Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/pc2w4w65

PROFILE OF ALCOHOL-RELATED LIVER DISEASE AMONG PATIENTS ATTENDING TERTIARY CARE HOSPITAL IN INDIA.(INSIGHTS FROM STUDY IN NORTHWESTERN INDIA)

Dr. Prashanth.k[1*], Dr Naman Bansal[2], Dr. Abhishek[3], Dr. Bhuddi Prakash Meena[4]

[1*]MBBS, MD (Internal Medicine), DM Resident (Medical Gastroenterology and Hepatology) (1)
[2]MBBS, MD (1) DM Resident (Medical gastroenterology and Hepatology) (1)
[3]MBBS, MD DM RESIDENT (Medical Gastroenterology and Hepatology)
[4]MD.DM Department of Gastroenterology and Hepatology, [NIMS&R], Jaipur, Rajasthan, India

***Corresponding Author:** Dr. Prashanth.K
*Department of Gastroenterology and Hepatology [NIMS &R] [DELHI JAIPUR ROAD SHOBHA NAGAR] Jaipur, Rajasthan, India MOBILE NO: +919553766701 Email: [STANLEYPRASHANTH1@GMAIL.COM] ORCID ID: [0009-0001-1855-2351]

Abstract
Alcohol related liver disease (ALD) is one of the most common cause for chronic liver disease and deaths in India. Material and methods This was a prospective observational study at the tertiary care centre to determine the clinical profile, pattern of drink, biochemistry of liver function test as well as prognostic factors in patients with ALD. We included a total of 281 patients with heavy drinking and liver disorder. Majority of the patients were males (90.75%), from lower middle and lower socio-economic status, Most of them being rurality, consuming country made liquor for more than 10–20 years on a daily basis. The most frequent complications were ascites, gastrointestinal bleeding, hepatic encephalopathy and hepatorenal syndrome.Laboratory test results included anaemia, hyperbilirubinemia, AST/ALT> 1:5, hypoalbuminemia and extended INR. Frequency of consumption of country liquor and >20 years alcohol consuming patients had significantly higher disease severity scores (Maddrey DF, MELD, and Child-Pugh). Neutrophil/lymphocyte ratio was significantly correlated with the prognostic scores, and may be a useful simple prognostic factor. General mortality was 18.15% and its occurrence increased, reaching maximum prevalences in hepatorenal syndrome and hepatic encephalopathy complexes.

Keywords: Alcohol-related liver disease, Drinking pattern, MELD score, Child-Pugh score, Neutrophil-lymphocyte ratio, Hepatic encephalopathy, HRS.

INTRODUCTION

Alcohol is a chemical substance capable of producing pharmacological effects consistent with narcotics including central nervous system and respiratory depression and induction of euphoria, tolerance and addiction. The word “Alcohol” has been derived from the Arabic word “ALKUHI” meaning essence[1]. Alcoholism is a major threat to public health in both developed and developing countries. In India, according to the Sample Registration Survey in 2014, the prevalence of alcohol consumption among adult males was found to be 10%[2]. The prevalence of alcohol use disorder in India in the year 2010 was found to be 4.5% and that of alcohol dependence was found to be 3.8%[3]. The National Mental Health Survey of India 2015–2016 found the prevalence of alcohol use disorder

to be 9% in adult men[4]. In India, in the year 2016, the alcohol-attributable fraction of all-cause deaths were found to be 5.4% and 62.9% of all the deaths because of liver cirrhosis, and 33.1% because of road traffic accidents were attributable to alcohol use[5].

According to recent data published by the World Health Organization (WHO), the total per capita consumption of alcohol by individuals above 15 years of age is 6.2 L of pure alcohol per year, which equals 13.5 g of pure alcohol per day. However, there is a wide variation between the WHO regions and member states. Nearly 5.1% of the global burden of disease is attributable to alcohol consumption, and it causes nearly 3.3 million deaths every year[6].

Over the last 30-40 years, alcohol consumption has increased in quantity and frequency world over. Today alcohol abuse has become a worldwide social and medical problem. With the rapid socio-economic and cultural changes, the alcohol is viewed as a symbol of social status and prestige as projected by their role models. The age at which people start alcohol has also declined.

Alcoholism is a repetitive pattern of drinking alcohol that has adverse effects on social, family, occupational or health status. It is a primary, chronic disease with genetic, psychosocial and environmental factors influencing its development and manifestations. The disease is often progressive and fatal. It is characterized by impaired control overdrinking, preoccupation with the drug alcohol, use of alcohol despite adverse consequences and distortions in thinking, most notably denial. Each of these symptoms may be continuous or periodic.

An alcoholic is a person who progressively consumes an amount of ethanol capable of producing pathological changes and exhibits a cumulative pattern of social behaviour associated with drinking including physical injuries, problems with family, job, and accidents while driving.

Alcohol dependence is a maladaptive pattern of alcohol use leading to clinically significant impairment or distress. Dependence may be physical or psychological. Physical dependence is characterized by the presence of withdrawal symptoms and tolerance[7].

The alcohol-related liver disease covers a spectrum of disorders beginning from the fatty liver, progressing at times to alcoholic hepatitis and culminating in alcoholic cirrhosis, which is the most advanced and irreversible form of liver injury related to the consumption of alcohol.

The first stage is hepatic steatosis. It involves the accumulation of small fat droplets around liver cells, specifically around the venules, and approach the portal tracts. The altered intracellular redox potential leads to the accumulation of intracellular lipids. Fatty liver is generally considered a reversible condition. On further progression, there is marked steatosis, hepatocellular necrosis, and acute inflammation. Eosinophilic fibrillar material (Mallory hyaline bodies) forms in swollen hepatocytes. This stage is known as alcoholic hepatitis. Severe lobular infiltration of polymorphonuclear leukocytes is abundantly present in this condition in contrast to most other types of hepatitis where mononuclear cells localize around portal triads.The end-stage of liver disease is alcoholic cirrhosis. At this stage, fibrotic septae surround regenerative nodules in the liver. The deposition of collagen typically occurs around the terminal hepatic vein and along the sinusoids, leading to a peculiar pattern of fibrosis in alcoholic cirrhosis.

Quantity and duration of the patient's alcohol intake are the highest risk factors for the development of liver disease. The beverage type plays a minimal role. Women are more susceptible than men. Obesity and high-fat diet also increase the risk of alcohol-related liver disease. Concurrent hepatitis C infection is associated with younger age of onset, more advanced histological damage, and decreased survival. In the health care delivery system, the physician is strategically placed to encounter patients with health-related problems associated with alcohol. Alcohol use is related to morbidity affecting various systems in the human body.

Alcoholism is influenced by socio-cultural factors and its impact is influenced by genetical factors. Though there are various studies on the profile of the alcohol-related liver disease in India, Hence, present research endeavours to study the profile of the alcohol-related liver disease in Tripura.

AIMS AND OBJECTIVES

AIM:

To study the profile of the alcohol-related liver disease in a tertiary care centre,

OBJECTIVES:

➢ To study the clinical profile of patients with alcohol-related liver disease.

➢ To study the Bio-chemical profile of patients with alcohol-related liver disease.

➢ To stratify the risk (Child pugh score) / prognosticate the morbidity and mortality of patients with alcohol-related liver disease.

➢ To correlate with alcohol-consuming patterns of study subjects.

MATERIAL AND METHODS

(i) Study area:

This is a hospital-based study both outpatients and admitted patients, at NIMS & R Jaipur Rajasthan

(ii) Study population:

All diagnosed cases of alcohol-related liver disease attending the Department of Medical Gastroenterology were my study subjects

Inclusion Criteria

Patients with clinical or investigational evidence of liver dysfunction with chronic significant alcohol intake are included.

Exclusion criteria:

- Patients with clinical or investigational evidence of liver dysfunction without chronic significant alcohol intake.
- Patients who have not consented.

(iii) Sample size

According to the landmark study by Girish et al, the prevalence of alcohol consumption was 23.7 %. [57]For confidence level of 95% with a margin of error of around 5 %, the sample size taken for the study was calculated using the following formula:

Variables	Values
Estimated true proportion (p)	0.237
Desired precision	0.05
Confidence level (CI)	0.95

Formula:

$n.= (z^2 \times p\,(1 - p))/e^2$

Where:

- Z = value from standard normal distribution corresponding to the desired confidence level (Z=1.96 for 95% CI)
- P is expected true proportion (of total admissions)
- e is the desired precision (half desired CI width).

After substituting the values, the sample size of 281 was obtained.

(iv)Study design:

A prospective observational study

(v) Study intervention if any should be listed out in detail.

No intervention needed

(vi) Study duration

12 months (01/07/2024 - 30/06/2025)

(vii)Method of measurement of the outcome of interest

Outcome Variable:

- Demographic characteristics
- Alcohol consuming patterns
- Prognostic scoring
- Mortality rate

Methodology

A structured questionnaire was prepared under the guidance of the thesis guide. Subjects were recruited in the study after their informed consent. A detailed socio-demographic, alcoholism history and clinical history was taken from each patient and complete physical examination was done. Various biochemical and ultrasonographic findings were recorded in a specially designed proforma. Alcoholism history included four measures – type, duration (years), amount (units per day) and frequency (days per month). Significant alcohol consumption was defined as an average consumption of >210 grams of alcohol per week in men or >140 grams of alcohol per week in women over at least a two-year period[86]. The study group was divided based on the amount of alcohol intake as light when alcohol intake was < 360 units per month, moderate 360-719 units per month and heavy 720 unit or more per month[84].

A unit of alcohol was defined as amounts of liquor equivalent to 10 gram of pure alcohol. This amount equates roughly to 30 ml of spirits (concentration 40% by volume, e.g. whisky, vodka, gin), 100 ml of wine or 250 ml of beer. The alcohol concentration in local country-made spirits has no fixed standards, ranging from 25% to 50% by volume. Hence in the present study, a unit of country liquor was also taken as 30 ml, equivalent to branded spirits.[84]

The duration was defined as short when it was up to 10 years, moderate when it was 11-20 years and longer if it was more than 20 years. Average amount per month was calculated by multiplying daily amount with frequency. Patients were divided according to their type (fermented alcohol , distilled alcohol or Indian made foreign liquor, Country or indigenous or local alcohol and mixture of above), amount and duration.

Alcohol-related liver disease was suspected in patients with a history of significant alcohol consumption who presented with[87]:

●Abnormal transaminases (particularly if the aspartate aminotransferase [AST] is greater than the alanine aminotransferase [ALT])

●Hepatomegaly

●Radiographic imaging suggesting hepatic steatosis or fibrosis/cirrhosis With or without Biopsy

Organ failures were defined as (a) renal (creatinine >1.5 mg/dL in the absence of other causes of kidney disease), (b) hepatic encephalopathy (as per West Haven criteria) in the absence of other acute neurological diseases, and (c) circulatory dysfunction (mean arterial pressure <70 mm Hg or needing inotrope support).

Sepsis was diagnosed in the presence of high total leukocyte count with neutrophilia and procalcitonin/C-reactive protein level (>0.5 μg/mL/>10 mg/L) and/or features of the systemic inflammatory response syndrome. [88]

Infections were diagnosed either by culture (of blood, urine, ascitic fluid) positivity or by evidence of infection on chest imaging, urine microscopy, ascitic fluid polymorphonuclear count more than 250 cells/mL, clinical examination compatible with cellulitis or bacterial infection at any other site.[89]

Based upon these clinical and biochemical data, various prognostic markers (Maddrey's Discriminant function score [DF] score, Model for end-stage liver disease [MELD] score and Child-Pugh score) were calculated for each participant. All the admitted participants were admitted during hospitalization for any complications of alcohol-related liver disease. Disease outcome in terms of either death or improvement was noted and the in-hospital mortality rate was calculated.

All these data were compared and correlated with each other to determine –
1. Comparison of complication rate, prognostic scores and mortality rate among different groups according to type, amount and duration of alcoholism.
2. Correlation of different biochemical and hematologic parameters with complication rate, prognostic scores and mortality rate.
3. Comparison of mortality rates between low risk and high-risk groups of different prognostic scores

OPERATIONAL DEFINITIONS

Tab. 4 Modified Kuppuswamy scale [90]

Education of head of family			Score
Professional degree			7
Graduate or postgraduate			6
Intermediate or post high school diploma			5
High school certificate			4
Middle school certificate			3
Primary school certificate			2
Illiterate			1
Occupation of head of family			
Professional (white collar)			10
Semi-professional			6
Clerical, shop-owner/farm			5
Skilled worker			4
Semi-skilled worker			3
Unskilled worker			2
Unemployed			1
Monthly income of family			
In 2001 (Base year)	**In 2017 (January 2017 CPI)**	**In 2019 (February 2019 CPI)**	**Score**
≥15,197	≥41,430	≥52,734	12
7,595-15,196	20,715-41,429	26,355-52,733	10
5,694-7,594	15,536-20,714	19,759-26,354	6
3,793-5,693	10,357-15,535	13,161-19,758	4
2,273-3,792	6,214-10,356	7,887-13,160	3
761-2,272	2,092-6,213	2,641-7,886	2
≤760	≤2,091	≤2,640	1
Socioeconomic class			**Total score**
I	Upper		26-29
II	Upper middle		16-25
III	Lower middle		11-15
IV	Upper lower		5-10
V	Lower		01-04

Rural: population less than 10,000.
Semi-Urban: 10,000 and above and less than 1 lakh.
Urban: 1 lakh and above and less than 10 lakh.
Metropolitan: 10 lakh and above.
Unemployed: a situation in which the person is capable of working both physically and mentally at the existing wage rate, but does not get a job to work
Unskilled: Person not skilled in a branch of work or lacking technical training
Semiskilled: Person who has only a part of the professional training of their trade but have sufficient experience in that field.

Clerical: A person employed in an office or government agency who performs various tasks such as keeping records or accounts, filing, letter writing, or transcribing.
Semi-professional: A person with an occupation that requires advanced knowledge and skills but is not widely regarded as a true profession.
Professional: A member of a profession or any person who earns their living from a specified professional activity.

Alcoholism

Either a pattern of pathological alcohol use or impairment in social or occupational functioning due to alcohol, and either tolerance or withdrawal.[91]

Alcohol abuse

It is defined as a pattern of pathological use for at least a month that causes impairment in social and occupational functioning.[92]

Alcohol Use Disorder (*DSM-5* criteria):

A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by 2 or more of the following, occurring at any time in the same 12-month period:

- Alcohol is often taken in larger amounts or over a longer period than was intended.
- There is a persistent desire or unsuccessful efforts to cut down or control alcohol use.
- A great deal of time is spent in activities necessary to obtain alcohol, use alcohol, or recover from its effects.
- Craving, or a strong desire or urge to use alcohol.
- Recurrent alcohol use failing to fulfil major role obligations at work, school, or home.
- Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol.
- Important social, occupational, or recreational activities are given up or reduced because of alcohol use.
- Recurrent alcohol use in situations in which it is physically hazardous.
- Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol.
- Tolerance, as defined by either of the following:

1. A need for markedly increased amounts of alcohol to achieve intoxication or desired effect.
2. A markedly diminished effect with continued use of the same amount of alcohol.

- Withdrawal, as manifested by either of the following:

3. The characteristic withdrawal syndrome for alcohol

2. Alcohol (or a closely related substance, such as a benzodiazepine) is taken to relieve or avoid withdrawal symptoms.[92]

Alcoholic fatty liver

It defined as an early and reversible consequence of excessive alcohol consumption on liver developing in an individual who consumes more than 60 g of alcohol per day.[93]

Alcoholic hepatitis

It was defined as the recent onset of jaundice with SGOT/SGPT (both in 100s) >1.5–2 without clinical/ultrasonological evidence of cirrhosis or liver decompensation (i.e., ascites and/or encephalopathy) in patients with recent (within 1 month) heavy consumption of alcohol.[93]

Cirrhosis

It is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury that leads to portal hypertension and end-stage liver disease. [93]

Sepsis

It is a life-threatening organ dysfunction caused by a dysregulated host response to infection. Septic shock is defined as lactate levels rising above 2 mmol L^{-1} without hypovolemia and initiation of vasopressor treatment to keep mean arterial pressure above 65 mmHg.[94]

Hepatic encephalopathy

It is a reversible syndrome of impaired brain function occurring in patients with advanced liver failure.[95]

Hepato renal syndrome[96]

Major criteria

- Chronic or acute liver disease with advanced hepatic failure and portal hypertension
- Low GFR: serum creatinine >133 μmol/L or 24h creatinine clearance <40 mL/min
- Absence of shock, ongoing bacterial infection or treatment with a nephrotoxic drug
- Absence of gastrointestinal or renal fluid losses
- No sustained improvement in renal function following diuretic withdrawal and expansion of plasma volume with 1.5 L of isotonic saline
- Proteinuria <0.5 g/d and no ultrasonographic evidence of obstructive uropathy or parenchymal renal disease

Additional criteria

- Urine volume <0.5 L/d
- Urine sodium <10 mmol/L
- Urine osmolality > plasma osmolality
- Urine red blood cells <50 high power field
- Serum sodium concentration <130 mmol/L

Maddrey's discriminant function[97]

$$DF = (4.6 \times \text{increase in PT sec.}) + (\text{serum bilirubin mg/dl})$$

Model for End-Stage Liver Disease[98]

$$\text{MELD} = 3.78 \times \log_e \text{serum bilirubin (mg/dL)} + 11.20 \times \log_e \text{INR} + 9.57 \times \log_e \text{serum creatinine (mg/dL)} + 6.43 \text{ (constant for liver disease etiology)}$$

Tab. 5 Child pugh score[99]

Clinical and Lab Criteria	Points*		
	1	2	3
Encephalopathy	None	Mild to moderate (grade 1 or 2)	Severe (grade 3 or 4)
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
Prothrombin time			
Seconds prolonged	<4	4-6	>6
International normalized ratio	<1.7	1.7-2.3	>2.3
Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points) Class A = 5 to 6 points (least severe liver disease) Class B = 7 to 9 points (moderately severe liver disease) Class C = 10 to 15 points (most severe liver disease)			

.

Statistical analysis:
The collected data was entered and analyzed using Microsoft office excel 2018 and MINITAB version 17. Frequencies of all variables were taken as check frequencies. Mean and standard deviation (SD) was calculated for continuous variables. Chi-square test was used to analysis of frequencies. . Odds Ratio and Relative risk was calculated using MEDICAL software. The test was considered statistically significant when the p-value was less than 0.05.

Ethical Consideration:
Informed written consent was obtained from every participant as per modified ICMR template, ensuring confidentiality while collecting and analyzing the data which was used for research purpose only. The application was placed before the Institutional Ethics Committee.

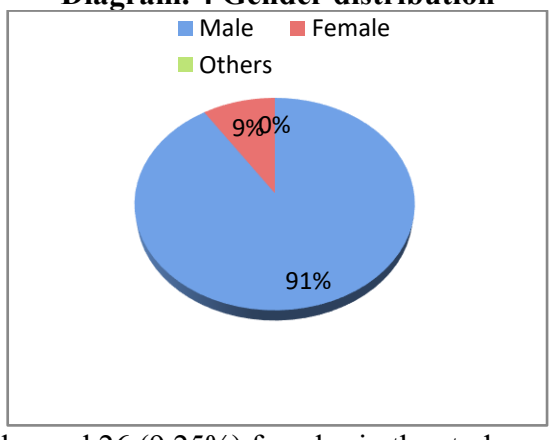
OBSERVATION AND RESULT

Tab.6 Socio-Demographic profile of subjects with alcoholism

Characteristics	Subcategories	No of Subjects
Gender	Male	255 (90.75%)
	Female	26 (9.25%)
Age distribution	20-40 yrs.	113 (40.21%)
	40-60 yrs.	92 (32.74%)
	60-80 yrs.	76 (27.05%)
	>80yrs	00 (o%)
Marital status	Married	268 (95.37%)
	Unmarried	13 (4.63%)
Locality	Rural	175 (62.28%)
	Semi urban	51 (18.15%)
	Urban	55 (19.57%)
Occupation	Professional	4 (1.42%)
	Semiprofessional	29 (10.32%)
	Clerical/shop/farm	58 (20.64%)
	Semiskilled	67 (23.84%)

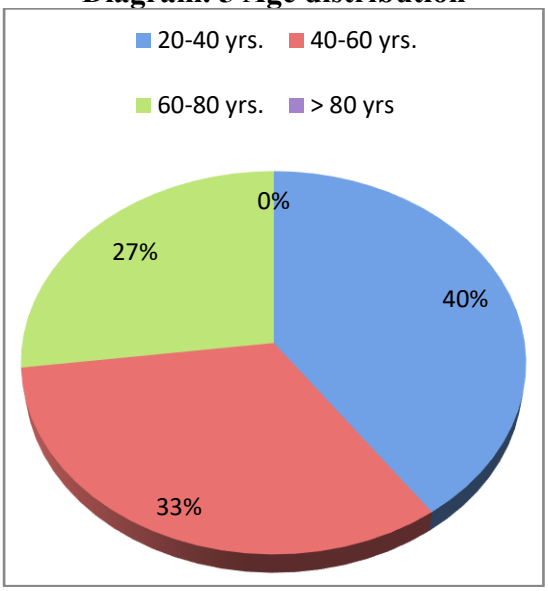
	Unskilled	89 (31.67%)
	Unemployed	34 (12.10%)
Education	Illiterate	43 (15.30%)
	Pre-primary	6 (2.14%)
	Primary	64 (22.78%)
	Upper primary	52 (18.51%)
	Secondary	67 (23.84%)
	Senior secondary	25 (8.90%)
	UG	21 (7.47%)
	PG	3 (1.07%)
	Doctorate	0 (0%)
Socio-economic category	Lower	89 (31.67%)
	Lower middle	102 (36.30%)
	Middle	35 (12.46%)
	Upper lower	33 (11.74%)
	Upper middle	22 (7.83%)
	Upper	00 (0%)
Religion	Hindu	269 (95.73%)
	Muslim	9 (3.20%)
	Christian	3 (1.07%)
	Others	0 (0%)

Diagram. 4 Gender distribution



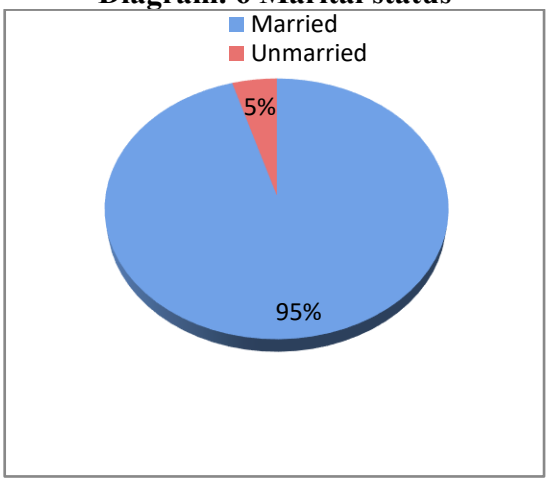
There were255 (90.75%) males and 26 (9.25%) females in the study.

Diagram. 5 Age distribution



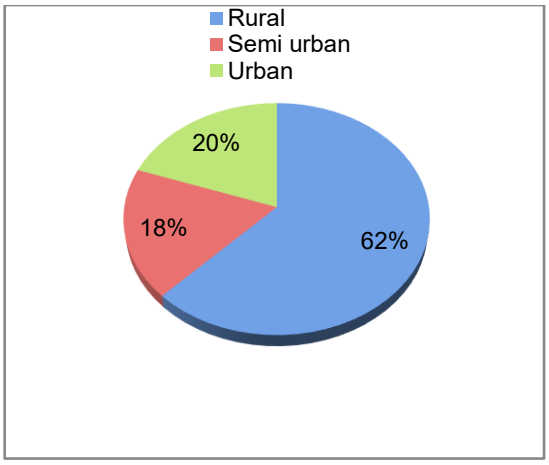
There were 113 (40.21%) patients within 20-40 years, 92 (32.74%) patient within 40-60 years, 76 (27.05%) patients within 60-80 years.

Diagram. 6 Marital status



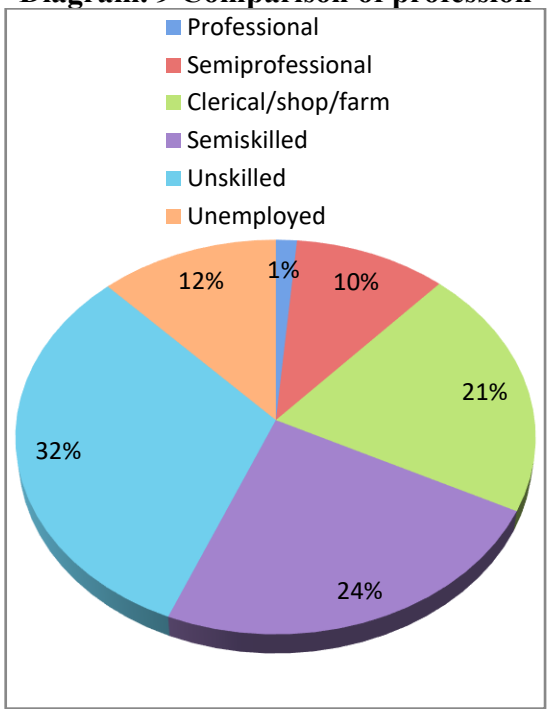
268 (95.37%) were married and 13 (4.63%) were unmarried.

Diagram. 8 Comparison of residence



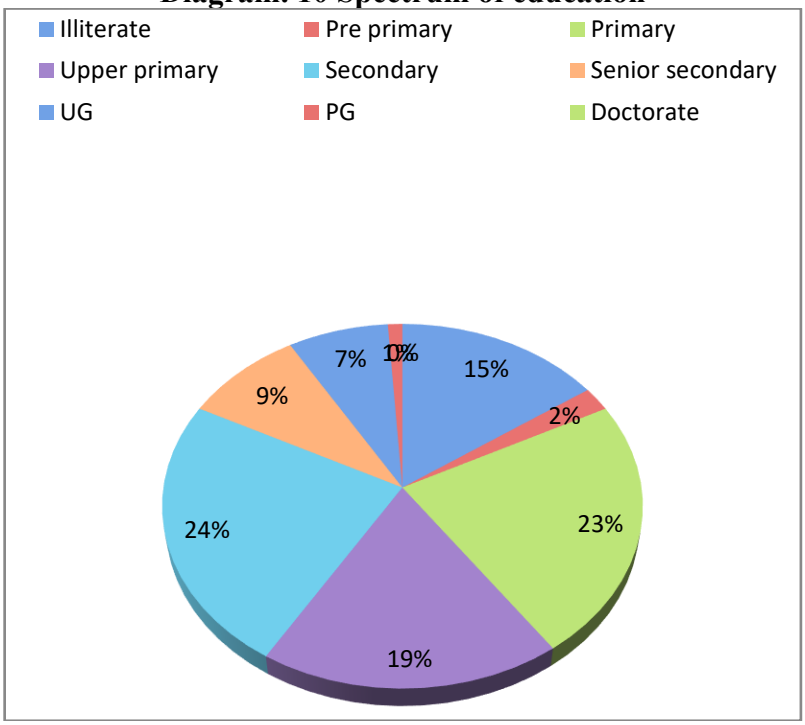
175 (62.28%) were rural, 51 (18.15%) were semi urban and 55 (19.57%) were urban.

Diagram. 9 Comparison of profession



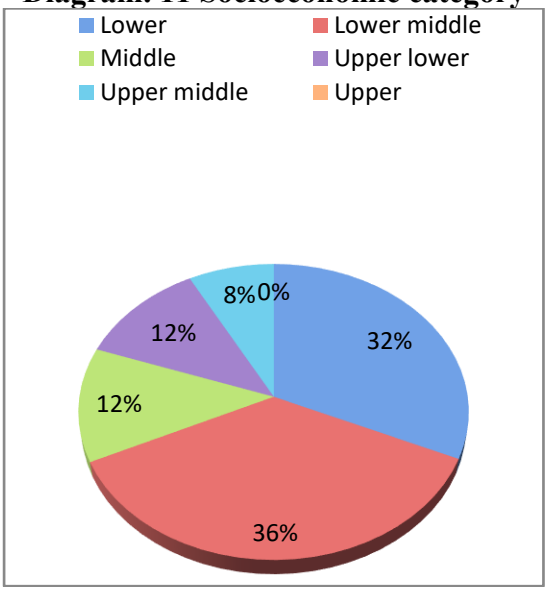
4 (1.42%) were professionals, 29 (10.32%) were semiprofessionals, 58 (20.64%) were in Clerical/shop/farm jobs, 67 (23.84%) were in semi skilled jobs, 89 (31.67%) were in unskilled jobs, 34 (12.10%) were unemployed.

Diagram. 10 Spectrum of education



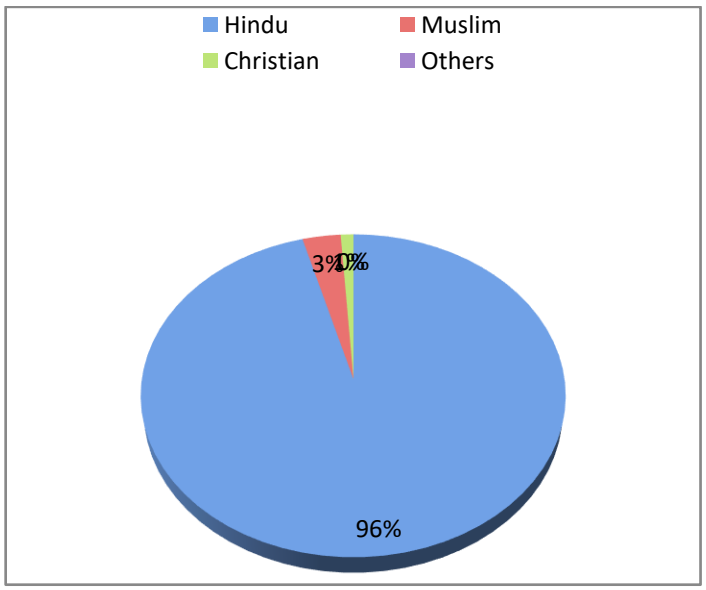
43 (15.30%) were illiterates, 6 (2.14%) studied preprimary, 64 (22.78%) studied primary, 52 (18.51%) studied upper primary, 67 (23.84%) studied secondary, studied senior secondary, 21 (7.47%) studied UG and 3 (1.07%) studied PG.

Diagram. 11 Socioeconomic category



89 (31.67%) belonged to the lower class, 102 (36.30%) belonged to the lower middle class, 35 (12.46%) belonged to the middle class, 33 (11.74%) belonged to the upper lower class, 22 (7.83%) belonged to the upper-middle class.

Diagram. 12 Spectrum of religion



269 (95.73%) were Hindus, 9 (3.20%) were Muslims, 3 (1.07%) were Christians.

Tab. 7 Characteristics of Alcohol consumption pattern

Characteristics	Subcategories	No of Subjects
Time of alcohol consumption	Only night	135 (48.04%)
	Only day	0 (0%)
	Both	146 (51.96%)

Frequency of alcohol consumption	< 1 time / month	0 (0%)
	1-3 times / month	0 (0%)
	1-2/W	16 (5.69%)
	3-6/W	84 (29.89%)
	Daily	181 (64.41%)
Age at start of alcohol consumption	< 20 yrs	87 (30.96%)
	20-30 yrs	152 (54.09%)
	> 30 yrs	42 (14.95%)
Age at start of alcohol liver disease	20-40 yrs	91 (32.38%)
	40-60 yrs	127 (45.20%)
	60-80 yrs	63 (22.42%)
	>80 yrs	0 (0%)
The occasion of drinking pattern	Single	175 (62.28%)
	Group	72 (25.62%)
	Both	34 (12.10%
Cause of Alcoholism	Environmental	44 (15.66%)
	Social	196 (69.75%)
	Psychosocial	41 (14.59%)
	Biological	0 (0%)
	Others	0 (0%)

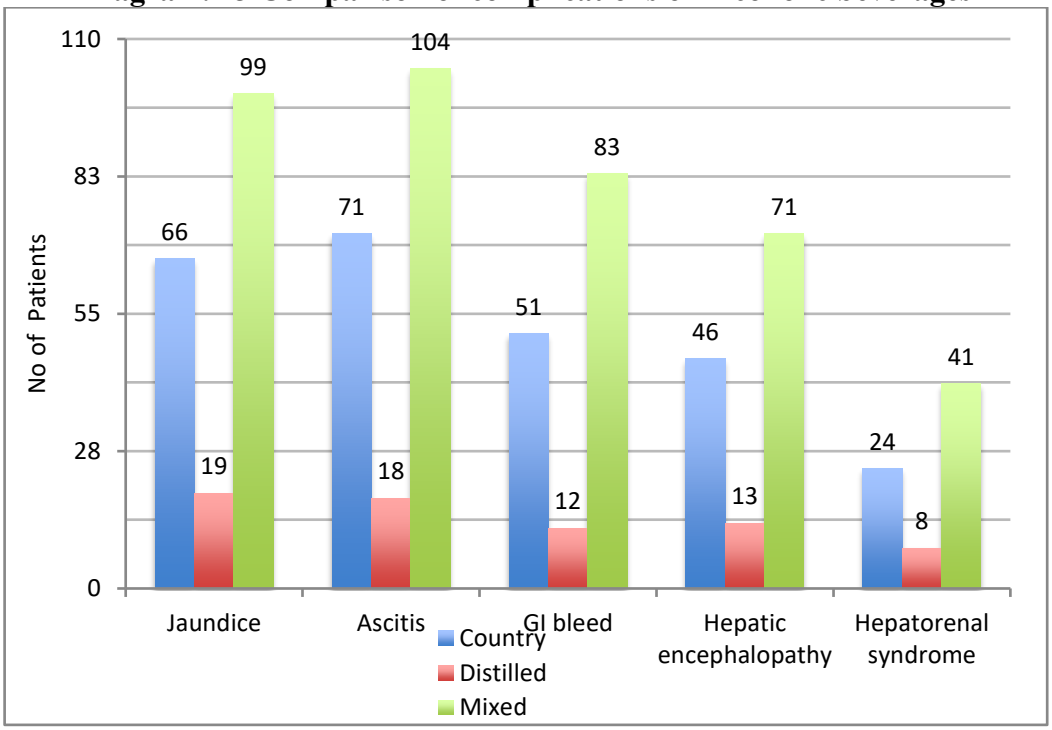
135 (48.04%) patients used to consume alcohol night and 146 (51.96%) patients used to consume alcohol both day and night.
16 (5.69%) patients used to consume alcohol 1-2 times a week, 84 (29.89%) patients used to consume alcohol 3-6 times a week, 181 (64.41%) patients used to consume alcohol every day.
87 (30.96%) patients started consuming alcohol < 20 years, 152 (54.09%) patients started consuming alcohol at 20 – 30 years, 42(14.95%) patients started consuming alcohol > 40 years.
91 (32.38%) patients were diagnosed for alcohol liver disease between 20-40 years, 127 (45.20%) patients were diagnosed for alcohol liver disease between 40-60 years,63(22.42%) patients were diagnosed for alcohol liver disease between 60-80 years.
175 (62.28%) patients consumed alcohol only single, 72 (25.62%) patients consumed alcohol only in a group, 34(12.10%) patients consumed alcohol in single and group.

44 (15.66%) patients had environmental as a cause of drinking, 196 (69.75%) patients had social reasons as a cause of drinking, 41(14.59%) patients had psychosocial as a cause of drinking.

Tab.8 Comparison of complications of Alcoholic beverages

Complications	Country (N=102)	Distilled (N=31)	Mixed (N=148)	P-value
Jaundice	66	19	99	0.067
Ascites	71	18	104	0.052
GI bleed	51	12	83	0.073
Hepatic encephalopathy	46	13	71	0.127
Hepatorenal syndrome	24	8	41	0.071

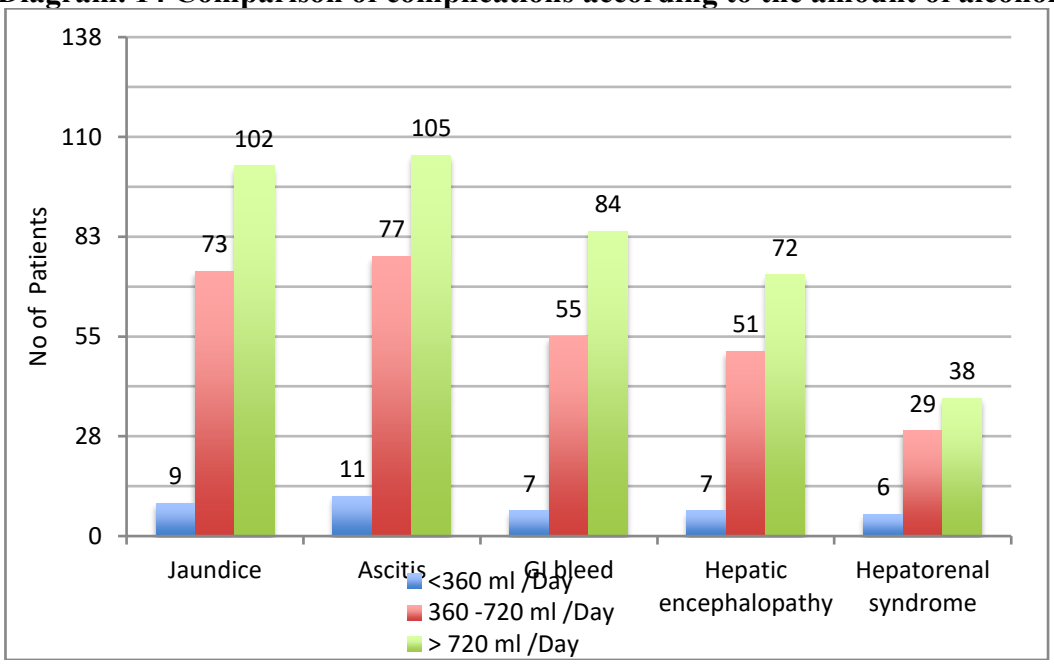
Diagram. 13 Comparison of complications of Alcoholic beverages



Tab.9 Comparison of complications according to the amount of alcohol

Complications	<360 ml /Day (N=19)	360 -720 ml /Day (N=117)	> 720 ml /Day (N=145)	P value
Jaundice	9	73	102	0.081
Ascites	11	77	105	0.172
GI bleed	7	55	84	0.051
Hepatic encephalopathy	7	51	72	0.073
Hepatorenal syndrome	6	29	38	**0.032**

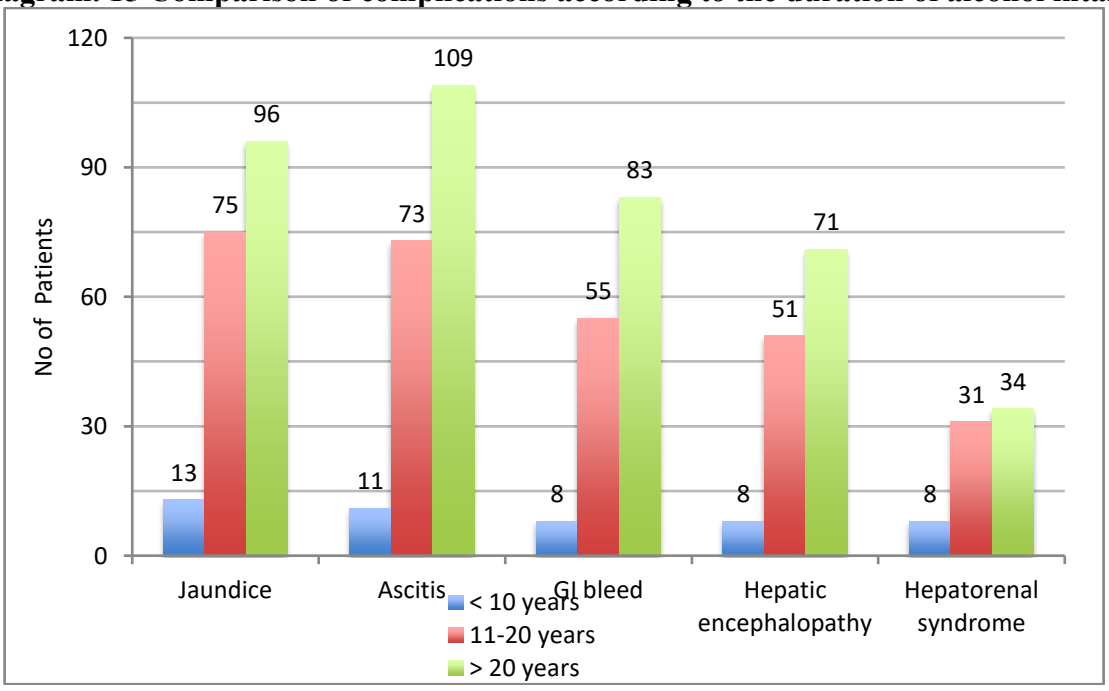
Diagram. 14 Comparison of complications according to the amount of alcohol



Tab.10 Comparison of complications according to the duration of alcohol intake

Complications	< 10 years (N=24)	11-20 years (N=115)	> 20 years (N=142)	P value
Jaundice	13	75	96	0.0619
Ascites	11	73	109	0.0532
GI bleed	8	55	83	0.0842
Hepatic encephalopathy	8	51	71	**0.0426**
Hepatorenal syndrome	8	31	34	0.0587

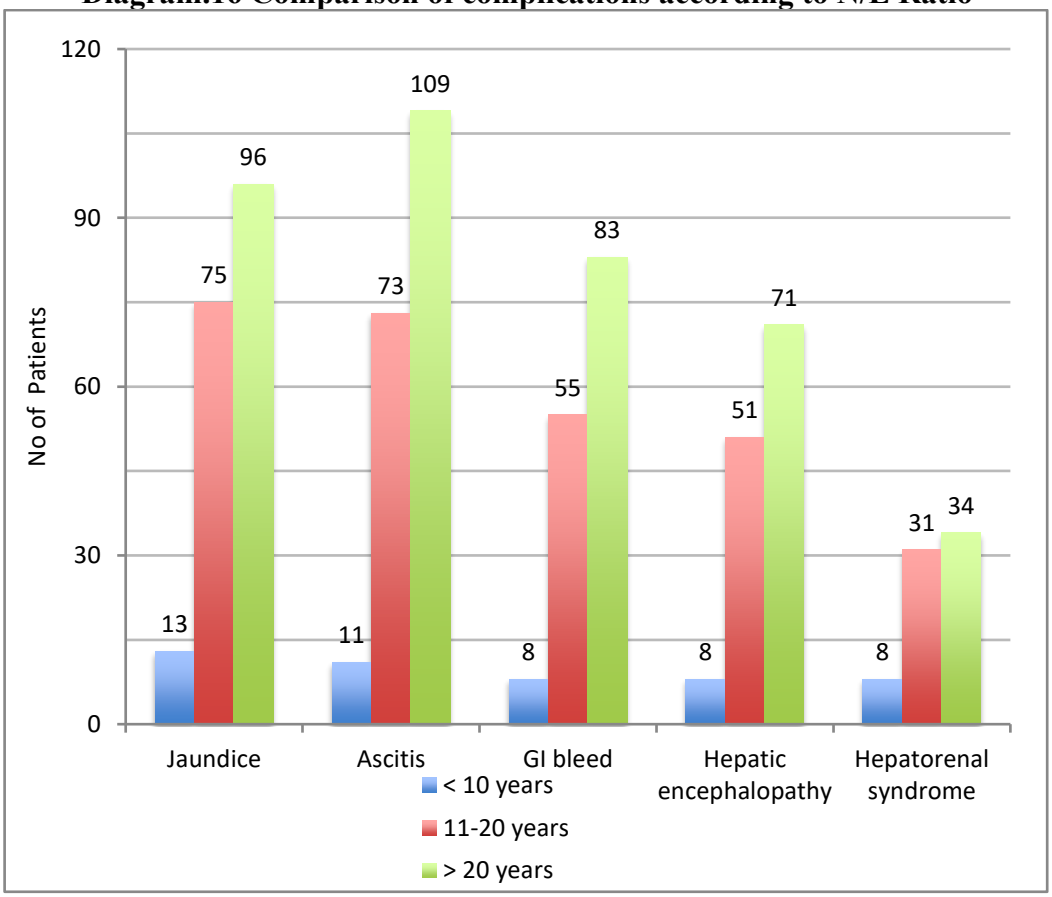
Diagram. 15 Comparison of complications according to the duration of alcohol intake



Tab.11 Comparison of complications according to N/L Ratio

Complications	< 2.5 (N=46)	2.5-4.5 (N=101)	> 4.5 (N=134)	P value
Jaundice	15	72	97	**0.0431**
Ascites	8	73	112	**0.0432**
GI bleed	6	55	85	**0.0321**
Hepatic encephalopathy	7	51	72	**0.0151**
Hepatorenal syndrome	7	31	35	**0.0132**

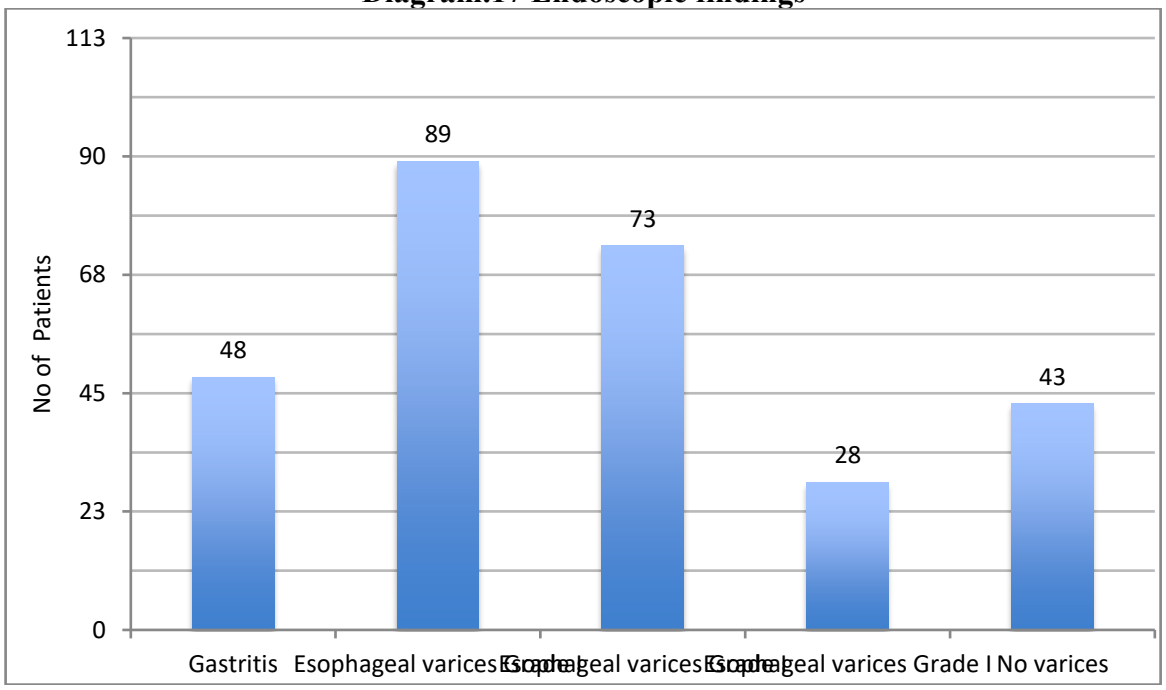
Diagram.16 Comparison of complications according to N/L Ratio



Tab.12 Endoscopic findings

Findings	No of patients	Percentage
Gastritis	48	17.08%
Esophageal varices Grade I	89	31.67%
Esophageal varices Grade II	73	25.98%
Esophageal varices Grade III	28	9.96%
No varices	43	15.30%

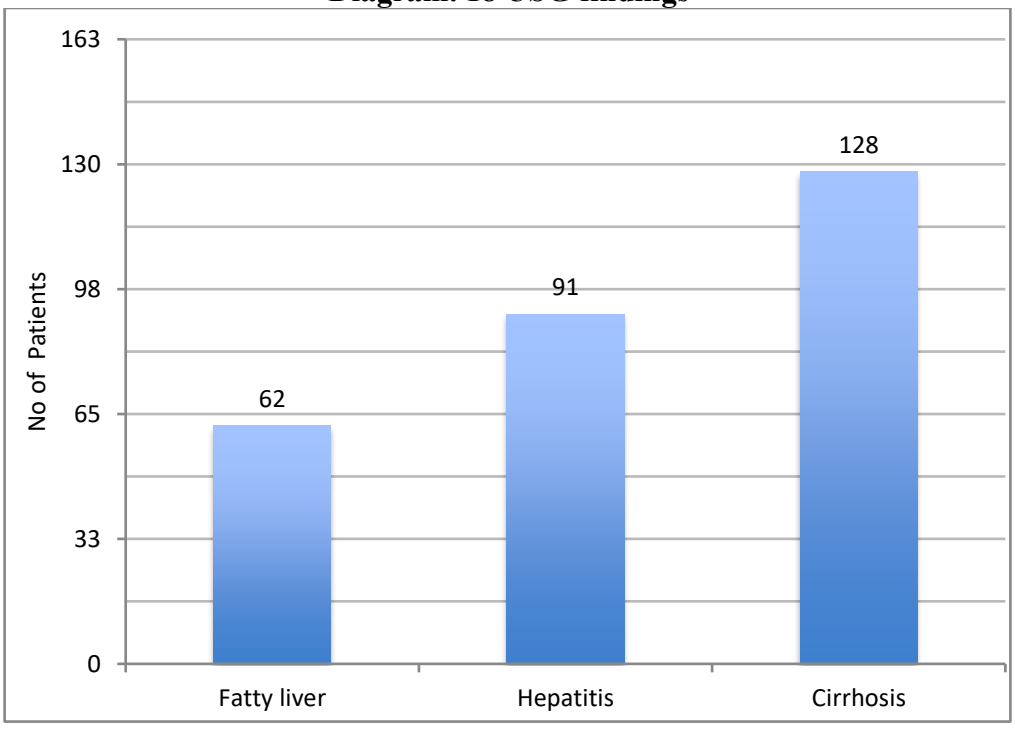
Diagram.17 Endoscopic findings



Tab.13 USG findings

Findings	No of patients	Percentage
Fatty liver	62	22.06%
Hepatitis	91	32.38%
Cirrhosis	128	45.55%

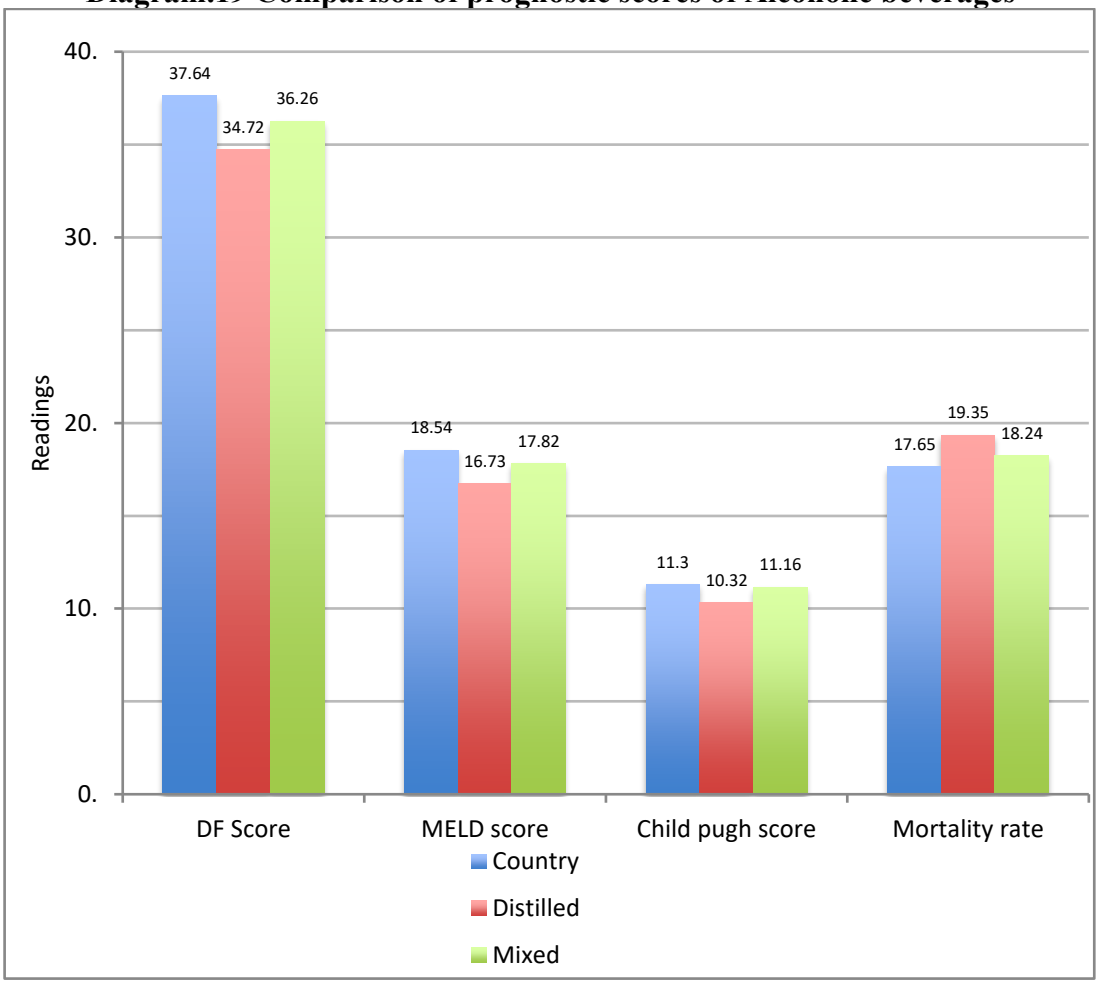
Diagram. 18 USG findings



Tab.14 Comparison of prognostic scores of Alcoholic beverages

Prognostic scores	Country (N=102)	Distilled (N=31)	Mixed (N=148)	P-value
DF Score	37.64	34.72	36.26	**0.042**
MELD score	18.54	16.73	17.82	**0.023**
Child pugh score	11.3	10.32	11.16	**0.046**
Mortality rate	17.65%	19.35%	18.24%	**0.032**

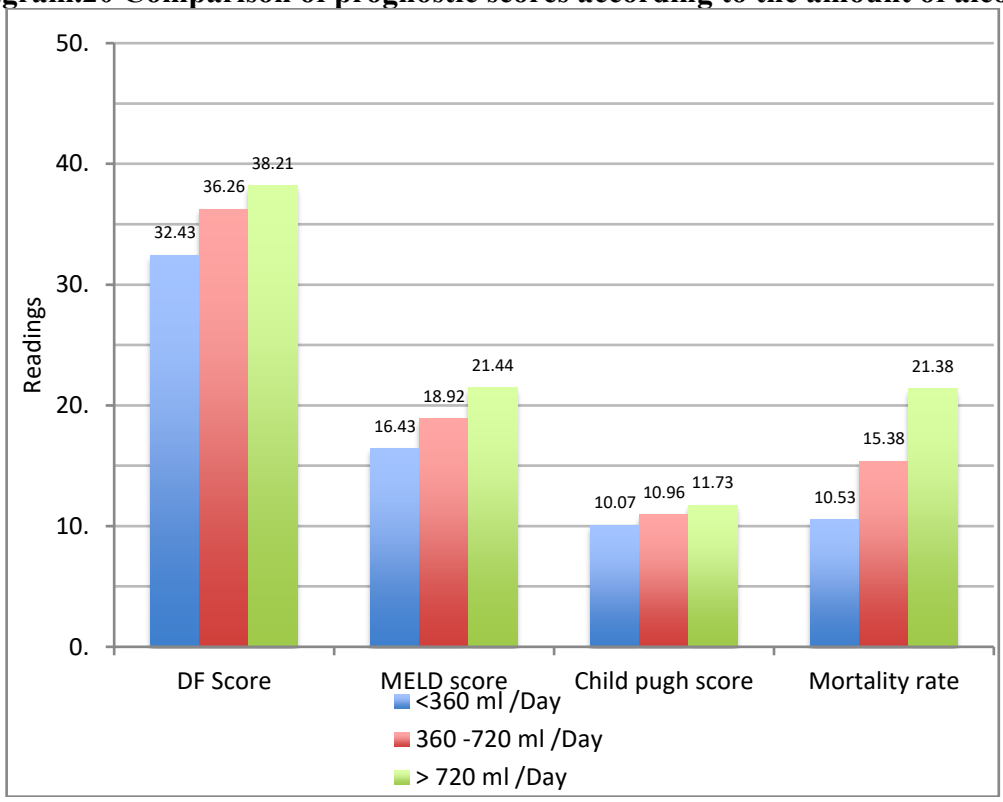
Diagram.19 Comparison of prognostic scores of Alcoholic beverages



Tab.15 Comparison of prognostic scores according to the amount of alcohol

Prognostic scores	<360 ml /Day (N=19)	360 -720 ml /Day (N=117)	> 720 ml /Day (N=145)	P value
DF Score	32.43	36.26	38.21	**0.048**
MELD score	16.43	18.92	21.44	**0.042**
Child pugh score	10.07	10.96	11.73	**0.049**
Mortality rate	10.53%	15.38%	21.38%	**0.031**

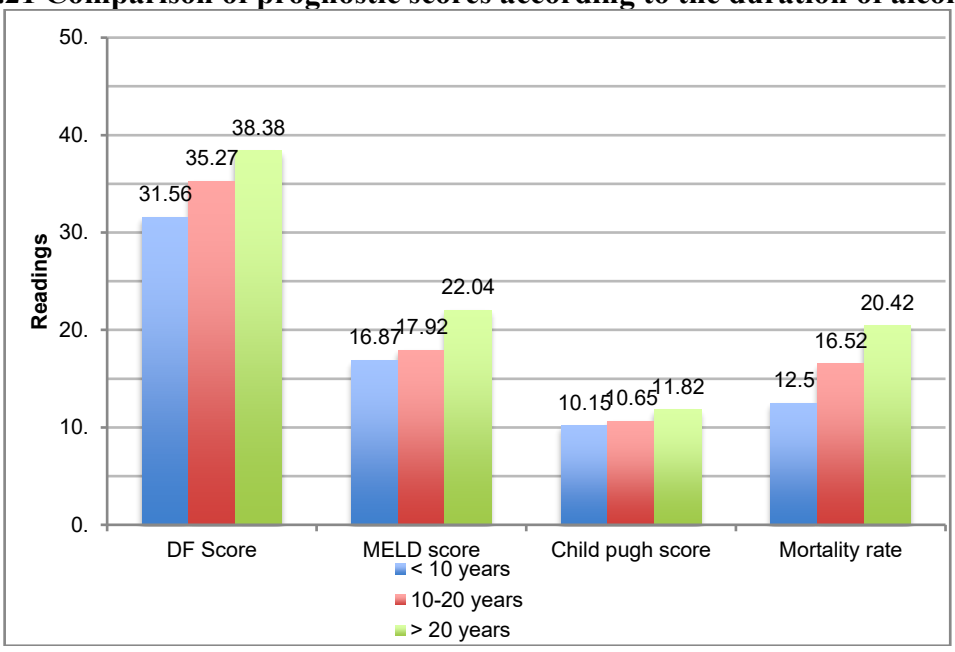
Diagram.20 Comparison of prognostic scores according to the amount of alcohol



Tab.16 Comparison of prognostic scores according to the duration of alcohol intake

Prognostic scores	< 10 years (N=24)	10-20 years (N=115)	> 20 years (N=142)	P value
DF Score	31.56	35.27	38.38	**0.033**
MELD score	16.87	17.92	22.04	**0.041**
Child pugh score	10.15	10.65	11.82	**0.032**
Mortality rate	12.50%	16.52%	20.42%	**0.027**

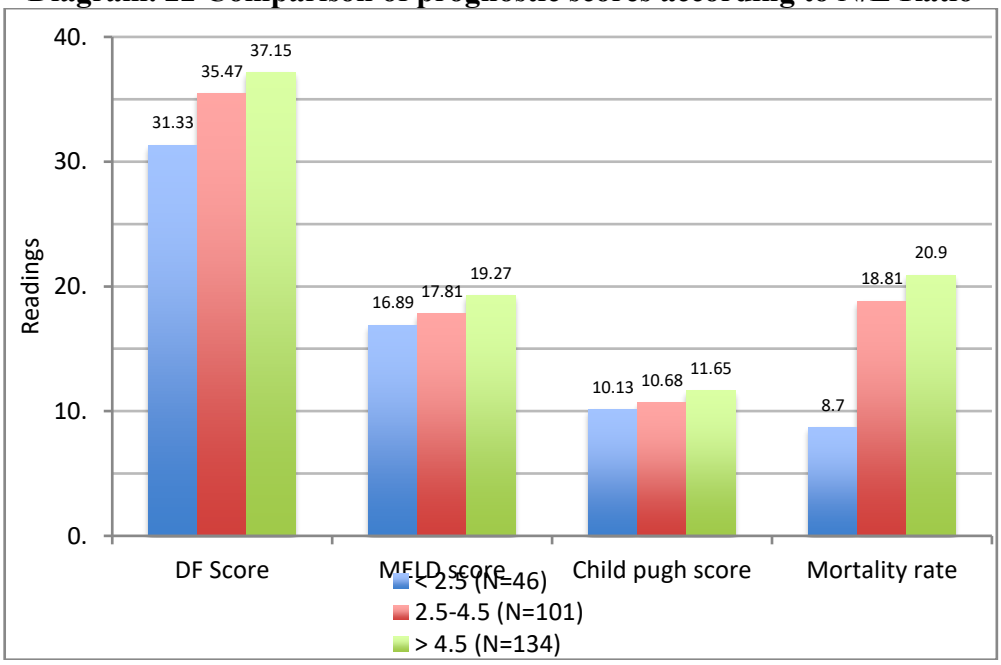
Diagram.21 Comparison of prognostic scores according to the duration of alcohol intake



Tab.17 Comparison of prognostic scores according to N/L Ratio

Prognostic scores	< 2.5 (N=46)	2.5-4.5 (N=101)	> 4.5 (N=134)	P value
DF Score	31.33	35.47	37.15	**0.021**
MELD score	16.89	17.81	19.27	**0.046**
Child pugh score	10.13	10.68	11.65	**0.037**
Mortality rate	8.70%	18.81%	20.90%	**0.011**

Diagram. 22 Comparison of prognostic scores according to N/L Ratio



DISCUSSION

Correlation between alcohol-related liver disease and the pattern of alcohol consumption is not well established. Missing link could be the intervening socio-demographic factors. Studies have demonstrated variable results with the pattern of alcohol consumed.

In-country like India, where social, cultural and economic factors determine the pattern of alcohol consumption, few studies may not suffice to understand the relation and hence warrants further research. Western data from research done on Caucasian population cannot be applied blindly to the Indian population because of the varied ethnicity, social, cultural and economic factors that determine the pattern of alcohol consumption.

However, very few studies have been carried out in Indians in general India in particular. Hence this study is planned to get a detailed profile of alcohol-related liver disease patients in the Indian population, including their alcohol consumption data, disease severity and outcome in **Socio-demographic pattern**

Age pattern

In our study, the mean age of alcoholics was 44.6±7.33 years. In a study by Nitya et al, the mean age of alcoholics was 46.2±9.86 years. In a study by **Nagamani et al**, mean age of Alcohol-related liver disease (ALD) patients was 45.4±8.68 years.[75] In a study by **Mitra et al**, mean age was 46.9±8.83 years.[82] In a study by **Vignesh et al**, mean age was 46.9±8.83 years.[100]

As the development of alcohol-related liver disease takes several years, most of the studies observed a mean age in the mid-decade between 40 – 50 years.

Another reason for the higher prevalence of patients in mid-40s could be that of a mid-life crisis caused by ageing itself, or ageing in combination with changes, problems, or regrets overwork. This would alter the drinking pattern in the mid-30s and subsequently may manifest in mid-40s with alcoholic liver disease.

Sex ratio

In our study, there were255 (90.75%) males and 26 (9.25%) females. In a study by Mitra et al, 78.3% of these patients were males and 21.69% females.[82]In a study by **Vigneshet al**, 98.93 % were males and 1.07 % were females. [100]

The overwhelming majority of males in studies of alcohol-related liver disease s can be explained by the socio-cultural factors that forbid females in many societies to consume liquor. As earning by a female is spent on essential needs of the family, the financial freedom to buy alcohol is relatively less compared to males.

Education

In our study, 239 (84.70 %) patients were literate and43 (15.30%) were illiterate. Among literates, 6 (2.14%) studied pre-primary, 64 (22.78%) studied primary, 52 (18.51%) studied upper primary, 67 (23.84%) studied secondary, studied senior secondary, 21 (7.47%) studied UG and 3 (1.07%) studied PG. Similarly, in a study by **MP Virukalpattigopalratnam**, 90 % were literate and 5 % were illiterate.[101]In a study by **Vignesh et al**, 62% studied primary, 15% studied secondary, 13% studied senior secondary, 6 % studied UG/PG.[100]

Observations of our study, as well as others, is contrary to popular belief that illiterates engage in drinking and end up with ALD. As observed in our study and similar other studies, education may have an only marginal effect against chronic alcoholism.

Occupation

In our study, 158 (56.23 %) were in skilled jobs, 89 (31.67%) were in unskilled jobs, 34 (12.10%) were unemployed. Similarly, in a study by **Vignesh et al**, 58 % were skilled workers and 41 % were unskilled workers. [100]In a study by **MP Virukalpattigopalratnam**, 89 % were employed and 11 % were unemployed.

Our study documented the popular belief that unemployed people engaged more in the consumption of alcohol. Other studies also support the fact that unemployment may not be a major cause of alcoholism. This may be because, employed people have a continuous cash flow to buy alcohol for a prolonged period of time, enough to cause ALD, in contrast to unemployed people.

Marriage

In our study, 268 (95.37%) were married and 13 (4.63%) were unmarried. Similarly, in a study by MP Virukalpattigopalratnam, 88 % were married and 12 % were unmarried. . In a study by **Vignesh et al**, 90 % were married and 9 & were single.

Again, observations by our study and others is in contrary to popular belief that bachelors/singles more frequently engage in chronic alcoholism.

Socio economic status

In our study, 89 (31.67%) belonged to the lower class, 102 (36.30%) belonged to the lower middle class, 35 (12.46%) belonged to the middle class, 33 (11.74%) belonged to the upper lower class, 22 (7.83%) belonged to upper-middle class. A trend of middle class dominating the study is visible.

Similar observations were noted by in a study by **MP Virukalpattigopalratnam**, a large proportion (75%) belonged to the middle and lower-middle economic strata and 25 % belonged to the poor class. In a study by **Vignesh et al,** 12 % were from the lower class, 41 % from the lower middle class and 46 % were from the middle class.

Studies before 2000, showed the predominance of the lower class in chronic alcoholism and ALD. However, the recent trend of middle class engaging in chronic alcoholism highlights the paradigm shift in per capita income and class migration.

Household location

In our study, 175 (62.28%) were rural, 51 (18.15%) were semi-urban and 55 (19.57%) were urban.In a study by **Vignesh et al,** 60 % were from Rural households and 38% were from urban households. The predominance of rural household needs to be considered in the context of hospital-based study wherein urban population prefers private health care and rural population prefers govt hospitals.

Religion

In our study, 269 (95.73%) were Hindus, 9 (3.20%) were Muslims, 3 (1.07%) were Christians.
While alcoholism is strictly prohibited among Muslims, alcohol is an integral part of cultural aspects among Christians. In Hindus, binge consumption of alcohol is seen during certain festivals and occasions of marriage ceremonies. However, our study does not show any out of range representation of any religion. This may also highlight the marginal effect religion has on controlling chronic alcoholism.

Alcoholic consumption pattern

Type of alcohol

In our study,102 (36.30%) patients consumed country-made alcohol, 31 (11.03%) patients consumed distilled alcohol and 148 (52.67%) consumed mixed.
Similar to our study, other studies also noted that the majority consumed country liquor. In a study by **Nagamani et al**, the majority of the patients have been consuming country made spirit (80%), other alcoholic beverages included whisky (11%) and beer (9%). In a study by **Nitya et al**, they observed a majority of them have been consuming country-made spirits (79%), other consuming branded spirits like whisky (6.5%) and variable drinkers (14%) while only 1 patient (0.5%) was consuming beer.[84]
Narawane et al studied 328 patients from a public hospital in Mumbai and found that liver disease was more common in those who consumed illicitly- brewed as compared to licit liquor.[102] In a study by **Mitra et al** 33.9% patients have been consuming only Hadiya (rice beer), 27.35% patients were consuming illicit Country Liquor, 22.64% patients have been consuming both Hadiya (rice beer) and country liquors and 16.03% patients were consuming only branded licit liquors.[82]
In a prospective cohort study conducted by **Askgaard G et al** on alcohol drinking pattern and development of cirrhosis on 55,917 participants of the Danish population, it was found that there was a lower risk of cirrhosis in wine drinking subjects compared to subjects who consume liquor and beer.[103]
Bellantoni S et al. in their cohort study on drinking habits as a cofactor of risk for alcohol-related liver injury, in 6917 subjects concluded that drinking multiple different alcoholic beverages increases the risk of developing an alcohol-related liver injury.[104]
Kerr WC et al compared beverage-specific alcohol consumption and mortality due to cirrhosis in a group of English specific beer-drinking countries. The results of the study point out spirit consumption rather than beer or wine is associated with a higher risk of cirrhosis mortality in primary beer-drinking countries.[105]
The predominance of country liquor can be because of selection bias, wherein majority were from rural households where availability of distilled liquor is a scarcity. Another reason is that country liquor is inexpensive compared to distilled.

Frequency of alcohol

In our study, 16 (5.69%) patients used to consume alcohol 1-2 times a week, 84 (29.89%) patients used to consume alcohol 3-6 times a week, 181 (64.41%) patients used to consume alcohol every day.

Narawane et al observed that 73 % engaged in drinking daily while Mitra et al observed daily dinking in 69 % of the sample.[102] In a study by **Ray et al**, 305 (96.8%) drank daily among those who died compared to only 25 (31.3%) who were alive ($p < 0.0001$). [88]In a study by **Vignesh et al**, 23 % drink night and 73 % drink throughout day and night.[100]
In a study by **Mitra et al**, comparing Discriminant factor in all the three groups showed a significant correlation between the amount of alcohol intake and development of ALD ($p=0.04$).[82]
Thus, daily drinking predominated our study and other studies that caused ALD.

Quantity of alcohol
In our study,19 (6.76 %) consumed < 360 ml /day, 117 (41.64 %) consumed 360 - 720 ml /day, 145 (51.60 %) consumed > 720 ml /day. In a study by **Mitra et al**, majority of them (78%) consumed more than 2 quarters a day.[82]Similar observations were also made by **Nawarane et al.**[102]
Becker et al in their large population-based prospective cohort study in Copenhagen also observed similar dose-dependent rise in the risk. The level of alcohol intake above which the relative-risk was significantly greater than 1 was observed at 7 to 13 beverages per week for women and 14 to 27 beverages per week for men.[106]**Savolainen et al** suggested that above a rather low level of alcohol consumption, the risk of development of cirrhosis was not further influenced by the amount of alcohol consumed.[107] Various studies have given different cut-off levels of safe drinking though it has been recognized that no level of alcohol amount is absolutely safe.

Duration of alcohol
In our study,24 (8.54%) patients consumed alcohol for < 10 years, 115 (40.93 %) patients consumed alcohol for 11-20 years, and 142 (50.53 %) patients consumed alcohol for > 20 years. The average duration of drinking was 19.24 years.
Similar observations were made by **Nitya et al**, where the average duration of alcohol intake was 17 years, which was not significantly different among various liquor groups.[84]**Narawane et al** observed that the average duration of drinking >14 years was significantly more common in those with liver disease. [102]
Observations by our study and others highlight the long duration of alcohol consumption that can cause ALD. Most of the studies observed an average of 10 – 20 years of alcohol intake.

Complications observed in ALD patients
In our study, 193 (68.68%) patients had ascites, 146 (51.96%) had GI bleed, 130 (46.26%) had Hepatic encephalopathy, followed by Hepatorenal syndrome in 73 (25.98%) patients.
In a study by **Nagamani et al**, complications observed in ALD patients were ascites (40%) followed by hepatic encephalopathy (22%), upper GI bleeding (18%), renal failure (15%) and spontaneous bacterial peritonitis (5%). Acute pancreatitis (38%) was seen in ALD patients whereas chronic pancreatitis (27%) was observed.
Nitya et al observed complications were like ascites (72%), hepatorenal syndrome (35%), hepatic encephalopathy (59%) and gastrointestinal bleeding (59%) .[84]
In a study by **Mitra et al**, the most complications observed among the patients were ascites (53.7%), followed by hepatic encephalopathy (20.7%), upper gastrointestinal bleed (18.86%), sub-acute bacterial peritonitis (3.7%) and hepatorenal syndrome (2.8%).[82]

Haematological and Biochemical findings
In our study, anemia was present in 243 (86.48%) of patients with a mean haemoglobin of 9.81±1.1 mg/dL. Leukocytosis was present in 112 (39.86%) patients. Liver function tests revealed elevated transaminases with mean AST/ALT ratio of 2.32±0.92. Hyperbilirubinemia was seen in 231 (82.21%) patients with a mean of 4.88±7.58 mg/dL. Mean albumin levels were 2.82±0.69 g/dl. Mean INR was found to be 2.2±0.71. Renal function was derranged in 16 (5.7%) patients.

In a study by **Nagamani et al**, Anaemia was common findings in 82% with mean Hb of 10.2±0.9 mg/dL, leukocytosis was seen in 43% of patients. Liver function tests showed elevated AST and ALT levels with mean AST/ALT ratio of 2.55±0.76. Hyperbilirubnemia was seen in 84% of patients with a mean range of 5.08±6.41 mg/dL. Mean albumin levels were found to be 3.1±0.34 g/dl. Mean INR was found to be 2.1±0.6.

Nitya et al, investigations showed anemia in 87% of patients and leukocytosis in 36% patients, differential counts showed polymorphonuclear predominance (as evidenced by mean N/L Ratio of 5.5±3.4). Liver function tests revealed elevated transaminases with mean AST/ALT ratio of 2.15±0.88. Hyperbilirubinemia was seen in 85% of patients (mean bilirubin 5.28±6.03 mg/dL). However, serum bilirubin >3 mg/dL was found in around 52% of patients. Mean albumin levels were 2.79±0.62 g/dL and severe hypoalbuminemia (<3 g/dL) was seen in 65%, and coagulopathy (mean INR 2.08±0.89) were also common biochemical findings.[84]

In a study by **Mitra et al**, Biochemical investigations showed Leukocytosis in 27% patients and anaemia in 68% patients. Hyperbilirubinemia was seen in 79% of patients with a mean total bilirubin level 6.2±2.5 mg/ dl and 70% of patients showed elevated liver enzymes with the mean AST/ALT ratio of 2.2±1.3. Mean albumin level was 2.11±0.9 g/ dl and mean INR was 2.7±0.7. Renal function was derranged in 2.8% patients.[82]

In a study by **Roy et al**, in those who died, SGOT/SGPT was >2 in all ACLF (3–4 times normal in 32) and 1.5–2 in 155 (85.2%) cirrhotic patients at presentation. SGPT was normal (<40 IU/mL) in 100 (75.2%) ACLF and 132 (72.5%) cirrhotic patients. Bilirubin was elevated in all patients (mean 10.41 ± 5.12 [5.1–24.6] mg/dL) and in 147 (80.2%) cirrhotic patients (mean 3.09 ± 1.13 [1.2–4.8] mg/dL). In those who survived, SGOT/SGPT was 1–1.3 in 68 (85%, <1 in the remaining 15%) but SGPT was normal in only 10 (12.5%) and bilirubin was elevated in 20 (25%) (Overall 0.8–3.5 mg/dL) with less deranged INR (<1.2).[108]

Prognostic markers of ALD

Type of beverages and correlation with prognostic markers

In our study, DF score, MELD score, Child-Pugh score and mortality differed significantly among consumers of different types of alcohol with the least values in distilled alcohol. P values for DF score, MELD score, Child-Pugh score and mortality were 0.042, 0.023,0.046 and 0.032 respectively.

Nagamani et al made a similar observation**,** where prognostic outcome measures correlated well with the type of alcoholic beverage consumed. For country-made alcohol, Maddrey DF score correlation was found to be 0.039 ($p< 0.05$), MELD score was significantly correlated with p-value of 0.017 ($p< 0.05$) and child-pugh score correlation was found to be 0.019 ($p< 0.05$).[75]

Our study and study by **Nagamani et al** highlights the type of alcoholic beverages and its impact of ALD and its prognosis. Studies point towards worst prognosis with country liquor than distilled one.

In contrast to observations of our study, **Nitya et al** found no significant difference was found between the disease severity in country liquor consumers and branded whisky consumers. Incidence of complications, various prognostic markers and mortality rate were similar in country liquor and branded liquor consumers. [84]

Duration, the quantity of alcohol and correlation with prognostic markers

In our study, DF score, MELD score, Child-Pugh score and mortality differed significantly according to the duration of alcohol. Duration of alcohol > 20 years had the worst prognostication values. In correlation of quantity of alcohol with prognostic markers, DF score, MELD score, Child-Pugh score and mortality did not differ significantly with the amount of alcohol. However, the Worst value was observed with alcohol intake of > 720 ml/day.

In a study by **Mitra et al**, DF correlated significantly amount and duration of alcohol consumption. However, MELD Score and Child-Pugh Score with p-value being 0.2 and 0.23 respectively did not correlate with the amount and duration of alcohol consumption.[82]

Nitya et al the duration of alcoholism also didn't affect the incidence of complications and prognostic

markers except for Child-Pugh score.[84]
While **Nitya et al** claims 74% of patients had been drinking for ≤20 years[84], Patek et al had found in their study that only 34% of cirrhotic patients had been drinking for <20 years[41]. **Narawane et al** observed that 86% of the cirrhotic patients were drinking for <20 years.[102]These studies give a fillip to the hypothesis that Indians develop liver disease in lesser duration than the Western population.
However, a notable difference between all the studies is the lack of standardization in the nomenclature of types of alcohol. Hence, unless studies compare similar types of alcohol with varied duration and quantity of intake, a conclusion can not be drawn.

Neutrophil –Lymphocyte Ratio (NLR) and prognostic markers
In our study, NLR correlated significantly with DF score, MELD score, Child-Pugh score and mortality with a p-value of 0.021, 0.046, 0.037 and 0.011 respectively. However, none of the correlation was extremely significant.
Nitya et al observed NLR correlated well with all three prognostic scores with a p-value of 0.164 ($p < 0.05$) with Maddrey DF score, 0.279 ($p < 0.001$) with MELD score and 0.169 ($p < 0.05$) with Child-Pugh score. Thus the correlation was significant with all three prognostic markers but it was extremely significant with MELD score.[84]
As NLR correlates well with different prognostic markers in ALD, it has the potential to be an independent and inexpensive prognostic marker of ALD.
Mortality in ALD
In our study, the overall mortality was 18.15 %. Patients with Hepatorenal syndrome and hepatic encephalopathy had a higher in-hospital mortality rate of 36.99 % and 32.31 % respectively. However, mortality rates were not seen to be higher in ascites (14.51%) and gastrointestinal bleed patients (15.07 %). However, this also can be due to our biased selection of patients (massively bleeding patients were excluded due to hemodynamic instability).
Similar observations were also made in studies by **Nitya et al** and **Nagamani et al**. [84,75]**Nitya et al** observed a linear correlation between mental status, serum creatinine with mortality implying high mortality among Patients with Hepatorenal syndrome and hepatic encephalopathy.[84]
Our study also observed higher mortality with country alcohol and duration > 20 years. While the study by **Nagamani et al** concurred with similar finding, the study by **Nitya et** al did not observe any difference. [75,84]

CONCLUSION
Male genders, middle age, rural household, the lower and middle class were predominantly observed with Alcohol-related liver disease (ALD). Literary status, marital status, unemployment, religion, ethnicity were not associated significantly with ALD.
Majority of patients consumed country liquor, drank day and night, and more than a quarter of alcohol per day, in-group and for > 10 years.
The commonest complication was ascites followed by GI bleed, Hepatic encephalopathy and hepatorenal syndrome.
There was significant correlation DF scores, MELD score, Child-Pugh score with the type of alcoholic beverage and duration of alcohol consumed. The country made alcoholic beverage and duration > 20 years showed a bad prognosis.
NLR concurred with prognostic markers and hence can be used as a surrogate prognostic marker in ALD.
Mortality was 18.15 % with Hepatorenal syndrome and hepatic encephalopathy registering higher in-hospital mortality than other complications.

SUMMARY
- Aim of the study was to study the profile of alcohol-related liver disease among patients attending tertiary care hospital inIndia.

- Data was collected from 1/04/2019 to 31/03/2020 in standard proforma and tabulated. Raw data was analysed with statistical tools.
- In our study, there were255 (90.75%) males and 26 (9.25%) females. There were 113 (40.21%) patients within 20-40 years, 92 (32.74%) patient within 40-60 years, 76 (27.05%) patients within 60-80 years. 268 (95.37%) were married and 13 (4.63%) were unmarried.
- 175 (62.28%) were rural, 51 (18.15%) were semi urban and 55 (19.57%) were urban.
- 4 (1.42%) were professionals, 29 (10.32%) were semiprofessionals, 58 (20.64%) were in Clerical/shop/farm jobs, 67 (23.84%) were in semi-skilled jobs, 89 (31.67%) were in unskilled jobs, 34 (12.10%) were unemployed.
- 43 (15.30%) were illiterates, 6 (2.14%) studied preprimary, 64 (22.78%) studied primary, 52 (18.51%) studied upper primary, 67 (23.84%) studied secondary, studied senior secondary, 21 (7.47%) studied UG and 3 (1.07%) studied PG.
- 89 (31.67%) belonged to the lower class, 102 (36.30%) belonged to the lower middle class, 35 (12.46%) belonged to the middle class, 33 (11.74%) belonged to the upper lower class, 22 (7.83%) belonged to upper-middle class.
- 269 (95.73%) were Hindus, 9 (3.20%) were Muslims, 3 (1.07%) were Christians.
- 135 (48.04%) patients used to consume alcohol night and 146 (51.96%) patients used to consume alcohol both day and night.16 (5.69%) patients used to consume alcohol 1-2 times a week, 84 (29.89%) patients used to consume alcohol 3-6 times a week, 181 (64.41%) patients used to consume alcohol every day.
- 87 (30.96%) patients started consuming alcohol < 20 years, 152 (54.09%) patients started consuming alcohol at 20 – 30 years, 42 (14.95%) patients started consuming alcohol > 40 years.91 (32.38%) patients were diagnosed for alcohol liver disease between 20-40 years, 127 (45.20%) patients were diagnosed for alcohol liver disease between 40-60 years, 63 (22.42%) patients were diagnosed for alcohol liver disease between 60-80 years.
- 175 (62.28%) patients consumed alcohol only single, 72 (25.62%) patients consumed alcohol only in a group, 34 (12.10%) patients consumed alcohol in single and group.44 (15.66%) patients had environmental as a cause of drinking, 196 (69.75%) patients had social reasons as a cause of drinking, 41 (14.59%) patients had psychosocial as a cause of drinking.
- The commonest complication in the study was ascites followed by GI bleed, Hepatic encephalopathy and hepatorenal syndrome. There was significant correlation DF scores, MELD score, Child-Pugh score with the type of alcoholic beverage and duration of alcohol consumed. The country made alcoholic beverage and duration > 20 years showed a bad prognosis. NLR concurred with prognostic markers and hence can be used as a surrogate prognostic marker in ALD. Mortality was 18.15 % with Hepatorenal syndrome and hepatic encephalopathy registering higher in-hospital mortality than other complications.

LIMITATIONS

- The alcoholic content of local and indigenous liquor was not measured and instead of standard concentration was considered.
- Our study did not consider liver biopsy and histopathological diagnosis of alcoholic liver disease was not done.
- As it was a cross-sectional study follow up of patients was out of the purview of the study.

REFERENCES

1. Pal HR, Jena R, Yadav D. Validation of the alcohol use disorders identification test (AUDIT) in urban community outreach and de-addiction centre samples in north India. J Stud Alcohol. 2004;65:794–800.
2. Sample Registration System Baseline Survey 2014. [Last cited on 2017 Aug 10].

3. World Health Organization (WHO). India. Alcohol Consumption levels and patterns. [Last cited on 2017 Jul 29]
4. World Health Organization. Global Health Observatory data repository. [Last cited on 2017 Aug 15].
5. Nayak MB, Bond JC, Cherpitel C. Detecting alcohol-related problems in developing countries: a comparison of 2 screening measures in India. Alcohol Clin Exp Res. 2009;33:2057–66.
6. Rajeev A, Abraham SB, Reddy TG, Skariah CM, Indiradevi ER, Abraham J. A community study of alcohol consumption in a rural area of South India. Int J Community Med Public Health. 2017;4:2172–7.
7. Levy RE, Catana AM, Durbin-Johnson B, et al. Ethnic differences in presentation and severity of the alcohol-related liver disease. Alcoholism: Clinical and Experimental Research. 2015;39(3):566–574.
8. Abdel-Misih SR, Bloomston M. Liver anatomy. Surg Clin North Am. 2010;90(4):643-653.
9. Sibulesky L. Normal liver anatomy. Clin Liver Dis (Hoboken). 2013;2(Suppl 1): S1-S3. Published 2013 Mar 29.
10. Zhao R, Duncan SA. Embryonic development of the liver. Hepatology. 2005 May;41(5):956-67.
11. Gerber MA, Thung SN. Histology of the liver. Am J Surg Pathol. 1987 Sep;11(9):709-22.
12. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Alcohol Drinking. Lyon (FR): International Agency for Research on Cancer; 1988. (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, No. 44.) 3, Chemical Composition of Alcoholic Beverages, Additives and Contaminants.
13. Lucey MR. Liver transplantation for alcohol-related liver disease. Nature Reviews. Gastroenterology &Hepatology. 2014;11(5):300–307.
14. Masson S, Emmerson I, Henderson E, et al. Clinical but not histological factors predict long-term prognosis in patients with histologically advanced non-decompensated alcohol-related liver disease. Liver International. 2014;34(2):235–242.
15. Meier P, Seitz HK. Age, alcohol metabolism and liver disease. Current Opinion in Clinical Nutrition and Metabolic Care. 2008;11(1):21–26.
16. Mohammad MK, Zhou Z, Cave M, et al. Zinc and liver disease. Nutrition in Clinical Practice. 2012;27(1):8–20.
17. Zakhari S. Bermuda Triangle for the liver: Alcohol, obesity, and viral hepatitis. Journal of Gastroenterology and Hepatology. 2013;28(Suppl 1):18–25.
18. Kakunje A, Kanaradi H, Pai G, Karkal R, Nafisa D, Chandrasekaran P. Drinking pattern in persons with alcohol dependence with and without cirrhosis: A hospital-based comparative study. Indian J Psychiatry. 2018;60(2):189-194.
19. Smith S, White J, Nelson C, Davies M, Lavers J, Sheron N, et al. Severe alcohol-induced liver disease and the alcohol dependence syndrome. Alcohol Alcohol. 2006;41:274–7.
20. Stickel F, Hampe J. Genetic determinants of alcohol-related liver disease. Gut. 2012;61(1):150–159.
21. Suraweera DB, Weeratunga AN, Hu RW, et al. Alcoholic hepatitis: The pivotal role of Kupffer cells. World Journal of Gastrointestinal Pathophysiology. 2015;6(4):90–98.
22. Patrick ME, Cronce JM, Fairlie AM, Atkins DC, Lee CM. Day-to-day variations in high-intensity drinking, expectancies, and positive and negative alcohol-related consequences. Addict Behav. 2016 Jul; 58():110-6.
23. Kamran U, Towey J, Khanna A, Chauhan A, Rajoriya N, Holt A. Nutrition in alcohol-related liver disease: Physiopathology and management. World J Gastroenterol. 2020;26(22):2916-2930.
24. Szabo G, Aloman C, Polyak SJ, et al. Hepatitis C infection and alcohol use: A dangerous mix for the liver and antiviral immunity. Alcoholism: Clinical and Experimental Research. 2006; 30(4):709–719.

25. Thomes PG, Osna NA, Davis JS, Donohue TM., Jr Cellular steatosis in ethanol oxidizing-HepG2 cells is partially controlled by the transcription factor, early growth response-1. International Journal of Biochemistry & Cell Biology. 2013;45(2):454–463.
26. Nagy LE, Ding WX, Cresci G, et al. Linking pathogenic mechanisms of alcohol-related liver disease with clinical phenotypes. Gastroenterology. 2016;150(8):1756–1768.
27. Osna NA, White RL, Krutik VM, et al. Proteasome activation by hepatitis C core protein is reversed by ethanol-induced oxidative stress. Gastroenterology. 2008;134(7):2144–2152.
28. Pavlov CS, Casazza G, Semenistaia M, et al. Ultrasonography for diagnosis of alcoholic cirrhosis in people with alcohol-related liver disease. Cochrane Database of Systematic Reviews. 2016; 3:CD011602.
29. Rathod SD, Nadkarni A, Bhana A, Shidhaye R. Epidemiological features of alcohol use in rural India: a population-based cross-sectional study. Br Med J. 2015;5:1–8. 22. Howard D, Nancy RC, Julie EB, Joann E, Manson, Michael G. Alcohol consumption and the risk of hypertension in women and men. Hypertension. 2008;51:1080–87.
30. Kanwar P, Sharma RC, Sharma DD, Kanwar N. A study of psychiatric co-morbidity among alcohol dependents. Int J Res Med Sci. 2017;5:4490–6.
31. Addolorato G, Mirijello A, Barrio P, Gual A. Treatment of alcohol use disorders in patients with alcohol-related liver disease. Journal of Hepatology. 2016;65(3):618–630.
32. Singal AK, Bashar H, Anand BS, et al. Outcomes after liver transplantation for alcoholic hepatitis are similar to alcoholic cirrhosis: Exploratory analysis from the UNOS database. Hepatology . 2012;55(5):1398–1405.
33. Ambade A, Mandrekar P. Oxidative stress and inflammation: Essential partners in alcohol-related liver disease. International Journal of Hepatology. 2012;2012;853175
34. Anstee QM, Seth D, Day CP. Genetic factors that affect risk of alcoholic and nonalcoholic fatty liver disease. Gastroenterology. 2016;150(8):1728–1744.e7.
35. Burza MA, Molinaro A, Attilia ML, et al. PNPLA3 I148M (rs738409) genetic variant and age at onset of at-risk alcohol consumption are independent risk factors for alcoholic cirrhosis. Liver International. 2014;34(4):514–520.
36. Shearn CT, Fritz KS, Shearn AH, et al. Deletion of GSTA4-4 results in increased mitochondrial post-translational modification of proteins by reactive aldehydes following chronic ethanol consumption in mice. Redox Biology. 2016;7:68–77.
37. Donnadieu-Rigole H, Olive L, Nalpas B, et al. Follow-up of alcohol consumption after liver transplantation: Interest of an addiction team? Alcoholism: Clinical and Experimental Research. 2017;41(1):165–170.
38. Singal AK, Chaha KS, Rasheed K, Anand BS. Liver transplantation in alcohol-related liver disease current status and controversies. World Journal of Gastroenterology. 2013;19(36):5953–5963.
39. Siu L, Foont J, Wands JR. Hepatitis C virus and alcohol. Seminars in Liver Disease. 2009 ;29(2):188–199.
40. Dong X, Liu H, Chen F, et al. MiR-214 promotes the alcohol-induced oxidative stress via down-regulation of glutathione reductase and cytochrome P450 in liver cells. Alcoholism: Clinical and Experimental Research. 2014;38(1):68–77.
41. Patek, Donohue TM., Jr Alcohol-induced steatosis in liver cells. World Journal of Gastroenterology. 2007;13(37):4974–4978.
42. European Association for the Study of the Liver. EASL clinical practical guidelines: Management of alcohol-related liver disease. Journal of Hepatology. 2012;57(2):399–420.
43. Frazier TH, Stocker AM, Kershner NA, et al. Treatment of alcohol-related liver disease. Therapeutic Advances in Gastroenterology. 2011;4(1):63–81
44. Fujita T, Soontrapa K, Ito Y, et al. Hepatic stellate cells relay inflammation signalling from sinusoids to parenchyma in mouse models of immune-mediated hepatitis. Hepatology . 2016;63(4):1325–1339.

45. Ganesan M, Natarajan SK, Zhang J, et al. Role of apoptotic hepatocytes in HCV dissemination: Regulation by acetaldehyde. American Journal of Physiology: Gastrointestinal and Liver Physiology. 2016;310(11): G930–G940.
46. Ganesan M, Zhang J, Bronich T, et al. Acetaldehyde accelerates HCV-induced impairment of innate immunity by suppressing methylation reactions in liver cells. American Journal of Physiology: Gastrointestinal and Liver Physiology. 2015;309(7): G566–G577.
47. Gitto S, Vitale G, Villa E, Andreone P. Update on alcohol and viral hepatitis. Journal of Clinical and Translational Hepatology. 2014;2(4):228–233.
48. Kang L, Chen X, Sebastian BM, et al. Chronic ethanol and triglyceride turnover in white adipose tissue in rats: Inhibition of the anti-lipolytic action of insulin after chronic ethanol contributes to increased triglyceride degradation. Journal of Biological Chemistry. 2007;282(39):28465–28473.
49. Zakhari S, Li TK. Determinants of alcohol use and abuse: Impact of quantity and frequency patterns on liver disease. Hepatology. 2007;46(6):2032–2039.
50. Kharbanda KK, Todero SL, Ward BW, et al. Betaine administration corrects ethanol-induced defective VLDL secretion. Molecular and Cellular Biochemistry. 2009;327(1–2):75–78.
51. Kirpich IA, Miller ME, Cave MC, et al. Alcohol-related liver disease: Update on the role of dietary fat. Biomolecules. 2016;6(1):1
52. Pati S, Swain S, Mahapatra S, Hussain MA, Pati S. Prevalence, pattern, and correlates of alcohol misuse among male patients attending rural primary care in India. J Pharm BioallSci 2017;9:66-72
53. Wang ZG, Dou XB, Zhou ZX, Song ZY. Adipose tissue-liver axis in alcohol-related liver disease. World Journal of Gastrointestinal Pathophysiology. 2016;7(1):17–26
54. Kim MS, Ong M, Qu X. Optimal management for alcohol-related liver disease: Conventional medications, natural therapy or combination? World Journal of Gastroenterology. 2016;22(1):8–23.
55. Seronello S, Montanez J, Presleigh K, et al. Ethanol and reactive species increase basal sequence heterogeneity of hepatitis C virus and produce variants with reduced susceptibility to antivirals. PLoS One. 2011;6(11):e27436.
56. Sofair AN, Barry V, Manos MM, et al. The epidemiology and clinical characteristics of patients with newly diagnosed alcohol-related liver disease: Results from population-based surveillance. Journal of Clinical Gastroenterology. 2010;44(4):301–307.
57. Girish N, Kavita R, Gururaj G, Benegal V. Alcohol use and implications for public health: Patterns of use in four communities. Indian J Community Med. 2010;35:238.
58. Ghosh S, Samanta A, Mukherjee S. Patterns of alcohol consumption among male adults at a slum in Kolkata, India. J Health Popul Nutr. 2012;30:73.
59. Kumar SG, Premarajan KC, Subitha L, Suguna E, Vinayagamoorthy, Kumar V. Prevalence and pattern of alcohol consumption using alcohol use disorders identification test (AUDIT) in rural Tamil Nadu. J ClinDiagn Res. 2013;7:1637–9.
60. Lakshmi A, Daisy D, Duttagupta, Lendi J, Padmanaban Drinking Habits, Health, Social and Behavioural Aspects of Alcohol Users in A Semi-Urban Population In Chennai. IOSR-JDMS. 2014;13:20–22.
61. Dewan G, Chowdhury FR. Alcohol use and alcohol use disorders in Bangladesh. Asia Pac J Med Toxicol. 2015;4:83–90.
62. Vidhukumar K, Nazeer E, Anil P. Prevalence and pattern of alcohol use in Kerala – A district-based survey. IJRSTAT. 2016;18:363–7.
63. Ramanan VV, Singh SK. A study on alcohol use and its related health and social problems in rural Puducherry, India. J Family Med Prim Care. 2016;5:804.
64. Manimunda SP, Sugunan AP, Thennarasu K, Pandian D, Pesala KS, Benegal V. Alcohol consumption, hazardous drinking, and alcohol dependency among the population of Andaman and Nicobar Islands, India. Indian J Public Health. 2017;61:105.

65. Bute J, Kori S, Arora VK, Sahasrabuddhe A. Prevalence and pattern of alcohol consumption among males attending primary health care setting using AUDIT in rural Indore, Madhya Pradesh, India. Int J Community Med Public Health. 2018;5:4461–9.
66. Eashwar VM, Gopalakrishnan S, Umadevi R, Geetha A. Epidemiology of alcohol consumption in an urban area of Kancheepuram district, Tamil Nadu. J Family Med Prim Care. 2019;8:1098–105.
67. Teli SA, Joshi Y. Prevalence of alcohol consumption in an urban population of Dehradun. Asian J Pharm Res. 2019;9:87–9.
68. Wangdi K, Jamtsho T. Prevalence and correlates of current alcohol use among Bhutanese adults: A nationally representative survey data analysis. Indian J Psychol Med. 2019;41:38
69. Gururaj G, Isaac MK. Psychiatric Epidemiology in India: Moving Beyond Numbers An Indian Perspective. New Delhi: Elsevier Publications; 1993. p. 37.
70. Bonu S, Rani M, Peters DH, Jha P, Nguyem SN. Does use of tobacco or alcohol contribute to impoverishment from hospitalisation costs in India? Health Policy Plan. 2005;20:41–9.
71. Benegal V, Velayudan A, Jain S. The social costs of alcoholism (Karnataka) NIMHANS J. 2000;18:67–76.
72. Wei X, Shi X, Zhong W, et al. Chronic alcohol exposure disturbs lipid homeostasis at the adipose tissue-liver axis in mice: Analysis of triacylglycerols using high-resolution mass spectrometry in combination with in vivo metabolite deuterium labeling. PLoS One. 2013;8(2):e55382.
73. Thursz M, Forrest E, Roderick P, et al. The clinical effectiveness and cost-effectiveness of Steroids Or Pentoxifylline for Alcoholic Hepatitis (STOPAH): A 2 × 2 factorial rando mised controlled trial. Health Technology Assessment. 2015;19(102):1–104.
74. Pathak OK, Paudel R, Panta OB, Pant HP, Giri BR, Adhikari B. Retrospective study of the clinical profile and prognostic indicators in patients of alcohol-related liver disease admitted to a tertiary care teaching hospital in Western Nepal. Saudi J Gastroenterol. 2009;15(3):171-175.
75. Nagamani R, Sakuntala. Clinical profile of alcohol-related liver disease patients in a tertiary care teaching hospital and its correlation with type of alcoholic beverage consumption. International Journal of Medical and Health Research Volume 2; Issue 6; June 2016; Page No. 12-14
76. Sarin SK, Malhotra V, Nayyar A, Sundaram KR, Broor SL. Profile of alcohol-related liver disease in an Indian hospital. A prospective analysis. Liver. 1988;8(3):132-137.
77. Khatroch, Shamsunder. Study of clinical and biochemical profile of acute alcohol-related liver disease in a teaching hospital in Telangana. International Journal of Advances in Medicine, [S.l.], v. 5, n. 4, p. 804-808, July 2018.
78. Phuntshochoden, Yadvendra singh Sirohi, Praveen kumarsharma, V k sashindaran, a study of clinical profile of alcohol-related liver disease and its relation to alcohol dependence syndrome, Indian journal of applied research: volume-8 | issue-6 | June-2018
79. Rishi, Jain AK, Nandy P. Study of clinical, laboratory, and upper gastrointestinal endoscopic profile in patients of alcohol-related liver disease in Sikkim. Med J DY Patil Vidyapeeth 2018;11:228-31
80. Sheshrao S Chavan, Siddheshvar V Birajdar, Nikhil R Pawar. Clinical profile of patients with alcohol use disorder. IAIM, 2018; 5(2): 48-56.
81. Ravindra K Hadakshi, DGaurav Modi. International Journal of Medical Science and Education Int. j.med. sci.Educ. July-September 2018; 5(3):364-368
82. Jeewan Kumar Mitra, PraneetAnsalMundu, Bindey Kumar, Rajendra Kumar Satapathy, Rashmi Sinha, Manish Kumar. Profile of alcohol-related liver disease in the population of Jharkhand: an insight into the realm of alcoholism from profligacy to burden. International Journal of Contemporary Medical Research 2017;4(3):770- 773.
83. Jawgam Umbon, Kalpana Niranjan. A study of clinical profile and prognostic indicators of alcohol-related liver disease in a tertiary care centre of northeast India. Journal of evidence-based medicine.4175-4180

84. NityaNand, Parveen Malhotra, Dipesh Kumar Dhoot. Clinical Profile of Alcohol-related liver disease in a Tertiary Care Centre and its Correlation with Type, Amount and Duration of Alcohol Consumption. June 2015 (63).
85. Swati Hegde, ArunVishnar, Girish B. Ramteke. Study of clinical and laboratory profile in alcohol-related liver disease with emphasis on renal function. Int Jour of research in med sci. 2015;2 (3)
86. Cabez J, bataller R. Alcohol-related liver disease: New UK alcohol guidelines and Dry January: enough to give up boozing? Nat Rev Gatroentrol Hepato 2016; 13:191.0
87. Torruellas C, French SW, Medici V. Diagnosis of alcohol-related liver disease. World J Gastroenterol. 2014;20(33):11684-11699.
88. Ray G, Manjubhargav P. Clinical Presentation and Mortality Determinants of Alcohol-Related Liver Disease: A Single-Center Experience of the Rising Menace from Eastern India. InflammIntest Dis. 2019;4(3):104-114.
89. Huang LL, Xia HH, Zhu SL. Ascitic Fluid Analysis in the Differential Diagnosis of Ascites: Focus on Cirrhotic Ascites. J ClinTranslHepatol. 2014;2(1):58-64.
90. O'Shea RS, Dasarathy S, McCullough AJ (January 2010). "Alcohol-related liver disease: AASLD Practice Guidelines" (PDF). Hepatology. 51 (1): 307–28.
91. Bruha R, Dvorak K, Petrtyl J. Alcohol-related liver disease. World J Hepatol. 2012;4(3):81-90.
92. Hasin DS, O'Brien CP, Auriacombe M, et al. DSM-5 criteria for substance use disorders: recommendations and rationale. Am J Psychiatry. 2013;170(8):834-851.
93. Schuppan D, Afdhal NH. Liver cirrhosis. Lancet. 2008;371(9615):838-851.
94. Gül F, Arslantaş MK, Cinel İ, Kumar A. Changing Definitions of Sepsis. Turk J AnaesthesiolReanim. 2017;45(3):129-138.
95. Hepatic encephalopathy in chronic liver disease: 2014 practice guideline by the European Association for the Study of the Liver and the American Association for the Study of Liver Diseases. J Hepatol. 2014; 61: 642-659
96. Ng CK, Chan MH, Tai MH, Lam CW. Hepatorenal syndrome. ClinBiochem Rev. 2007;28(1):11-17.
97. Roth JA, Chrobak C, Schädelin S, Hug BL. MELD score as a predictor of mortality, length of hospital stay, and disease burden: A single-centre retrospective study in 39,323 inpatients. Medicine (Baltimore). 2017;96(24):e7155.
98. Kamath PS, Kim WR; Advanced Liver Disease Study Group. The model for end-stage liver disease (MELD). Hepatology. 2007 Mar;45(3):797-805.
99. Peng Y, Qi X, Guo X. Child-Pugh Versus MELD Score for the Assessment of Prognosis in Liver Cirrhosis: A Systematic Review and Meta-Analysis of Observational Studies. Medicine (Baltimore). 2016;95(8):e2877
100. Vignesh BT, Singh AK, Mohan SK, Murthy S, Joshi A. Association between socio-demographics and alcohol dependence among individuals living in an Indian setting. Glob J Health Sci. 2014;6(3):16-26.
101. MP Virukal pattigopalratnam, Anil Arora,S. Mukhewar .Socio economic and demographic profile of patients with alcohol-related liver disease in India. Journal of clinical and experimental hepatolog.2013;22-28
102. Narawane NM, Bhatia S, Abraham P, Sanghani S, Sawant SS. Consumption of 'country liquor' and its relation to alcohol-related liver disease in Mumbai. J Assoc Physicians India. 1998 Jun;46(6):510-3.
103. Askgaard G, Grønbæk M, Kjær MS, Tjønneland A, Tolstrup JS. Alcohol drinking pattern and risk of alcoholic liver cirrhosis: a prospective cohort study. J Hepatol. 2015 May;62(5):1061-7.
104. Bellentani S, Saccoccio G, Costa G, Tiribelli C, Manenti F, Sodde M, SaveriaCrocè L, Sasso F, Pozzato G, Cristianini G, Brandi G. Drinking habits as cofactors of risk for alcohol-induced liver damage. The Dionysos Study Group. Gut. 1997 Dec;41(6):845-50.

105. Kerr WC, Fillmore KM, Marvy P. Beverage-specific alcohol consumption and cirrhosis mortality in a group of English-speaking beer-drinking countries. Addiction. 2000 Mar;95(3):339-46.
106. Becker U, Deis A, Sørensen TI, Grønbaek M, Borch-Johnsen K, Müller CF, Schnohr P, Jensen G. Prediction of risk of liver disease by alcohol intake, sex, and age: a prospective population study. Hepatology. 1996 May;23(5):1025-9.
107. Savolainen. Oscar-Berman M, Marinkovic K. Alcohol: effects on neurobehavioral functions and the brain. Neuropsychol Rev. 2007;17(3):239-257.
108. Anand Roy N. Prevalence and determinants of co-use of alcohol and tobacco among men in the working-age group (18-59 years) in India. EpidemiolBiostat Public Health. 2016;13:804–08.