India.

ABSTRACT

Background: Alcoholic liver disease (ALD) is a leading cause of chronic liver morbidity in India, particularly in tribal populations with limited healthcare access. Data from rural tribal settings in Gujarat remain scarce.**Objective:** To evaluate the clinical characteristics, disease severity, complications, and short-term outcomes in ALD patients attending a tertiary care hospital in the tribal belt of Gujarat.**Methods:** This prospective observational study enrolled 100 patients diagnosed with ALD at Zydus Medical College and Hospital, Dahod, from June 2023 to November 2024. Demographic data, clinical findings, biochemical parameters, imaging results, and prognostic scores (Child–Pugh and MELD 3.0) were systematically recorded. Statistical analysis was performed using SPSS v25.**Results:** The majority were male (92%), middle-aged, and employed as labourers or farmers. Country liquor was consumed by 64%. Child–Pugh Class B and C were present in 70%. Ascites was found in 72%, esophageal varices in 77%, hepatic encephalopathy in 60%, and variceal bleeding in 20%. In-hospital mortality was 34%. At 28-day follow-up, 93% were alive.**Conclusions:** ALD in Gujarat's tribal belt presents at advanced stages with high complication rates and significant in-hospital mortality. Community screening, alcohol cessation programmes, and strengthening rural healthcare are urgently needed.

KEYWORDS

Alcoholic Liver Disease, Tribal Gujarat, Child–pugh Score, Meld 3.0, Portal Hypertension, Hepatic Encephalopathy, India

INTRODUCTION

Alcohol-related liver disease (ALD) spans a pathological continuum from simple hepatic steatosis through alcoholic hepatitis, progressive fibrosis, cirrhosis, and end-stage complications. Its global burden has grown substantially, and in countries like India, alcohol now stands as the leading etiological factor in chronic liver disease presentations at tertiary centres [1]. Epidemiological projections underscore the urgency: metabolic dysfunction-associated steatotic liver disease is expected to affect close to 800 million individuals globally by 2045, and when alcohol intake co-exists with obesity or type 2 diabetes, the additive hepatotoxic effect substantially accelerates progression to cirrhosis [2].

In India, a twelve-year observational study encompassing over sixteen thousand gastrointestinal ward admissions found that 63.3% of cirrhosis cases were attributable to alcohol [3]. This burden is amplified in low-resource tribal communities where social drinking is embedded in culture, locally brewed spirits carry variable and often high ethanol concentrations, and access to diagnostic facilities is limited [4].

The tribal districts of eastern Gujarat exemplify this challenge. These populations face a convergence of risk: traditional communal drinking customs, labour-based alcohol payment schemes, protein-calorie malnutrition reducing hepatic resilience, and geographical isolation delaying presentation until advanced disease stages [5]. Biochemical evidence from Indian ALD cohorts demonstrates that CD14 gene polymorphisms influence endotoxemia susceptibility, suggesting genetic predisposition adds a further layer of risk [6]. Despite this, region-specific prospective data remain scarce.

Complications of ALD — portal hypertension, variceal bleeding, hepatic encephalopathy, spontaneous bacterial peritonitis (SBP), and hepatorenal syndrome — are the primary drivers of mortality. Nationally, portal vein thrombosis in cirrhotic patients warrants dedicated risk analysis [3], and acute-on-chronic liver failure from overlapping injury mechanisms accounts for a measurable proportion of ICU admissions [7].

This study was designed to prospectively characterize the clinical profile, severity, complications, and short-term outcomes of patients presenting with ALD to a tertiary teaching hospital serving the tribal belt of Dahod, Gujarat.

MATERIALS AND METHODS

Study Design and Setting

This was a prospective observational study conducted in the Department of General Medicine at Zydus Medical College and Hospital, Dahod between June 2023 and November 2024.

Study Population and Sample Size

One hundred consecutive patients meeting inclusion criteria were enrolled from the OPD, IPD, and Emergency Department. Sample size was calculated using the formula $n = Z^2 \times p \times (1–p) / d^2$, with $Z = 1.96$, $p = 0.07$, and $d = 0.05$.

Inclusion and Exclusion Criteria

Patients were included if they had a documented history of chronic alcohol consumption with clinical, biochemical, and radiological evidence of liver disease.Patients seropositive for HBV, HCV, or HIV; those with other causes of chronic liver disease (Wilson's disease, haemochromatosis, autoimmune hepatitis); or those receiving hepatotoxic medications were excluded.

Data Collection and Severity Assessment

After written informed consent, standardized case record forms captured demographic, clinical, laboratory (bilirubin, AST, ALT, ALP, albumin, PT/INR, renal profile), radiological (abdominal ultrasonography), and endoscopic (variceal grading) data. Severity was graded by the Child–Pugh scoring system and MELD 3.0 score.

Statistical Analysis

Data were analyzed using SPSS v25. Descriptive statistics were computed for all variables. The chi-square goodness-of-fit test was applied for categorical associations; $p < 0.05$ was considered significant.

RESULTS

Demographic and Drinking Profile

A total of 100 patients were enrolled. The cohort was predominantly male (92%; $p < 0.001$). The peak age group was ≥60 years (30%), followed by 40–49 years (23%). Labourers (39%) and farmers (25%) were the dominant occupational groups, reflecting the rural manual-labour economy of the region. Country liquor was the most frequently consumed beverage (64%), and 62% of patients had been drinking for more than ten years.

Table 1. Demographic and Alcohol Use Profile (n = 100)

Characteristic	Category	n	%	p-value
Sex				<0.001
	Male	92	92.0	

	Female	8	8.0	
Age Group				0.054
	<30 years	12	12.0	
	30–39 years	17	17.0	
	40–49 years	23	23.0	
	50–59 years	18	18.0	
	≥60 years	30	30.0	
Occupation				<0.001
	Labourers	39	39.0	
	Farmers	25	25.0	
	Unemployed/Homemakers	21	21.0	
	Self-employed	10	10.0	
	Service	5	5.0	
Alcohol Type				<0.001
	Country liquor	64	64.0	
	IMFL	18	18.0	
	Mixed	11	11.0	
	Beer	7	7.0	
Duration of Use				<0.001
	<5 years	8	8.0	
	5–9 years	18	18.0	
	10–14 years	32	32.0	
	15–19 years	12	12.0	
	≥20 years	30	30.0	

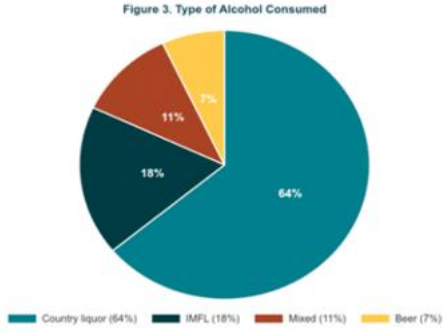

Figure 3. Type of Alcohol Consumed

Disease Severity and Biochemical Profile

Severe disease was recorded in 40% of patients, moderate in 34%, and mild in 26%. Child–Pugh Class B was most common (36%), with Class C in 34% — giving 70% in advanced classes. MELD 3.0 scores ranged predominantly between 20–29 (39%), with 18% scoring ≥30. Serum bilirubin exceeded 5 mg/dL in 65% of patients, and serum albumin was below 3.0 g/dL in 50%, indicative of substantial synthetic failure.

Table 2. Disease Severity and Biochemical Parameters (n = 100)

Parameter	Category	n	%	p-value
Severity Grade				**0.228**
	Mild	26	26.0	
	Moderate	34	34.0	
	Severe	40	40.0	
Child–Pugh Class				**0.756**
	A	30	30.0	
	B	36	36.0	
	C	34	34.0	
MELD 3.0 Score				**0.0002**
	<10	11	11.0	
	10–19	32	32.0	
	20–29	39	39.0	
	≥30	18	18.0	
Serum Bilirubin				**0.017**
	<2 mg/dL	14	14.0	
	2–5 mg/dL	21	21.0	
	5–10 mg/dL	34	34.0	
	>10 mg/dL	31	31.0	
Serum Albumin				**0.350**
	≥3.5 g/dL	30	30.0	
	3.0–3.49 g/dL	20	20.0	
	2.5–2.99 g/dL	29	29.0	
	<2.5 g/dL	21	21.0	

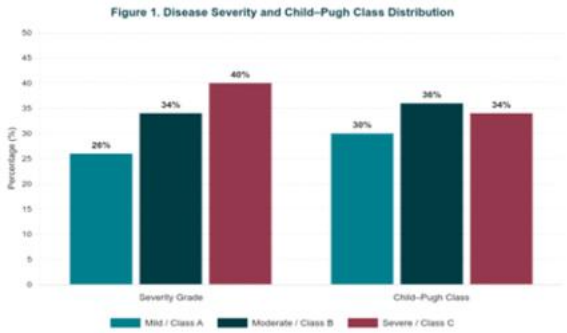

Figure 1. Disease Severity and Child–Pugh Class Distribution

Complications

Ascites was present in 72% of patients, with moderate grade being most common (32%). Ultrasound revealed splenomegaly in 45% and collateral veins in 22%. Endoscopy showed oesophageal varices in 77%, with 44% at Grade III–IV. Hepatic encephalopathy was documented in 60%, predominantly West Haven Grade I. Variceal bleeding occurred in 20%. Hepatorenal syndrome was identified in 9% and SBP in 14%. Wernicke's encephalopathy was present in 8% of patients. Infections were documented in 59% of cases, with pneumonia being the most common (31%).

Table 3. Portal Hypertension and Ascites (n = 100)

Finding	Category	n	%	p-value
Ascites Grade				**0.024**
	No ascites	28	28.0	
	Mild	28	28.0	
	Moderate	32	32.0	
	Severe/Refractory	12	12.0	
Endoscopic Varices				**0.037**
	No varices	23	23.0	
	Grade I–II	33	33.0	
	Grade III–IV	44	44.0	
Ultrasound Findings				**0.019**
	Splenomegaly	45	45.0	
	Ascites on USG	33	33.0	
	Collateral veins	22	22.0	

Table 4. Neurological Complications and Infections (n = 100)

Complication	Category	n	%	p-value
Hepatic Encephalopathy				**0.001**
	Grade I	43	43.0	
	Grade II	20	19.0	
	Grade III	16	16.0	
	Grade IV	21	21.0	
Wernicke's Features				**0.0004**
	Confusion	51	51.0	
	Ataxia	30	30.0	
	Ophthalmoplegia	19	19.0	
Infection Profile				**<0.001**
	No infection	41	41.0	
	Pneumonia	31	31.0	
	SBP	15	15.4	
	UTI	7	7.0	
	Cellulitis	6	6.0	

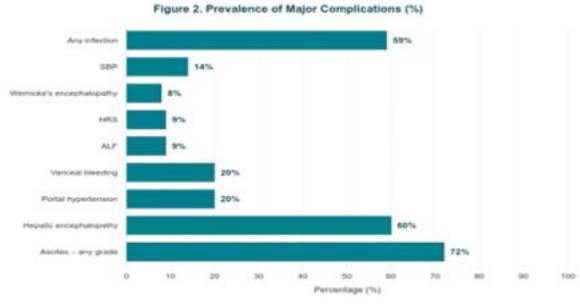

Figure 2. Prevalence of Major Complications (%)

Clinical Outcomes

In-hospital, 62% improved, 34% died, and 4% were referred for tertiary care. The majority of patients were discharged within 3–5 days (53%). At 28-day follow-up, 58% were alive, 35% had been readmitted, and 7% had died. At 90 days, cumulative mortality reached 15%. Statistically significant predictors of mortality included Child–Pugh class ($p = 0.021$), hepatic encephalopathy grade ($p = 0.034$), and presence of hepatorenal syndrome ($p = 0.028$).

Table 5. Renal Dysfunction, UGI Bleeding, SBP, and ALF (n = 100)

Complication	Category	n	%	p-value
UGI Bleeding				**<0.001**
	No active bleed	71	71.0	
	Variceal bleed	20	20.0	
	Non-variceal bleed	9	9.0	
Renal Status				**<0.001**
	No AKI	81	81.0	
	AKI	10	10.0	
	HRS	9	9.0	
SBP (Ascitic Fluid)				**<0.001**
	Culture negative	67	67.0	
	Neutrophils >250/mm³	20	20.0	
	Culture positive	13	13.0	
ALF Outcome				**0.326**
	Recovered	40	40.0	
	Chronic sequel	32	32.0	
	Death	28	28.0	

Table 6. Clinical Outcomes and Follow-Up (n = 100)

Outcome Measure	Category	n	%	p-value
Hospital Stay				**<0.001**
	<3 days	26	26.0	
	3–5 days	53	53.0	
	6–10 days	12	12.0	
	>10 days	9	9.0	
In-Hospital Outcome				**<0.001**
	Improved	62	62.0	
	Death	34	34.0	
	Referred	4	4.0	
28-Day Outcome				**<0.001**
	Alive	58	58.0	
	Readmitted	35	35.0	
	Death	7	7.0	
Summary: Major Complications				**0.014**
	Portal hypertension	20	20.0	
	Ascites (any)	72	72.0	
	Hepatic encephalopathy	60	60.0	
	UGI bleed	29	29.0	
	SBP	14	14.0	
	HRS	9	9.0	
	ALF	9	9.0	
	Wernicke's	8	8.0	

DISCUSSION

The present study provides prospectively collected data on ALD presentation, severity, complications, and mortality in a tertiary hospital serving the tribal belt of Gujarat — a population substantially underrepresented in existing clinical literature.

The overwhelming male predominance (92%) is consistent with the sociocultural acceptability of male alcohol consumption in rural Indian contexts and with the broader epidemiology of ALD [8]. Labourers and farmers together constituted 64% of the cohort, reflecting the documented association between manual occupational roles, economic stress, and alcohol dependence [4]. Country liquor was consumed by 64% of patients, consistent with published observations from rural India highlighting the widespread use of unregulated locally brewed spirits [9]. Such beverages may carry higher ethanol concentrations and additional hepatotoxic compounds, further amplifying liver injury.

Disease severity at presentation was striking. Child–Pugh B and C together accounted for 70%, and 39% had MELD 3.0 scores in the 20–29 range — indicating advanced disease in the majority at first hospital contact. This pattern of late presentation is a recurring finding in rural Indian studies, driven by geographic and financial barriers to early care and by cultural perceptions that normalise alcohol use [4]. The hypoalbuminaemia and hyperbilirubinaemia observed are consistent with established cirrhosis and decompensation.

Ascites in 72% and Grade III–IV oesophageal varices in 44% reflect the high portal hypertension burden in this cohort. Variceal bleeding in 20% aligns with rates of 18–25% reported in decompensated ALD series [10]. Endoscopic surveillance and prophylactic band ligation remain underutilised in rural settings due to resource constraints.

Hepatic encephalopathy in 60% — predominantly Grade I — reflects advanced liver dysfunction and underscores the importance of early detection. Grade I–II encephalopathy is often reversible with lactulose and rifaximin, but delayed recognition in peripheral facilities risks progression to higher grades [11]. Hepatorenal syndrome in 9% and SBP in 14% are markers of severe multiorgan involvement, both strongly associated with mortality in this cohort [12].

Wernicke's encephalopathy in 8% of patients highlights the nutritional deficiency — particularly thiamine depletion — inherent in prolonged alcohol dependence among economically deprived populations [8]. Empirical parenteral thiamine on admission is warranted as routine protocol. Infections in 59%, led by pneumonia (31%), reflect the immunosuppressed state of cirrhotic patients compounded by malnutrition [13].

In-hospital mortality of 34% falls within the 20–30% range reported from comparable Indian decompensated ALD studies [3, 10]. At 28-day follow-up, 93% survival and at 90 days, 85% survival reflect the reversibility of some complications but also the ongoing vulnerability of this population. The significant predictors of mortality — Child–Pugh class, encephalopathy grade, and hepatorenal syndrome — are consistent with established prognostic models [3, 12].

From a public health standpoint, country liquor consumption, labour-based alcohol payment schemes, and cultural embeddedness of communal drinking sustain high-frequency hepatotoxic exposure [4, 9]. Nutritional deficiencies further compress the timeline to decompensation. Community-level screening using portable ultrasound and biochemical panels, culturally sensitive cessation programmes, nutritional rehabilitation, and strengthened primary care capacity for early complication recognition are the most actionable interventions.

Limitations: Single-centre design, a sample of 100, absence of objective quantitative alcohol intake data, and lack of uniform histopathological staging limit generalisability. However, the prospective design and comprehensive clinical evaluation strengthen the validity of findings.

Strengths: This is among the few prospective studies characterising ALD specifically in a tribal Gujarat population. The systematic complication-specific analysis and short-term outcome data have direct utility for clinical and public health planning in resource-constrained settings.

CONCLUSION

Alcoholic liver disease in the tribal belt of Gujarat presents predominantly at advanced stages, with the majority exhibiting Child–Pugh Class B or C disease and high MELD 3.0 scores at first contact. Ascites, oesophageal varices, hepatic encephalopathy, and infections are prevalent complications, and in-hospital mortality is substantial. Country liquor consumption, prolonged drinking, occupational risk, and delayed presentation reflect the socioeconomic and cultural landscape of this population. Prospective surveillance, community screening, nutritional support, and contextually adapted alcohol cessation programmes represent the most actionable pathways to reducing this preventable burden.

DECLARATIONS

Funding: - This study received no external funding.
Conflicts of interest: - The authors declare no conflicts of interest.
Ethics: - The present study was approved by the IEC of Zydus Medical College and Hospital, Dahod (No. ZMCH/IEC/041(11)-2024), in compliance with ICMR Guidelines 2017 and NDCT Rules 2019.

REFERENCES

[1] Mukherjee PS, Vishnubhatla S, Amarapurkar DN, Das K, Sood A, Chawla YK, et al. Etiology and mode of presentation of chronic liver diseases in India: a multi centric study. PLoS ONE. 2017;12(10):e0187033. https://doi.org/10.1371/journal.pone.0187033

[2] Avila MA, Dufour JF, Gerbes AL, Zoulim F, Bataller R, Burra P, et al. Recent advances in alcohol-related liver disease (ALD): summary of a gut round table meeting. Gut. 2020;69:764–800. https://doi.org/10.1136/gutjnl-2019-319720

[3] Koumar L, Senthamizhselvan K, Barathi D, et al. Portal vein thrombosis in patients with cirrhosis of the liver: prevalence and risk factors. Cureus. 2023;15(12). https://doi.org/10.7759/cureus.50134

[4] Singh G, Mitra Y, Singh J, Padda AS. Prevalence of health problems among the regular alcohol users in urban and rural area of district Amritsar, Punjab, India. Public Health Review. 2019;6(1):35–40.

[5] Naskar A, Mondal A, Chatterjee R, et al. Assessing liver fibrosis in type 2 diabetes mellitus patients with metabolic dysfunction-associated steatotic liver disease. Cureus. 2024;16(6):e62405. https://doi.org/10.7759/cureus.62405

[6] Roy N, Nadda N, Kumar H, et al. Pattern recognition receptor CD14 gene polymorphisms in alcohol use disorder and liver disease susceptibility. Front Immunol. 2022;13:975027. https://doi.org/10.3389/fimmu.2022.975027

[7] Ravindra BS, Lahoti D, Das K. Epidemiology and treatment of chronic cholestatic liver disease in India. Int J Sci Rep. 2023;9(1):17–27.

[8] Mackowiak B, Fu Y, Maccioni L, Gao B. Alcohol-associated liver disease. J Clin Invest. 2024;134(3):e176345. https://doi.org/10.1172/JCI176345

[9] Pandit N, Patel V. Child birth practices and utilization of antenatal care services among migrant tribal women in urban Gujarat. Cureus. 2023;15(5):e39363. https://doi.org/10.7759/cureus.39363

[10] Kim HI, Park SY, Shin HP. Incidence and management patterns of alcohol-related liver disease in Korea. Sci Rep. 2021;11:6648. https://doi.org/10.1038/s41598-021-86197-z

[11] Otero Sanchez L, Karakike E, Njimi H, et al. Clinical course and risk factors for infection in severe alcohol-associated liver disease. Hepatology. 2021;74(5):2714–24. https://doi.org/10.1002/hep.31984

[12] Wani AA, Rashid Y. Epidemiology of liver cirrhosis, complications and management: a review. Saudi J Med Pharm Sci. 2024;10(8):603–7. https://doi.org/10.36348/sjmps.2024.v10i08.013

[13] Solomon SS, Srikrishnan AK, McFall AM, et al. Burden of liver disease among community-based people who inject drugs in Chennai, India. PLoS ONE. 2016;11(1):e0147879. https://doi.org/10.1371/journal.pone.0147879