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Observing trends in clinical features and etiopathology in Liver cirrhosis patients coming to a tertiary care center of a Metropolitan

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A B S T R A C T

Background: Liver cirrhosis is the final common pathway of various chronic liver insults and remains an important cause of morbidity and mortality globally. Its clinical presentation ranges from asymptomatic to overt hepatic decompensation, with etiological factors varying among populations. Trends in clinical features and etiopathology in tertiary care centers need to be understood to aid early diagnosis and management.

Objectives: To witness the clinical presentation, etiologic spectrum, and severity patterns of liver cirrhosis patients visiting a tertiary level care center in a metropolitan area.

Methods: Cross-sectional observational study was performed in 110 patients diagnosed with liver cirrhosis. Comprehensive demographic, clinical, biochemical, and etiological profiles were documented. Clinical manifestations were divided into symptoms and signs, whereas laboratory tests comprised hematological and biochemical parameters. Severity of the disease was evaluated by Child–Turcotte–Pugh (CTP) classification and Model for End-Stage Liver Disease (MELD) scores. Descriptive statistics were used to analyze the information.

Results: The patients' mean age was 48.2 ± 10.6 years with a peak incidence (40.9%) in the 41–50 years age group. There was an astonishing male predominance (male:female ratio 10:1). Abdominal distension (87.3%), jaundice (83.6%), and pedal edema (81.8%) were the most frequent symptoms, whereas pallor (42.7%), icterus (83.6%), and ascites (87.3%) were common clinical findings. Alcohol was the most common etiologic factor, responsible for 87.3% of the cases, followed by viral hepatitis (5.5%). Laboratory derangements were anemia (56.4%), hyperbilirubinemia (70.9%), hypoalbuminemia (81.8%), deranged liver enzymes (elevation of AST in 72.7%; ALT in 65.5%), and coagulopathy (77.3%). The mean MELD score was 16.8 ± 6.2. On CTP classification, 20% were class A, 45.5% class B, and 34.5% class C, indicating predominance of moderate-to-severe disease at presentation.

Conclusion: Liver cirrhosis in this urban population mostly involved middle-aged men, with alcohol being the major etiological factor. The clinical presentation was characterized by dominance of ascites, jaundice, and pedal edema, along with prominent biochemical derangements. The dominance of CTP classes B and C reflects late healthcare presentation. These results emphasize the need for early screening, public awareness, and proper intervention strategies to correct modifiable risk factors, particularly alcohol abuse.

Keywords: Liver cirrhosis, Clinical profile, Etiopathology, Alcohol, Child–Turcotte–Pugh, MELD score.

INTRODUCTION:

Liver cirrhosis is an irreversible and progressive disease that is marked by the extensive hepatic fibrosis and formation of regenerative nodules, which distort normal lobular architecture of the liver. As a terminal stage of several chronic liver injuries, it is a leading cause of worldwide morbidity and mortality that results in about 1.32 million deaths every year worldwide [1]. The natural course of cirrhosis comprises a compensated phase as an introduction followed by a decompensated phase characterized by complications like portal hypertension, variceal hemorrhage, ascites, spontaneous

bacterial peritonitis, hepatic encephalopathy, and hepatocellular carcinoma (HCC) [2]. These complications impose heavy clinical, economic, and social loads on patients, families, and the healthcare system.

The etiopathogenesis of cirrhosis is heterogenous and has changed significantly over time. Chronic viral hepatitis, particularly hepatitis B virus (HBV) and hepatitis C virus (HCV) infection, has been a leading etiology of cirrhosis worldwide [3]. Yet with the advent of successful HBV immunization programs, improvement in antiviral treatments, and universal adoption of direct-acting antivirals (DAAs) for HCV infection, the burden of viral cirrhosis has progressively diminished in most high-income countries [4]. On the other hand, alcoholic liver disease and non-alcoholic fatty liver disease (NAFLD) are becoming leading causes of cirrhosis, especially in the face of rising alcohol use, obesity, diabetes, and metabolic syndrome [5].

In low- and middle-income countries (LMICs), the situation is different. Though viral hepatitis remains a significant cause, restricted access to preventive interventions, diagnostic centers, and treatment results in late disease presentation [6]. In India, cirrhosis is one of the leading causes of death and is responsible for as much as 2–3% of all hospitalizations in tertiary care [7]. Etiologic distribution varies widely by region: alcohol is becoming the predominant cause in northern India, while in southern India viral hepatitis and cryptogenic etiologies contribute more significantly [8]. In addition, NAFLD, fueled by rapid urbanization and lifestyle transformation in urban settings, is rapidly being identified as an emerging etiology of cirrhosis [9].

Urban tertiary referral centers are central to cirrhosis investigation and control. The centers act as referral centers, and they attract patients from varying socioeconomic, cultural, and geographic backgrounds. Consequently, they offer a unique opportunity to observe heterogeneity in patterns of the disease and temporal trends. The spectrum of presentation at the time of presentation may range extensively—from minimal findings in compensated individuals to extreme decompensation necessitating intensive care. An awareness of such trends at the metropolitan level not only mirrors the changing dynamics of cirrhosis burden but also helps in maximizing clinical facilities like endoscopy facilities, intensive care units, and liver transplant services [10].

Although there has been a recognized burden, as of today, there is relatively less longitudinal data on both clinical characteristics and etiopathology of cirrhosis in urban Indian metropolitan tertiary care centers. The majority of published articles either highlight a single etiological cause (e.g., alcohol or hepatitis viruses) or concentrate on a specific outcome like HCC or variceal bleeding. Such global assessments combining both clinical and etiological patterns are sparse, thus remaining significant areas of ignorance. In addition, most existing research is limited to a particular geographic area, and it is unclear whether results in such settings reflect disease patterns in major urban referral centers.
Accordingly, this research aimed to overcome these shortcomings through the systematic analysis of patients with cirrhosis admitted to a metropolitan tertiary hospital. Particularly, it seeks to:

Describe the spectrum of clinical presentation on admission, with focus on complications including ascites, hepatic encephalopathy, variceal hemorrhage, jaundice, and renal impairment.

Consider the prevalence of etiopathological determinants, such as HBV, HCV, alcohol, NAFLD, autoimmune etiology, and cryptogenic cirrhosis.

Find temporal changes in relative contribution of various etiologies, observing the impact of prevention efforts, lifestyle changes, and enhanced diagnostic methods.

By the identification of both clinical characteristics and etiological determinants, this investigation aims to provide meaningful insights into trends in cirrhosis in metropolitan India. Such information are necessary for the customization of preventive interventions, informing policy actions, and optimizing clinical readiness for the increased burden of chronic liver disease in urban communities. Finally, results from metropolitan tertiary facilities can act as sentinel indicators for larger national trends, thus facilitating significant public health responses to liver disease.

MATERIAL AND METHODS:

This was a cross-sectional observational study conducted at a metropolitan tertiary care hospital in India. The institution serves as a referral hub for both urban and peri-urban populations. The study was conducted over 18 months (January 2022 to June 2023) in the Department of Medicine. The primary objective was to evaluate clinical features and etiopathological causes of liver cirrhosis among patients presenting to this center.

A total of 110 patients diagnosed with cirrhosis were included. Diagnosis of cirrhosis was based on a combination of:
Clinical findings: ascites, hepatic encephalopathy, jaundice, and stigmata of chronic liver disease.
Laboratory parameters: abnormal liver function tests, hypoalbuminemia, prolonged INR.

Radiological evidence: coarse hepatic echotexture, surface nodularity, caudate hypertrophy, splenomegaly, or evidence of portal hypertension on ultrasonography or CT.
Where available, histopathology was considered confirmatory [11,12].

Inclusion Criteria

- Patients aged ≥18 years with confirmed diagnosis of cirrhosis.
- Patients admitted during the study period.
- Those providing informed written consent (or via legal guardians if encephalopathy was present).

Exclusion Criteria

- Patients with acute liver failure without underlying cirrhosis.
- Cases with incomplete clinical records.
- Patients or families unwilling to participate.

Data were collected using a structured proforma and included:
Demographics: age, sex, residence, socioeconomic background, alcohol history, smoking status, and comorbidities.
Clinical features: jaundice, ascites, pedal edema, hepatic encephalopathy, gastrointestinal bleeding, hepatorenal syndrome, spontaneous bacterial peritonitis, and hepatocellular carcinoma.

Laboratory investigations: complete hemogram, liver and renal function tests, coagulation profile, viral markers (HBsAg, anti-HCV).
Imaging: abdominal ultrasonography with Doppler; contrast-enhanced CT when clinically indicated and diagnosis purpose.
Etiological attribution:
Viral hepatitis: Based on serology (HBsAg, anti-HCV), HBV DNA, or HCV RNA in our own institutional laboratory [13].
Alcoholic liver disease: Documented alcohol intake >40 g/day in males or >20 g/day in females for more than 5 years, with exclusion of other causes [14].
Non-alcoholic fatty liver disease (NAFLD): Imaging showing hepatic steatosis along with metabolic risk factors in the absence of significant alcohol intake [15].
Autoimmune hepatitis: Suggested by autoimmune markers (ANA, ASMA, AMA), biochemical profile, and supporting histology.
Cryptogenic cirrhosis: Diagnosed when no definite cause could be established despite complete evaluation.
Primary outcomes: Spectrum of clinical presentations and distribution of etiological factors.
Secondary outcomes: Relationship of etiological categories with severity of cirrhosis assessed by Child–Turcotte–Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores [16].
Statistical Analysis
Data from 110 patients were entered into Microsoft Excel and analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY).
Continuous variables were summarized as mean ± standard deviation (SD) or median with interquartile range (IQR).
Categorical variables were presented as frequencies and percentages.
Statistical tests: Chi-square or Fisher's exact test for categorical variables; independent t-test or Mann–Whitney U test for continuous variables as and when required.
A p-value <0.05 was considered statistically significant.
Ethical Considerations
The study protocol was approved by the Institutional Ethics Committee of the hospital. Written informed consent was obtained from all patients or their guardians. Patient confidentiality was maintained by anonymizing personal identifiers. The study adhered to the principles of the Declaration of Helsinki [17].

RESULTS:

Table 1. Age Distribution of patients studied

AGE IN YEARS	NO. OF PATIENTS	Percentage
<30	4	3.63
31-40	23	20.90
41-50	45	40.90
51-60	24	21.85
61-70	10	9.09
71-80	4	3.63
TOTAL	75	100

Above table shows the distribution of cases according to the age. Incidence Of cirrhosis was maximum in the age group of 41-50years followed by 51-60 Years

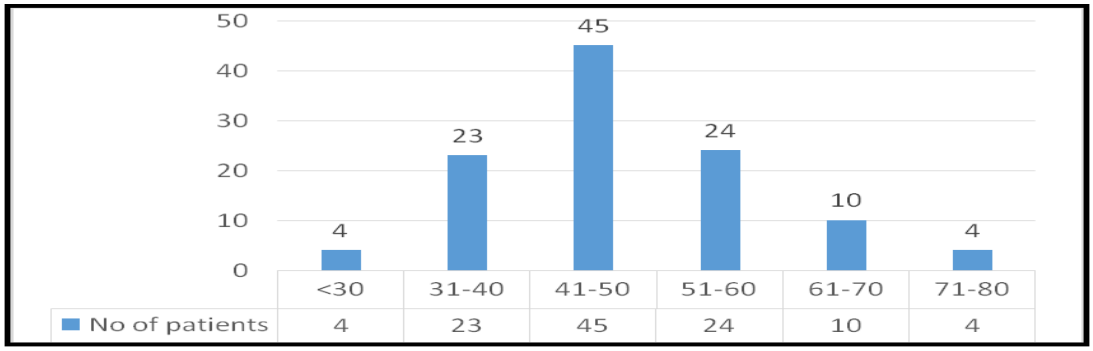

FIG.1 Distribution of patients according to age

Table 2: gender distribution of patients studied

Sex	Number of patients	Percentage
Male	100	90.91%
Female	10	9.09%
Total	110	100%

Among the patients studied males predominate the study populations with 91.91% with females accounting for only 9.09%

Table 3. Clinical features

Clinical symptoms	No. Of patients n=110	Percentage
DISTENDED ABDOMEN	96	87.27
JAUNDICE	92	83.6
PEDAL EDEMA	90	81.82
NAUSEA/VOMITNG	68	61.81
FATIGUE	53	48.18
HAEMETEMESIS	29	26.36
OLIGURIA	26	23.63
ALTERED SENSORIUM	15	13.63

Among the study population majority presented with ascites constituting 87.27% followed by jaundice constituting 83.6%

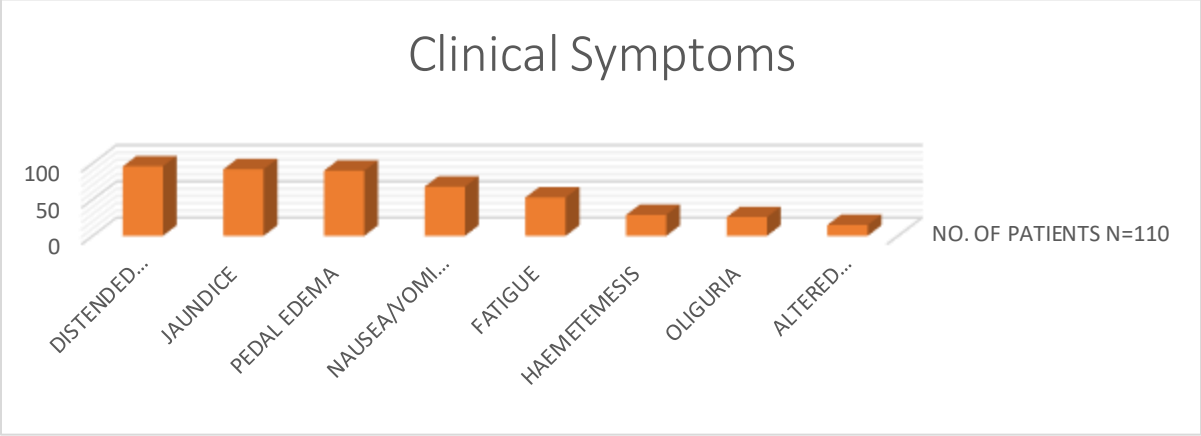

Figure 2: Distribution based on Clinical Symptoms

In the present cohort of 110 cirrhosis patients, the most frequently observed clinical sign was ascites, documented in 96 patients (87.27%), followed closely by icterus in 92 patients (83.6%) and pedal edema in 90 patients (81.82%), reflecting advanced disease and decompensation. Splenomegaly was detected in 56 patients (50.91%), while pallor was present in 47 patients (42.73%), indicating underlying anemia and hypersplenism. Less frequent findings included distended abdominal veins in 28 patients (30.8%) and hepatomegaly in 25 patients (27.5%), suggestive of portal hypertension and ongoing parenchymal liver changes. Fever was noted in 24 patients (21.82%), which may reflect underlying infections, spontaneous

bacterial peritonitis, or systemic inflammatory response. Overall, the predominance of ascites, icterus, and pedal edema highlights that most patients presented with clinically advanced and decompensated liver disease at the time of admission.

Table 4. Clinical Signs

CLINICAL SIGNS	NO.OF PATIENTS	%
1.PALLOR	47	42.73
2.ICTERUS	92	83.6
3.PEDAL EDEMA	90	81.82
4.ASCITES	96	87.27
5.DISTENED VEINS	28	30.8
6.HEPATOMEGALY	25	27.5
7.SPLENOMEGALY	56	50.91
8.FEVER	24	21.82

The clinical signs noted among the patients were most commonly Ascites constituting 87.27% and Icterus accounting to about 83.6%.

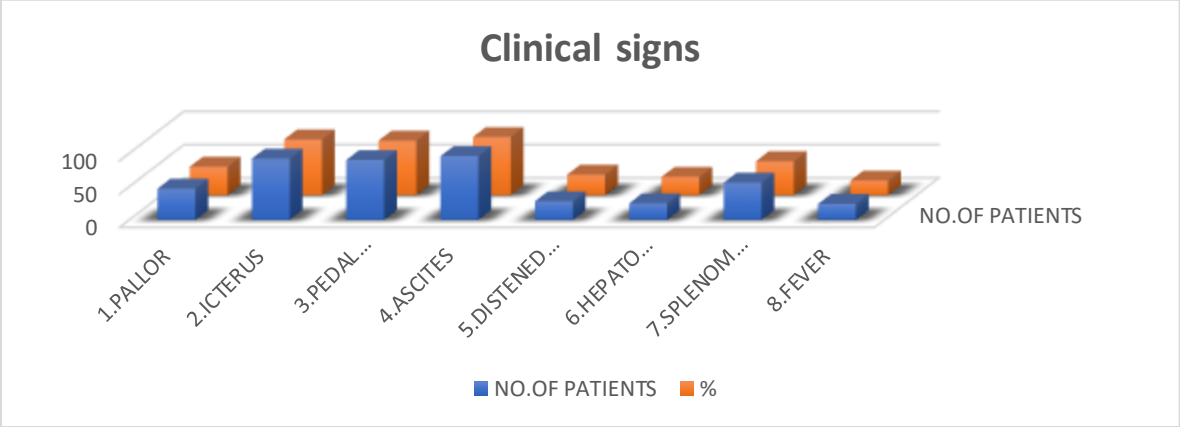

Fig.3. Distribution based on clinical Signs.

The etiological analysis of cirrhosis cases in the present study revealed that alcohol was the predominant cause, accounting for 96 patients (87.27%), thereby establishing it as the leading risk factor in this metropolitan cohort. Viral hepatitis was identified in 6 patients (5.45%), while less common causes included Wilson's disease in 2 patients (1.82%), Budd–Chiari syndrome in 1 patient (0.91%), and primary biliary cirrhosis in 1 patient (0.91%). Notably, in 4 patients (3.64%), no clear etiology could be established, and these cases were categorized as cryptogenic cirrhosis. The overwhelming contribution of alcohol to disease burden underscores its major role in the pathogenesis of cirrhosis in this region, while viral and metabolic causes contributed to a much smaller proportion of cases.

Table 5. Causes of Cirrhosis

PROBABLE CAUSE	NO.OF PATIENTS	Percentage
Alcohol	96	87.27
Viral	6	5.45
Wilson's disease	2	1.82
Budd Chiari Syndrome	1	0.91
Primary Billiary Cirrhosis	1	0.91
Unidentified	4	3.64
TOTAL	110	100

Laboratory Profile

Patients typically demonstrated deranged liver function tests. The mean serum bilirubin was 3.6 ± 2.4 mg/dL, and mean serum albumin was 2.7 ± 0.6 g/dL. INR prolongation (mean: 1.9 ± 0.4) was common, reflecting impaired synthetic function.

Table 6. Laboratory findings

Parameter	Mean ± SD	Abnormal in n (%)
Hemoglobin (g/dL)	10.1 ± 2.1	62 (56.4%)
Serum bilirubin (mg/dL)	3.6 ± 2.4	78 (70.9%)
Serum albumin (g/dL)	2.7 ± 0.6	90 (81.8%)
AST (IU/L)	88 ± 40	80 (72.7%)
ALT (IU/L)	74 ± 35	72 (65.5%)
INR	1.9 ± 0.4	85 (77.3%)
Serum creatinine (mg/dL)	1.3 ± 0.5	28 (25.5%)

Severity Assessment

When assessed by Child–Turcotte–Pugh (CTP) classification, the majority of patients were in Child-Pugh Class B (45.5%), followed by Class C (34.5%) and Class A (20.0%). The mean MELD score was 16.8 ± 6.2, reflecting moderate disease severity.

Table 7. Distribution by severity scores

Severity scale	Number (%)
Child–Turcotte–Pugh (CTP)	
Class A	22 (20.0%)
Class B	50 (45.5%)
Class C	38 (34.5%)
MELD score (mean ± SD)	16.8 ± 6.2

DISCUSSION:

Liver cirrhosis is the common final pathway of a host of chronic liver illnesses, which causes major morbidity and mortality globally. The current study, carried out in a tertiary care metropolis, offers important information on changing clinical presentations, etiopathological pattern, and laboratory features in patients with cirrhosis. Based on a sample of 110 patients, the results not only confirm classical correlations like the dominance of alcohol as a causative agent but also highlight significant changes in demographic presentation, clinical range, and severity parameters. This discussion critically evaluates these results against the background of prevailing literature and relates them to modern hepatology practice.

In the present study, the mean presenting age was 48.2 years, with most patients falling into the 41–50 years group (40.9%), followed by 51–60 years (21.8%). This is consistent with the mid-life predominance reported in earlier studies from India and other countries in the Indian subcontinent [18,19], which have seen a mean age of cirrhotic cohorts between 45–55 years. The youthful relative age of cirrhotic patients in the Indian subcontinent, as opposed to Western countries where cirrhosis occurs in the sixth decade, could be due to the earlier age of alcohol initiation, increased incidence of viral hepatitis among younger patients, and genetic factors [20].

Surprisingly, very few patients in our series were older than 70 years (3.6%). This underrepresentation of older patients can be explained in part by survival bias, when cirrhotics who develop decompensation die sooner, and in part by health-seeking patterns in which older people do not make it to tertiary centers. Earlier multicenter research in the United States and Europe has shown elderly cirrhotic patients presenting with unusual manifestations and poorer prognosis [21]. Thus, the lower ratio in our study underscores the need for early intervention among middle-aged individuals to avert early death.

The striking male excess (90.9%) in our group reaffirms well-established epidemiological trends in cirrhosis. Cirrhosis is more common among men than women worldwide, with a ratio of 2:1 to 5:1 based on etiology [22]. Alcohol-induced cirrhosis especially shows male predominance because of increased rates of risky drinking in men, with sociocultural reinforcements deterring women from admitting alcohol consumption [23]. Biological susceptibility, however, can vary because women have been found to develop alcohol-induced liver damage at lower cumulative doses due to varied metabolism and hormonal effects [24].

The comparatively low ratio of female cirrhotics in our study may therefore not so much represent an actual biological resilience but, in fact, sociocultural underreporting. In addition, the observation that nearly 9% of the patients were female emphasizes the need not to underestimate women in cirrhosis screening programs, especially with increasing trends in non-alcoholic fatty liver disease (NAFLD) in women worldwide [25].

The most frequent presenting symptom in our series was distended abdomen (87.3%), in keeping with ascites as the defining feature of decompensated cirrhosis. This was succeeded by jaundice (83.6%) and pedal edema (81.8%). Such a pattern is closely replicated by that of previous Indian studies wherein abdominal distension and jaundice continue to be leading complaints on presentation to hospital [26].

Interestingly, nausea/vomiting (61.8%) and fatigue (48.1%) were common yet underappreciated symptoms. These non-specific symptoms tend to precede more ominous decompensatory events and, when identified early, might result in timely interventions [27]. Alarmingly, hematemesis was seen in 26.3% of the patients, an overt sign of variceal bleeding due to portal hypertension. Prior research estimates that almost one-third of cirrhotics would develop variceal hemorrhage within their disease course, which carries high short-term mortality [28].

In addition, oliguria (23.6%) and altered sensorium (13.6%) were found, indicative of hepatorenal syndrome and hepatic encephalopathy, respectively. Both are complications indicative of advanced decompensation and are well-documented predictors of mortality [29]. The high percentage of such patients in our series may indicate late referral to tertiary care and emphasizes the importance of detection of cirrhosis at the level of the community and referral mechanisms in a timely manner.
Physical findings showed high frequencies of ascites (87.3%), jaundice (83.6%), and pedal edema (81.8%), all in keeping with the above-mentioned symptomatic profile. Splenomegaly (50.9%) was also common, representing chronic portal hypertension. Equivalent prevalence has been documented in African and South Asian research, in which portal hypertensive presentations continue to predominate owing to late presentation [30].

Other significant findings were dilated abdominal veins (30.8%), a stigmata of long-standing portal hypertension, and hepatomegaly (27.5%), which in end-stage disease is not as prevalent because of progressive fibrosis and atrophy of the liver. Of interest, pallor was present in 42.7% of patients, indicating chronic anemia. This anemia can be caused by hypersplenism, nutritional deficiencies, blood loss from the gastrointestinal tract, or bone marrow suppression secondary to chronic inflammation [31].

The identification of such signs is of clinical significance since they contribute to bedside diagnosis even prior to confirmatory imaging and laboratory studies. Additionally, their frequency highlights that cirrhosis still presents with classic decompensatory signs in Indians, in contrast with developed nations where cirrhosis related to NAFLD tends to have early and nonspecific presentations [32].

The near-ubiquity of alcohol (87.3%) as the causative agent in this group is both alarming and surprising. Various Indian reports have all uniformly listed alcohol as the predominant etiology of cirrhosis, and one that has eclipsed that of viral hepatitis, which was previously the dominant etiology [33]. By way of contrast, Western reports also identify alcohol as a predominant cause, but with an increasing role from NAFLD and the epidemic of obesity [34].

The other etiologies in our series were viral hepatitis (5.5%), Wilson's disease (1.8%), Budd–Chiari syndrome (0.9%), and primary biliary cirrhosis (0.9%). Notably, only 3.6% of cases were not determined. The comparatively modest contribution of viral etiologies may be a result of the effectiveness of hepatitis B immunization programs and improved knowledge, although hepatitis C is still underdiagnosed in India [35]. The infrequent presentation of Wilson's disease and autoimmune diseases underscores the necessity of sustaining diagnostic alertness in the presence of cirrhosis, especially in children.

The high alcohol correlation has significant public health connotations. It calls for awareness at a population level, more stringent controls over alcohol access, and the blending of hepatology with addiction medicine [36]. It also underscores the critical necessity of early screening of alcohol consumers for liver disease before decompensation ensues.

The laboratory results in our case series illustrate the traditional biochemical pattern of cirrhosis. Anemia (mean Hb 10.1 g/dL; 56.4% abnormal) is in keeping with the multifactorial pathogenesis outlined above. Elevated serum bilirubin (mean 3.6 mg/dL; abnormal in 70.9%) emphasizes defective hepatocellular excretory function, and hypoalbuminemia (mean 2.7 g/dL; abnormal in 81.8%) indicates defective synthetic function [37].

Raised transaminases (AST 88 IU/L, ALT 74 IU/L) were prevalent, although an AST:ALT ratio greater than 1 indicates alcohol-induced liver damage, in agreement with the etiological diagnosis. In fact, the AST:ALT ratio has been known for many years to be a biochemical sign of alcoholic cirrhosis [38].

Coagulopathy (mean INR 1.9; abnormal in 77.3%) indicates impaired synthesis of clotting factors and is a major determinant of both risk of bleeding and mortality. The mean creatinine of 1.3 mg/dL (abnormal in 25.5%) indicates renal impairment in a quarter of patients, compatible with developing hepatorenal dysfunction. Interplay between cirrhosis and renal damage is more recognized as a strong determinant of outcomes [39].

Severity scoring identified 45.5% of patients in Child–Turcotte–Pugh class B and 34.5% in class C, suggesting significant disease severity at presentation. The average MELD score was 16.8 ± 6.2, again showing high disease severity.

These observations are consistent with a number of Indian studies in which most cirrhotics present in Child B or C class, in contrast to developed countries where universal health screening results in earlier presentation [40]. The prognostic value of CTP and MELD scores is well documented, and both have general use in transplant allocation programs [41]. The high prevalence of advanced cases in our study highlights the absence of early referral systems and points to the necessity of community-based cirrhosis detection programs.

Worldwide, etiology of cirrhosis is evolving. Although alcohol and hepatitis viruses remain preponderant in most parts of the world, non-alcoholic steatohepatitis (NASH) secondary to metabolic syndrome has become a major etiology of cirrhosis and hepatocellular carcinoma in developed countries [42]. Notably, NAFLD-related cirrhosis was not significantly reported in our cohort, perhaps because of underdiagnosis or conflation with alcoholic cases.

In addition, whereas the West is experiencing the older age groups being impacted, our study reaffirms that Indian cirrhosis still disproportionately impacts young persons at their most productive ages [43]. This age discrepancy has huge socioeconomic implications, resulting in loss of workforce productivity and financial burden to families.

The overwhelming preponderance of alcohol as an etiologic factor points to a significant public health issue. Effective prevention interventions need to address more than clinical practice and also include policy-level interventions like raising taxation on alcohol, implementing more stringent restrictions on advertising, and community-level de-addiction services [44]. Additionally, cirrhosis care needs to be addressed through integrated care models that include hepatology, psychiatry, nutrition, and primary care.

Vaccination against hepatitis B and enhanced screening for hepatitis C have to go on, as these are significant but decreasing contributors. Furthermore, with the increasing obesity epidemic in India, NAFLD-related cirrhosis is likely to become a leading cause in the near future and proactive measures need to be developed to address this emerging burden .

Although the study yields important findings, several restrictions need to be noted. First, as a single-center hospital-based report, generalizability of the findings to community settings may not be complete. Second, the cross-sectional nature of the study prevents measurement of longitudinal endpoints such as survival and transplant-free duration. Third, certain etiologies might have been underdiagnosed because of the absence of sophisticated diagnostic facilities, especially NAFLD and occult viral infections. Lastly, recall bias might have affected patient reporting, especially on alcohol intake.

In spite of limitations, the study is reinforced through a thorough assessment of demographic, clinical, laboratory, and etiologic variables in a fairly large cohort of 110 patients. The observations present an unambiguous picture of cirrhosis trends within a metropolitan referral care environment and point towards areas of concern that demand immediate attention, most notably alcohol-induced liver disease.

Clinically, the evidence of a high rate of advanced disease at the time of presentation necessitates earlier detection by community-level screening with particular emphasis on high-risk individuals like chronic alcoholics. Additionally, the high rate of complications such as variceal bleeding, ascites, and encephalopathy mandates fortification of emergency hepatology services.

Future studies should be directed towards future multicenter cohorts to effectively capture the changing spectrum of cirrhosis in India. Special attention should be given to emerging etiologies such as NAFLD and autoimmune liver disease. Moreover, interventional trials on alcohol abstinence programs incorporated into hepatology services are an urgent requirement to stem the tide of alcohol-related cirrhosis .

Improvements in non-invasive assessment of fibrosis, including elastography and serum biomarkers, should be incorporated into screening programmes to facilitate earlier detection. Additionally, longitudinal follow-up studies need to assess survival predictors, responses to treatment, and quality of life outcomes.

CONCLUSION:

This report highlights that alcohol remains the most common etiology of liver cirrhosis in urban India, with patients presenting predominantly at middle age and with severe decompensatory features. Clinical presentation continues to be dominated by ascites, jaundice, and pedal edema, with laboratory profiles reinforcing compromised synthetic and excretory activity. The large number of Child B and C cases indicates late presentation, with important prognostic and healthcare burden implications.

Global comparisons draw attention to distinct demographic trends in India, most notably the earlier age of presentation and the dominance of alcohol over metabolic causes. Such observations call for immediate public health interventions directed toward alcohol use, as well as upgraded screening programs to enable early detection and timely treatment.

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