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General Medicine

CLINICO-ETIOLOGICAL PROFILE OF CHRONIC LIVER DISEASE AMONG FEMALE PATIENTS ATTENDING A TERTIARY CARE CENTRE

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ABSTRACT **Background:** Chronic liver disease is a major cause of morbidity and mortality worldwide. The epidemiology of cirrhosis of liver is characterised by marked differences between genders, ethnic groups and geographic regions. Cirrhosis is not uncommon in women. Female physiology, particularly the hormonal influences of estrogen and progesterone, plays a pivotal role in the progression and severity of chronic liver disease. **Aim:** To evaluate the clinico-etiological profile of chronic liver disease among female patients attending a tertiary care centre, and to document their presenting complaints, complications, variceal grading, radiological findings, and CTP class at first assessment. **Materials and Methods-:** Total 116 female patients fulfilling the GARCIA – TSAO criteria for cirrhosis of liver aged more than and equal to 13 years, who were attending Medicine out-patient department and those who were admitted in the Department of Medicine, AMCH during the period of one year were taken. Patients were divided according to the etiology of chronic liver disease and their clinical presentation. Patients were scored according to the Child-Turcotte-Pugh classification. **Result-** Our study displays, that alcohol related CLD is the most common cause of cirrhosis among females in our region and that the average duration of alcohol intake among alcohol related female CLDs was 22.42 ± 7.44 years. The most common presenting complain was swelling of feet (79.31%) and abdominal distension (75.86%) overall and as well as in the alcoholic CLD group. Portal hypertension (87.93%) was overall the most common complication. Most of the study subjects had Grade II varices (32.76%). Radiological diagnosis of chronic liver was done in majority by ultrasound whole abdomen with portal vein doppler (94.83%). Most cases during first assessment had CTP score of Class C (57.76%). **Conclusion-** In conclusion, our study showed that alcohol has become the leading cause of cirrhosis among women in North-Eastern of the country. Our study showed that most of the patients during first assessment belonged to CTP class C which correlates with the fact that most patients seek medical care only during the later stage of cirrhosis as well as due to the difficulty in diagnosing CLD in its early stages.

KEYWORDS : Female; Chronic liver disease; Cirrhosis; Etiology; Portal hypertension.

INTRODUCTION

Cirrhosis represents the final stage of numerous chronic liver diseases (CLD)[1] and is defined by extensive liver fibrosis that disrupts normal hepatic architecture and replaces it with regenerative nodules[2]. Cirrhosis develops gradually due to persistent liver injury over weeks to years, although recent advances indicate that fibrosis may be reversible if the underlying cause is effectively managed[3]. Female patients demonstrate distinct risk patterns compared with males. Women are more vulnerable to cirrhosis even at lower levels of alcohol consumption, largely due to differences in alcohol metabolism, hormonal influences, and genetic factors[4]. Moreover, autoimmune liver diseases are more frequent in females, and metabolic dysfunction-associated steatotic liver disease (MASH) is increasingly recognized among women, particularly after menopause due to rising obesity, diabetes, and insulin resistance.

The Garcia–Tsao criteria serve as a non-histopathological diagnostic framework for cirrhosis. A case is defined when clinical signs of hepatocellular dysfunction such as jaundice, hepatic encephalopathy, cutaneous stigmata (spider angiomas, palmar erythema), and endocrine changes are present. Additionally, signs of portal hypertension including hematemesis, melena, splenomegaly, ascites, or varices on endoscopy strengthen the diagnosis. Supportive ultrasonographic findings include coarse echotexture, a nodular liver surface, an increased caudate-to-right lobe ratio, and features of portal hypertension such as ascites, splenomegaly, varices, and reduced portal venous flow. The 2023 update from the Global Burden of Liver Disease reports that cirrhosis is responsible for nearly two million deaths annually, accounting for about 4% of all deaths and ranking as the 11th leading cause of mortality worldwide[5].

MATERIALS AND METHODS

Hospital-based cross-sectional observational study conducted in the Department of Medicine, Assam Medical College & Hospital (AMCH), Dibrugarh, over one year (1 November 2023–31 October 2024). The study included female patients attending the Medicine OPD and those admitted to Medicine wards. All consecutive female patients aged ≥13 years with chronic liver disease (CLD) were enrolled. The calculated sample size was 116.

Eligibility Criteria

Inclusion: Females ≥13 years meeting Garcia–Tsao criteria for cirrhosis.

Exclusion: Pregnancy, acute liver failure, post-hepatic transplant status, lack of consent, and age <13 years.

Methodology

Demographic details, risk factors (including alcohol and drug/herbal use), comorbidities, and presenting complaints were recorded. General and systemic examinations were performed with documentation of CLD stigmata and complications.

Diagnostic Definition of Cirrhosis: Based on clinical hepatocellular failure, features of portal hypertension, and ultrasound findings suggestive of cirrhosis.

Etiology Classification:

- **HBV:** HBsAg positive.
- **HCV:** Anti-HCV antibody positive.
- **Alcohol-related:** Consistent history of harmful alcohol intake in women with supportive biochemistry.
- **MASH:** In patients with metabolic risk factors and fatty liver after excluding other causes and excess alcohol.
- **Autoimmune Hepatitis:** Autoantibody profile and scoring after relevant exclusions.
- **Wilson Disease:** Ceruloplasmin, 24-hour urinary copper, and slit-lamp examination for Kayser–Fleischer ring.
- **Hemochromatosis:** Serum ferritin and transferrin saturation.
- **Primary Biliary Cholangitis:** Elevated ALP with positive AMA.
- **Cryptogenic:** Assigned after exclusion of the above causes.

Laboratory tests included CBC, renal function, liver function panel, electrolytes, PT/INR, fasting lipid profile, and HbA1c. Etiology work-up comprised HBsAg, Anti-HCV, autoimmune profile (ANA, AMA, ASMA, anti-LKM1, pANCA), ceruloplasmin, serum ferritin, transferrin saturation, and 24-hour urinary copper. Imaging included abdominal ultrasonography with portal vein doppler; elastography (point shear wave on SAMSUNG RS80A platform) was performed when indicated. Upper gastrointestinal endoscopy (Olympus CV-70) was done for variceal screening/assessment as clinically warranted. Instruments/analyzers used on site included Sysmex XN-550 (CBC)

and VITROS 5600 (biochemistry). All tests were performed in AMCH laboratories as per standard departmental protocols.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using IBM SPSS v21. Categorical variables are presented as counts and percentages; continuous variables as mean ± standard deviation.

RESULTS

Table 1 : Clinico-Etiological Profile

The mean age of the study population was 44.9 ± 10.9 years, with the majority belonging to the 41–50 years age group (37.9%), followed by 31–40 years (24.1%). Only one patient (0.9%) was below 20 years, indicating that chronic liver disease (CLD) in females predominantly affects those in the middle age groups. Most patients were from rural areas (62.9%), reflecting either higher prevalence in rural populations or better referral rates to tertiary centers from these regions.

The most common presenting complaints were swelling of feet (79.3%), abdominal distension (75.9%), and abdominal pain (62.9%). Other frequent complaints included jaundice (33.6%), altered sensorium (33.6%), and loss of appetite (49.1%). Menstrual irregularities were reported in 37.1% of cases, indicating the systemic impact of CLD on female reproductive health.

On clinical examination, ascites (80.2%), pedal edema (79.3%), and pallor (75.0%) were the most frequent findings, followed by splenomegaly (67.2%) and peripheral stigmata of CLD (57.8%). Asterixis was seen in 32.8% cases, signifying hepatic encephalopathy. Rare findings included KF ring (2.6%). Peripheral stigmata such as loss of axillary/pubic hair (35.3%) and breast atrophy (34.5%) were also frequent.

With respect to risk factors, alcohol use (62.9%) and indigenous medicine intake (40.5%) were common. Etiologically, alcohol-related CLD accounted for 54.3%, followed by MASLD (15.5%) and cryptogenic cases (15.5%). Viral hepatitis (HBV 4.3%, HCV 2.6%), autoimmune hepatitis (4.3%), and Wilson's disease (2.6%) were less frequent. Hemochromatosis was not observed.

Table 2: Ascites and Hepatic Encephalopathy Grading

Regarding severity, ascites was almost universal with 89.7% of patients showing fluid accumulation. Massive ascites was seen in 42.2%, and moderate ascites in 37.9%. Only 10.3% had no ascites. In terms of hepatic encephalopathy, more than half (55.2%) had no encephalopathy at presentation. However, Grade II (24.1%) and Grade III (9.4%) encephalopathy were not uncommon, while 3.4% presented with severe Grade IV encephalopathy, highlighting the advanced disease state in a subset of patients.

Table 3 : Complications by Etiology

Portal hypertension was the most common complication, observed in 53.4% of alcohol-related cases and 12.9% of cryptogenic cases. UGI bleed occurred in 24.1% of alcohol-related CLD, while spontaneous bacterial peritonitis (SBP) was seen in 19.8%.

Anemia (44.8%), hepatic encephalopathy (32.7%), and coagulopathy (34.4%) were common among alcohol-related CLD, reflecting multi-system involvement. MASLD showed lower but notable rates of infections (7.7%) and SBP (7.7%). Viral hepatitis contributed to coagulopathy (2.5–4.3%) and anemia. Autoimmune hepatitis cases had coagulopathy (4.3%) and anemia (4.3%), while Wilson's disease showed a small contribution to anemia (2.5%). HCC was observed only in one cryptogenic case (0.8%).

Table 4: Laboratory and Biochemical Findings

Laboratory findings revealed severe anemia (mean Hb 8.47 g/dl), thrombocytopenia (mean platelet count 1.06 × 10^5/cumm), and leukopenia (1.04 × 10^3/cmm), reflecting portal hypertension and hypersplenism. Renal impairment was common with mean urea 68.05 mg/dl and creatinine 1.42 mg/dl. Hyponatremia (mean Na^+ 132 mmol/l) and mild hypokalemia were also noted. Coagulopathy was significant, with mean PT 17.0 sec and INR 1.63.

Liver function tests showed low albumin (2.53 g/dl), raised AST (116 U/L), ALT (61.6 U/L), and ALP (140.9 U/L). Hyperbilirubinemia was marked (mean total 4.3 mg/dl). The AST/ALT ratio was elevated (2.27), typical for alcohol-related liver disease.

Metabolic evaluation showed near-normal glycemic control (HbA1c 5.6%), with dyslipidemia: low HDL (34.5 mg/dl), raised triglycerides (144 mg/dl), and elevated LDL (106 mg/dl), consistent with MASLD and advanced liver disease.

Table 5 : Radiological, Endoscopic, and CTP Scores

Ultrasound with Doppler was diagnostic in 94.8% cases, while Fibroscan was performed in 23 patients. Among these, 69.5% had F4 fibrosis, confirming cirrhosis, and 30.4% had F2–F3 fibrosis.

Endoscopic evaluation revealed esophageal varices in 90.8% of patients undergoing upper GI endoscopy (n=76). Grade II varices (38.2%) were most common, followed by Grade III (27.6%) and Grade I (25%). Only 9.2% had no varices.

On severity assessment using the Child–Turcotte–Pugh (CTP) score, most patients were in Grade C (57.8%), reflecting advanced decompensated CLD. Grade B accounted for 38.8%, while only 3.5% were compensated (Grade A).

Table 1: Clinico-Etiological Profile of Chronic Liver Disease in Female Patients (n = 116)

Category	Variable	Number (n=116)	Percentage (%)
Age Distribution	13–20 yrs	1	0.86
	21–30 yrs	14	12.07
	31–40 yrs	28	24.13
	41–50 yrs	44	37.93
	51–60 yrs	20	17.24
	>60 yrs	9	7.76
	Mean ± SD	44.92 ± 10.88	—
Area of Residence	Rural	73	62.93
	Urban	43	37.06
Presenting Complaints	Swelling of feet	92	79.31
	Abdominal distension	88	75.86
	Jaundice	39	33.62
	Hematemesis	14	12.07
	Malaena	37	31.90
	Hematochezia	0	0.00
	Altered sensorium	39	33.62
	Fever	44	37.93
	Abdominal pain	73	62.93
	Bruises	19	16.38
	Pruritus	5	4.31
	Loss of appetite	57	49.14
	Menstrual irregularity	43	37.07
Clinical Signs	Pallor	87	75.00
	Icterus	44	37.93
	Pedal edema	92	79.31
	Ascites	93	80.17
	Splenomegaly	78	67.24
	Peripheral stigmata of CLD	67	57.76
	Asterixis	38	32.76
	KF ring (slit lamp)	3	2.58
	Poor nutrition	48	41.37
	Hypertension	19	16.38
Peripheral Stigmata of CLD	Bilateral parotid swelling	20	17.24
	Spider naevi	5	4.31
	Loss of axillary/pubic hair	41	35.34
	Breast atrophy	40	34.48
Risk Factors	Alcohol use	73	62.93
	Blood transfusion history	24	20.69
	IV drug abuse	5	4.31
	Tattooing	13	11.21
	Multiple sexual partners	0	0.00
	Indigenous medicine use	47	40.52
	Metabolic syndrome	14	12.07
Etiology of CLD	Alcohol	63	54.31
	MASLD	18	15.51
	Chronic Hepatitis C	3	2.58
	Chronic Hepatitis B	5	4.31
	Autoimmune hepatitis	5	4.31

	Wilson disease	3	2.58
	Hemochromatosis	0	0.00
	Primary biliary cirrhosis	1	0.86
	Others	0	0.00
	Cryptogenic	18	15.51

Table 2: Distribution of Cases Based on Ascites and Hepatic Encephalopathy Grading (n = 116)

Ascites Grading	Number (n=116)	Percentage (%)	Hepatic Encephalopathy Grading	Number (n=116)	Percentage (%)
None	12	10.34	None	64	55.17
Mild	11	9.48	Grade I	9	7.70
Moderate	44	37.93	Grade II	28	24.13
Massive	49	42.24	Grade III	11	9.40
—	—	—	Grade IV	4	3.40

Table 3: Complications of Chronic Liver Disease in the Various Sub-Groups (n = 116)

Complications	Alcohol (%)	MASLD (%)	Chronic Hepatitis C (%)	Chronic Hepatitis B (%)	AIH (%)	Wilson Disease (%)	Cryptogenic (%)
Portal HTN	53.4	1.2	1.7	3.4	2.5	0.8	12.9
UGI bleed	24.1	1.7	0.8	1.7	0	0	3.4
Infections	21.5	7.7	0.8	0.8	1.7	0	5.1
HRS	25.8	4.3	0.8	0	0	0	3.4
HE	32.7	4.3	0.8	0.8	0.8	0.8	4.3
Coagulopathy	34.4	6.0	0.8	2.5	4.3	0	6.0
Anemia	44.8	7.7	1.7	4.3	4.3	2.5	9.5
SBP	19.8	7.7	0	2.5	1.7	0	7.7
HCC	0	0	0	0	0	0	0.8

Table 4 : Laboratory Parameters and Liver Function Tests in the Study Group (n = 116)

Parameter	Mean	SD
General Laboratory Parameters		
Hb% (mg/dl)	8.47	2.84
Total Count (per/cmm × 1000)	1.04	0.72
Platelet Count (per/cumm × 100000)	1.06	0.39
Creatinine (mg/dl)	1.42	1.32
Urea (mg/dl)	68.05	53.73
Serum Sodium (mmol/l)	132.00	5.60
Serum Potassium (mmol/l)	3.76	0.96
Prothrombin Time (sec)	17.03	5.51
INR	1.63	0.57
Liver Function Tests		
Total Protein (gm/dl)	6.41	1.26
Globulin (gm/dl)	3.96	0.94
Albumin (gm/dl)	2.53	0.59
AST (U/L)	116.25	62.91
ALT (U/L)	61.61	33.69
Total Bilirubin (mg/dl)	4.33	4.46
Direct Bilirubin (mg/dl)	2.88	3.51
ALP (U/L)	140.98	67.84
AST/ALT Ratio	2.27	1.58
Metabolic Parameters		
RBS (mg/dl)	114.41	40.28
HbA1c (%)	5.62	1.18
Total Cholesterol (mg/dl)	166.26	43.18
Triglycerides (mg/dl)	144.06	102.92
LDL (mg/dl)	106.57	37.94
HDL (mg/dl)	34.48	9.83

Table 5: Radiological, Fibroscan, Endoscopic, and CTP Score Findings in the Study Population

Parameter	Number	Percentage(%)
Radiological Findings (n=116)		
Diagnosed using USG Whole Abdomen & Portal Vein Doppler	110	94.83
Diagnosed using Fibroscan	23	19.82
Fibroscan Grading (n=23)		
N	0	0.00
F1	0	0.00
F2–F3	7	30.4
F4	16	69.5
Upper GI Endoscopy – Esophageal Varices (n=76)		
No varices	7	9.21
Grade I varices	19	25.00
Grade II varices	29	38.15
Grade III varices	21	27.63
Child–Turcotte–Pugh (CTP) Score (n=116)		
Grade A (5–6)	4	3.45
Grade B (7–9)	45	38.79
Grade C (10–15)	67	57.76

Figure 1 : A Female CLD Patient with Autoimmune Hepatitis with Vitiligo

Figure 2 : Alcoholic CLD Female with Massive Ascites

DISCUSSION

The present study included female CLD patients aged more than and equal to 13 years. In our research, the average age of the research population was 44.92 ± 10.88 years, with most participants (37.93%) between 41 and 50 years of age group, followed by 24.13% in the 31–40 years of age group; the youngest (≤20 years, 0.86%) and oldest (>60 years, 7.76%) groups were the fewest. This pattern aligns with reported middle-age clustering of female cirrhotics; Brij Sharma et al. (2016)[6] found most female CLD patients between 40–59 years with a mean age of 51.2 ± 8.9 years. The findings of our study were consistent with those observations, showing maximum prevalence in 41–50 years with an average age of 44.92 ± 10.88 years.

In the area-wise distribution, most subjects (62.93%) belonged to rural areas, and 37.06% were from urban areas. Alcoholic cirrhosis has been more frequently reported in rural populations; A.M. Patil (2015)[7] observed higher rural burden, which is comparable to our rural predominance and may reflect environmental, socioeconomic, and access-to-care differences.

In the present study, the most frequently observed symptoms were swelling of the feet (79.31%) and abdominal distension (75.86%). A

tertiary-center series mirrored heavy symptom burden with abdominal distension and jaundice prominent; Chandra et al.[8] reported abdominal distension in 87.1% and jaundice in 69.4%, which is broadly comparable to our pattern, though swelling of feet was more frequent in our cohort.

In the present study, ascites (80.17%) and pedal edema (79.31%) were the most commonly observed clinical signs. Pallor (75%), splenomegaly (67.24%), peripheral stigmata of chronic liver disease (57.76%), poor nutrition (41.37%), and icterus (37.93%) were other suggestive findings. A cross-sectional study from Eastern India showed prominent pallor and icterus and variable edema rates; Rupak Chatterjee et al.[9] observed high frequencies of pallor and icterus, and our edema rate (79.31%) was higher than several prior reports, indicating decompensated presentation.

The present study showed alcohol consumption as the most prominent risk factor (62.93%), with use of indigenous medicine notable (40.52%); blood transfusion history (20.69%) and metabolic syndrome (12.07%) were additional risks, while IV drug use was uncommon and no subject reported multiple sexual partners. Blood-borne transmission emerged as the likely route for viral hepatitis in females. Historical series have also emphasized transfusion-related risk; Di Martino et al.[10] found a high proportion of CLD patients with prior transfusions, which supports our inference regarding parenteral routes in female HBV/HCV.

In the present female-only cohort, the most common cause of chronic liver disease was alcohol-related liver disease (54.31%), followed by MASLD and cryptogenic cirrhosis (each 15.51%); viral hepatitis was less frequent (HBV 4.31%, HCV 2.58%), autoimmune hepatitis (4.31%) and Wilson's disease (2.58%) were rarer, with one case of primary biliary cirrhosis. Female-focused etiologic spectra vary across regions; Rajarajan A.[11] reported cryptogenic predominance with lower alcohol-related cirrhosis among women, whereas our region shows alcohol predominance, reflecting local cultural and exposure patterns.

Alcoholic liver disease in females deserves emphasis. Women consume less alcohol overall yet are more susceptible to alcohol's hepatotoxicity due to lower gastric alcohol dehydrogenase and a smaller volume of distribution; liver injury progresses faster in women. The quantity and duration of alcohol consumption are key determinants. A large population-based cohort demonstrated dose-dependent risk escalation with increasing intake; Becker et al.[12] reported that cirrhosis risk rises with weekly alcohol dose, supporting our observation that duration (22.42 ± 7.44 years) and amount (≈48.7 g/day) were substantial among alcoholic cirrhotics in this region, where access to country liquor is high.

Regarding MASLD in females, steatotic liver disease becomes more prevalent after menopause due to hormonal shifts, rising metabolic syndrome, and changes in body composition. A global synthesis indicated higher prevalence in men than premenopausal women, with a marked rise in women post-menopause; Younossi et al.[13] highlighted the sex-hormone link to steatotic liver disease, which matches our finding that MASLD-related cirrhosis clustered beyond 40 years with frequent diabetes and hypertension.

Women are significantly more likely than men to develop autoimmune liver diseases. In our study there were five cases of probable autoimmune hepatitis and one case of primary biliary cirrhosis. A recent Indian overview confirmed female predominance and Type 1 AIH as the most common form; Kumar et al.[14] documented the higher burden of AIH and PBC among women, which is consistent with our low but notable autoimmune representation.

Our cohort recorded 4.31% chronic hepatitis B and 2.58% chronic hepatitis C among women. Data indicating more favorable HBV immunologic transitions in females have been reported; Zacharakis et al.[15] described higher rates of HBeAg to anti-HBe seroconversion in women, which aligns with the relatively lower HBV/HCV frequencies observed in this female-only study in the era of vaccination and improved transfusion safety.

Our severity data showed extensive fluid overload: 89.65% had ascites, with massive (42.24%) and moderate (37.93%) grades predominating; only 10.34% had no ascites. Hepatic encephalopathy was present in 44.82% overall, with Grade II (24.13%) most frequent and Grades III–IV in 12.8%. Another South Asian series reported lower ascites and encephalopathy at presentation; Suhail Ahmed Almani et al.[16] observed ascites in 59% and encephalopathy in 24%, suggesting our cohort sought care later with greater decompensation.

Complications reflected portal hypertensive and systemic involvement: portal hypertension was the prime complication (overall burden highest across sub-groups), followed by anemia and coagulopathy; spontaneous bacterial peritonitis, infections, and encephalopathy contributed substantially, while hepatocellular carcinoma was rare. Variceal evolution is a key risk in cirrhosis, with cumulative development common and a sizeable proportion bleeding; Bruce R. Bacon[17] noted that a substantial fraction will develop varices over time and about one-third will bleed, underscoring our high decompensation burden and the need for surveillance.

On prognostic stratification, most patients were Child–Turcotte–Pugh class C at first assessment (57.76%), followed by class B (38.79%) and a small fraction class A (3.45%). Such distribution indicates advanced decompensation at presentation in females in this setting. A regional female-focused series similarly found a predominance of intermediate-to-advanced CTP classes; S. Talukdar[18] reported B and C classes composing the majority, mirroring the late presentation seen in our cohort and reinforcing the need for earlier detection, risk-factor modification, and structured referral pathways.

CONCLUSION

Alcohol emerged as the predominant cause of cirrhosis among women in our setting. At first presentation, most patients were classified as Child–Turcotte–Pugh class C, highlighting late-stage presentation—likely due to delayed health-seeking and the challenges of detecting chronic liver disease in its early phases.

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