

CHRONIC LIVER DISEASE IN WOMEN: A STUDY ON THE CLINICAL PROFILE IN A TERTIARY HOSPITAL IN CENTRAL KERALA

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ABSTRACT

Background: The escalating prevalence of chronic liver disease (CLD) in pandemic proportions crossing geographic and socioeconomic boundaries poses a major public health challenge. Sex-specific data on chronic liver disease (CLD) are limited, yet patterns of etiology, presentation and severity often differ between women and men. There is an alarming increase in the proportion of CLD in women. We describe clinical characteristics in a single-centre cohort of 100 women with Chronic liver disease in a tertiary Hospital in a Metropolitan city. **Materials and Methods:** This was a cross-sectional observational study to assess the clinical, etiological, laboratory profile and severity indices of chronic liver disease among 100 female patients admitted in the medical department of a Government Medical College hospital in Ernakulam. **Result:** In this cohort of 100 female patients with CLD, Metabolic dysfunction associated steatotic liver disease (MASLD) accounted for 64% of the cases making it the leading etiology. Mean age group affected was between 60 and 70 years of age. Most common presentation was abdominal distension in 31 patients. Hypoalbuminemia was seen in 90% of the patients. Deranged prothrombin time/INR was noted in 31 patients. Thrombocytopenia was noted in 59% of the patients. Ultrasonography showed coarse or abnormal liver parenchymal changes in all the patients. Anemia was noted in all. HbA1C ≥ 6.5 in 47 patients. **Conclusions:** In women with chronic liver disease in this cohort, MASLD with T2DM is the dominant etiologic factor. The findings suggest that metabolic dysregulation exerts deleterious effects on hepatic synthetic function, even before classical liver severity changes become evident.

INTRODUCTION

Chronic liver disease (CLD) is often described as a silent epidemic due to its rising prevalence, delayed diagnosis and significant impact on morbidity and mortality.^[1] CLD develops from progressive and long-term liver damage over months to years leading to fibrosis and eventually cirrhosis. Cirrhosis is the 11th leading cause of death and 15th leading cause of morbidity accounting for 2.2% of death.^[2]

Review of literature

The Global epidemiology of chronic liver disease indicate a shift in etiologies with a rising metabolic burden in recent years. Declining rates of hepatitis C

and B in vaccinated or treated populations have been offset by the sharp rise of metabolic dysfunction-associated steatotic liver disease/ Non alcoholic fatty liver disease. (MASLD/NAFLD).^[3] This transition reflects changing dietary and metabolic profiles globally, and has specific relevance for women, particularly after menopause, when metabolic risk accelerates.^[1]

NAFLD / MASLD is the single biggest driver of rising CLD worldwide, and prevalence and progression show sex and age-specific patterns. Men have higher NAFLD prevalence overall, but post-menopausal women show faster progression to NASH and fibrosis.^[4] Progression to Metabolic

associated steatohepatitis (MASH) is a key risk factor for cirrhosis, hepatocellular carcinoma and cardiovascular events. Alterations in reproductive hormones are linked to the development and progression of NAFLD/NASH in women.^[5]

In 2019, Hepatitis B virus, hepatitis C virus, and alcohol were observed to be the primary causes of liver cirrhosis and other chronic liver diseases deaths at the global level, accounting for 74.61% of liver cirrhosis and other chronic liver diseases-related mortalities.^[6]

Indian systematic reviews and multicentric studies reveal high MASLD prevalence (~38.6%) among adults, with a notable burden among women. However, alcohol and viral hepatitis remain important contributors in hospital-based series, particularly in northern and eastern regions. The relative contributions of MASLD, alcohol, and viral causes vary by geography, access to care, and referral bias.^[7,8,9]

In 2023, the international hepatology community, through a multi-society consensus, formally renamed non-alcoholic fatty liver disease (NAFLD) as metabolic dysfunction-associated steatotic liver disease (MASLD). This change was made to emphasize the central role of metabolic dysfunction in disease pathogenesis.^[10] MASLD is diagnosed by the presence of hepatic steatosis (fatty liver) on ultrasound imaging along with at least one of 5 cardio metabolic risk factors namely obesity, type 2 diabetes, hypertension, high triglyceride or low HDL cholesterol.

MASLD is increasingly recognized as the predominant cause of fatty liver disease, superseding older terms like NAFLD.^[10]

Although direct markers of MASLD are currently not available, Obesity & Type2 diabetes are strong clinical risk factors for disease progression.^[11]

While MASLD is more prevalent in men, post-menopausal women are at increased risk due to a complex interplay between estrogen deficiency, visceral fat accumulation and metabolic syndrome.^[12] Sex hormones and adiposity distribution influence liver disease progression. Estrogen has antifibrotic effects pre-menopause, but post-menopausal hormonal shifts promote visceral adiposity and insulin resistance, increasing the risk of fibrosis and metabolic CLD progression. Alcohol metabolism differences also amplify female susceptibility to alcohol-related injury.^[13,14]

Much of CLD goes undiagnosed due to lack of significant symptoms until complications of cirrhosis sets in.^[15] People with compensated chronic liver disease have a better outlook than people with decompensated liver disease like upper gastrointestinal bleeding, hepatic encephalopathy or worsening jaundice.^[12]

The burden of liver disease in India is significant because it contributed to 18.3% of the 2 million global liver disease related deaths in 2015.^[16] The lifestyle transition that India is passing through currently with adoption of a western diet, sedentary

habits grounds for a spectrum of liver disease which includes NAFLD/ MASLD. An issue that impedes early treatment of CLD in India is that they present late in the clinical course, most commonly after decompensation sets in.^[17]

Prevalence of MASLD is more than 25% globally and 9% -32% in general Indian population. Higher prevalence is associated with overweight, obese and diabetes.^[18]

Recent reviews emphasize that female-only cohorts are rare and that sex-aware analyses are urgently needed. With global trends moving toward metabolic aetiologies, studies focusing on women can clarify sex-specific differences in natural history, progression, and treatment outcomes.^[19,20]

We had noticed a rise in cases of CLD in women in recent years in our medical college hospital. Hence, we set out to study the clinical profile, aetiology and laboratory parameters in CLD in women.

MATERIALS AND METHODS

This was an observational cross-sectional case study conducted in the medicine department in the Government Medical College hospital, Ernakulam. IEC approval was obtained before starting the study. The study involved female patients above the age of 18 years admitted in the women's ward or ICU with features of CLD either clinically or from investigations.

A medical history and clinical examination was done for all patients who consented to be part of the study. Laboratory investigations included Complete blood picture, Liver function test, Renal function test, coagulation profile, serum electrolytes, uric acid, blood sugars, viral hepatitis profile HBs Ag, HCV antibody, HIV if indicated. Imaging in the form of USG was carried out for all patients. Iron studies, ANA profile was done when warranted. Ascitic fluid analysis was done for those who presented with ascites.

Study Population: The study involved 100 female patients aged above 18 years who were admitted in our medical department and diagnosed with CLD. This study was conducted between February 2024 to August 2025.

Data collection& Analysis: Data was entered into a pre-structured proforma for each patient after informed consent & maintaining confidentiality. The collective data was entered into MS excel before analysis. SPSS 23 software was used for the statistical analysis. Data was reported as standard deviation for quantitative variable and as percentage for qualitative variable.

Inclusion & Exclusion criteria: All female patients aged above 18 years who were diagnosed with chronic liver disease based on clinical evaluation, liver function test and abdominal ultrasonography were included in the study.

Pregnancy related liver disease, acute hepatitis, acute drug induced hepatitis & metastatic liver disease were excluded from the study.

RESULTS

A total of 100 patients with chronic liver disease were included in the study. Demographic data, clinical features, laboratory profile, ultrasound findings were collated & analysed. The variables used were age, symptoms, signs, haematological and biochemical parameters, etiological distribution of CLD,

ultrasound findings, the clinical outcome and complications if any during their hospital admission.

Socio-Demographic characteristics:

1. Age of the patients: Majority of CLD patients were in the 61–70 (29%), showing higher disease burden among post-menopausal & elderly women. The age group 51 to 60 and 71 to 80 group both had a proportion of 27% each. This is suggestive of post-menopausal worsening of chronic liver disease. Late presentations indicate lack of early significant symptoms or signs and also probable gender bias. Table one illustrates the age distribution among 100 female patients with chronic liver disease.

Table 1: Socio-demographic characteristics (n=100)

Age	Number	Proportion
31-40	3	3
41-50	8	8
51-60	27	27
61-70	29	29
71-80	27	27
81-90	6	6

Table 2: Distribution of patients based on Symptoms and Signs

Symptoms and Signs	No. of Patients
Abdominal Distension	32
Abdominal Pain	12
Altered Sensorium	24
Fever	16
Pedal Edema	2
Stroke	1
Upper G I Bleed	13
Total	100

Most common presenting symptoms were abdominal distension (32%), altered sensorium (24%), and fever (16%). Upper GI bleed was noted in 13%. Altered sensorium was suggestive of Hepatic encephalopathy & end stage liver disease. Peripheral stigmata of chronic liver disease as in males with alcoholic liver disease were not seen.

Table 3: Distribution of patients based on INR values

INR Value	No. of Patients
Normal	69
Abnormal	31
Total	100

31 patients had a prolonged INR suggesting coagulopathy & severe liver disease at presentation.

Table 4: Distribution of patients to Child Pugh Criteria

Child Pugh Criteria	No. of Patients
Class A	57
Class B	22
Class C	21
Total	100

Child Pugh score indicated severity of CLD: The variable used for calculation of score was bilirubin, albumin, prothrombin time, ascites, encephalopathy.

57% patients were in Class A. 21% presented in Class C

Majority (57%) were Child-Pugh Class A, indicating compensated liver disease; 43% were decompensated (Class B & C).

Table 5: Distribution of patients based on Platelet values

Platelet Value	No. of Patients
Below Normal	59
Normal	41
Total	100

Thrombocytopenia was seen in 59% of cases suggestive of hypersplenism.

Table 6: Distribution of patients based on USG Imaging

USG Imaging	No. of Patients
Coarse liver echotexture/ Fatty liver	53
Nodular liver	1
Nodular liver/ Portal Hypertension	46
Total	100

Ultrasound revealed coarse echotexture of liver in 53 patients and cirrhosis with portal hypertension was present in 46 patients indicative of progression of liver disease.

Table 7: Haematological and biochemical parameters

Variables	Mean $\pm$ SD
Hb	9.32 $\pm$ 2.26
WCC	7656.4 $\pm$ 3884.76
Platelet (Lakhs)	179612 $\pm$ 183423
Bilirubin (mg/dl)	2.21 $\pm$ 2.65
SGOT (U/L)	74.45 $\pm$ 145.51
SGPT (U/L)	54.24 $\pm$ 113.06
PT	21.08 $\pm$ 10.47
INR	1.32 $\pm$ 0.55
T. Proteins	6.15 $\pm$ 0.86
Albumin (g/dl)	2.69 $\pm$ 0.63
Globulin (g/dl)	3.47 $\pm$ 0.69
ALK PO4 (U/L)	136.01 $\pm$ 74.93
Uric Acid (mg/dl)	6.01 $\pm$ 4.77
Urea (mg/dl)	49.36 $\pm$ 35.38
Creatinine (mg/dl)	1.58 $\pm$ 1.28
Sodium (mEq/L)	131.46 $\pm$ 5.87
Potassium (mEq/L)	3.84 $\pm$ 0.79
RBS (mg/dl)	178.76 $\pm$ 70.33
HbA1C	6.45 $\pm$ 2.06

Interpretation:

- Low mean albumin and elevated bilirubin and liver enzymes confirm hepatic dysfunction.
- Mean Hb of 9.32 g/dl indicates anemia of chronic disease.
- Sodium levels slightly reduced, possibly due to hyponatremia secondary to cirrhosis.
- Biochemical profile showed abnormal elevation of liver enzymes
- Hypoalbuminemia was seen in a vast majority and was an eye opener to looking further for a cause for CLD
- Uric acid was found to be elevated in a few number of cases of chronic liver disease.
- Urea was noted to be low in advanced liver disease.
- Serum bilirubin was below 2 milligrams in most cases reflecting normal LFT can be deceiving in chronic liver disease. This suggests that it cannot be used a tool for screening for CLD.

Table 8: Glycemic Status

HbA1C	No. of Patients	Percentage (%)
< 6.5	53	53%
$\geq$ 6.5	47	47%

Nearly half the patients (47%) had diabetic-range HbA1C, correlating with MASLD predominance. In diabetics, insulin resistance, excess FFAs and chronic inflammation lead to fat accumulation, oxidative stress and fibrosis in the liver.

Table 9: Frequency Distribution of Etiological Factors

Etiology	Frequency
Alcohol induced liver disease	5
Autoimmune	3
Cause not known	16
Chronic Hepatitis B	6
HIV	1
Drug induced liver disease	14
Hepatitis C	3
Obesity	1
T2 DM/MASLD	64

Metabolic dysfunction-associated steatotic liver disease (MASLD) with T2DM was the predominant etiology (64%), drug-induced liver injury with indigenous medication, 1 patient was on methotrexate (14%) and

cryptogenic (unknown) causes (16%). Out of 100 pts 6 were positive for Hepatitis B infection, 3 for hepatitis C & 1 was HIV positive. Only 9 patients were serology positive for chronic hepatitis.

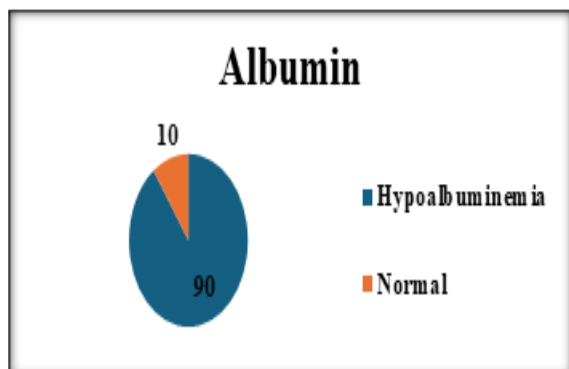


Figure 1: Graphical representation of Albumin

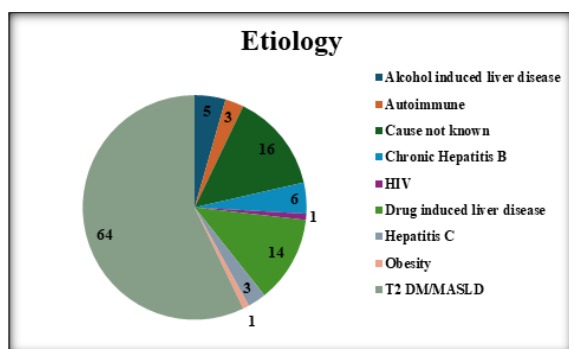


Figure 2: Graphical Representation of Etiology

DISCUSSION

In this cross-sectional study of 100 female patients with chronic liver disease (CLD), several salient observations emerge that highlighted the evolving epidemiology of CLD in women. The present study evaluated the clinical, biochemical, and etiological profile of chronic liver disease in women, focusing on the emerging influence of metabolic dysfunction. The coexistence of diabetes and MASLD in nearly half of the patients supports the growing recognition of metabolic dysfunction as a leading cause of chronic liver disease among women. This observation corresponds with global trends where MASLD has surpassed viral hepatitis as the predominant cause of liver disease. Post menopausal women manifested with the disease probably due to lack of protection from hormonal estrogen. This reinforces the concept that gender specific differences and metabolic risk factors must be addressed as central therapeutic targets in women with CLD.

The presence of significant liver damage even in people with normal liver tests during screening was an eye opener. It made us realise that normal liver test may falsely reassure people at risk of liver disease. The National Program for prevention and control of Cancer, Diabetes, Cardiovascular disease, and Stroke

has added Fatty liver disease to its screening efforts.^[21]

Age and demographic profile

In our cohort of 100 female patients, the mean age distribution peaked between 61–70 years (29%) and 71–80 years (27%), consistent with the postmenopausal age predominance reported by Trembling et al,^[15] who identified higher CLD risk in older women due to metabolic insults and estrogen decline. The age clustering in our population thus reflects the cumulative impact of metabolic syndrome, obesity, and estrogen decline on hepatic function. Indian studies also reflect a similar age trend in CLD between 4-6th decade.^[8,21,22]

Etiology

In our study, Metabolic dysfunction-associated steatotic liver disease (MASLD) due to Type 2 Diabetes Mellitus (T2DM) was the predominant etiology (64%), other main causes being drug-induced (14%) and cryptogenic (unknown) (16%) causes. These findings mirror global trends highlighted by Chan et al,^[23] and Younossi et al⁴ who reported MASLD as the leading cause of CLD worldwide, overtaking viral hepatitis.

Similarly, Tham et al's,^[19] data, demonstrated a rising global incidence of MASLD/MASH with a particularly sharp increase among women. In India, Mondal et al,^[8] and Elhence et al.⁷ reported NAFLD/MASLD prevalence rates between 25–35%, confirming the growing metabolic burden in South Asian females.

Mukherjee et al,^[17] found viral hepatitis caused 33.3% of CLD cases, while in our study it accounted for only 9%, reflecting a global trend driven by vaccination and antiviral efforts (Moon et al., Lazarus et al.). This shift from infectious to metabolic liver disease is also highlighted by Ramakrishnan,^[21] who noted that liver disease now extends beyond urban areas.

Even though body mass index was not included as a variable, majority of our patients were lean or average built & nourishment.

Clinical presentations

Abdominal distension (32%) altered sensorium (Hepatic encephalopathy) 24%, and fever (16%) were the most common presentations, suggesting that many women presented with decompensated disease. Comparable symptom profiles were noted in Indian cohorts from Uma Shankar et al,^[24] and Manikat et al,^[20] who also reported abdominal distension and encephalopathy as leading features in MASLD-related cirrhosis.

The occurrence of upper GI bleed (13 %) further indicates complications of portal hypertension.

Hematological and Biochemical Profile

Anemia was noted in all patients with a mean of 9.32 ± 2.26 . Thrombocytopenia was seen in 59 patients. Our patients showed marked hypoalbuminemia (90%) with mean albumin = 2.69 ± 0.63 g/dL and bilirubin = 2.21 ± 2.65 mg/dL. These findings are

similar to those reported by Meda et al,^[25] and Manikat et al,^[20] in female CLD cohorts, supporting the presence of low albumin with significant hepatic dysfunction and portal hypertension. This suggest that low albumin levels in blood could be used as a tool for further evaluation for CLD. Combined with elevated bilirubin, prolonged INR (31patients), and reduced platelets [thrombocytopenia (59%)] many patients were in advanced cirrhosis and portal hypertension.

Our data showed low urea in many cases with a mean of 49.36±35.38. This is because the urea cycle is localized to hepatocytes; when these cells are damaged or lost (as in end-stage liver disease), the capacity to convert ammonia and amino nitrogen to urea falls.^[26] The study also showed elevated uric acid in some cases with a mean of 6.01± 4.77 reflecting altered renal handling, reduced clearance, increased cell turnover, or shifts in renal excretion which may lead to elevated serum uric acid in many patients with cirrhosis.^[27]

Child–Pugh score

Using the Child–Pugh score, 57 % of patients were class A (compensated), while 43 % were classes B or C (decompensated). This mix suggests a spectrum of disease severity. The relatively higher proportion of class A could be due to earlier detection.

The predominance of Child–Pugh Class A (57%) in our study suggests that most patients were in early compensated disease stage, probably reflecting increased metabolic screening. Cooper et al,^[13] and Pemmasani et al,^[14] observed earlier diagnosis of CLD among women undergoing metabolic evaluations.

Glycemic Status and Association with MASLD

Our data collection showed known T2 DM as 64%. This high prevalence of diabetes aligns with the dominant etiological factor of MASLD/T2DM (64%) observed in the study.^[7,22]

In this cohort, 47% of patients had diabetic-range HbA1C ($\geq 6.5\%$), while 53% had lower values. Some patients with T2DM showed hypoglycemia, likely due to end-stage liver disease that impairs glycogen metabolism and insulin clearance, resulting in lower blood glucose levels.^[7]

Ultrasonography

Imaging findings of coarse/nodular liver texture and or features of portal hypertension in all patients validate the chronic and progressive nature of liver injury in this group.

Comparison with postmenopausal risk studies

Trembling et al,^[15] study demonstrated that obesity and high alcohol intake increased CLD risk in postmenopausal women. The predominance of metabolic risk factors and minimal alcohol exposure in our cohort emphasizes that in Indian women, metabolic, not alcoholic, pathways dominate disease causation. Marianna Chinucci et al,^[12] established links between estrogen deficiency, adiposity, and hepatic lipid metabolism, explaining the vulnerability of postmenopausal women to MASLD.

Indian context and public-health perspective

The Indian data presented by Ramakrishnan (2022) and Elhence et al. (2021) revealed rising CLD prevalence in both rural and urban women, primarily linked to obesity, diabetes, and dietary transitions. Our study corroborates this epidemiologic shift. The high proportion of MASLD and diabetes in our cohort supports the concept of a “silent dual epidemic” — the concurrent, subclinical rise of metabolic dysfunction and chronic liver disease among women.

Glycemic control, albumin, and disease severity associations

Patients with HbA1C $\geq 6.5\%$ exhibited lower albumin levels. The link between hyperglycemia and poorer albumin parallels findings that metabolic derangements accelerate progression of steatotic disease.^[30] These results are similar with Zeng et al,^[28] reinforcing metabolic dysfunction as a pathophysiologic cause. The absence of strong correlation between HbA1C and Child–Pugh class echoes other studies showing that liver disease severity is multifactorial and not solely dependent on metabolic parameters.^[29]

Clinical and research implications

1. Metabolic screening in CLD evaluation should routinely include glycemic profiling, even in patients without known diabetes.
2. The association of higher HbA1C with lower albumin suggests that improving glycemic control may help reduce progression of CLD
3. Lifestyle interventions (e.g. Mediterranean diet, weight loss of 7–10 % for overweight patients) are cornerstone recommendations in current MASLD guidelines.³⁰
4. Study strengths: A key strength is the focus on a female cohort with detailed metabolic, biochemical, and imaging data, which is relatively underrepresented in liver disease literature.
5. Under-reporting of female CLD, limited representation of women in research, and variation in access to diagnostics should be addressed.
6. A National registry for CLD should be maintained

CONCLUSION

Among women with chronic liver disease in this cohort, MASLD with T2DM is the dominant etiologic factor. Nearly half the patients had HbA1C in the diabetic range, and those with higher HbA1C had significantly lower albumin levels. These findings suggest that metabolic dysregulation exerts deleterious effects on hepatic synthetic function, even before classical liver severity changes become evident.

Limitations

- Sample size may limit statistical power in subgroup analyses

- Lack of advanced fibrosis scoring or liver histology
- BMI and Lipid profile were not included in the study.

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