Section: Hepatobiliary Sciences

Original Research Article

LIVER CIRRHOSIS AND TYPE 2 DIABETES – COMPLICATIONS AND OUTCOME FROM A TERTIARY CARE CENTRE IN CHENNAI - A PROSPECTIVE OBSERVATIONAL STUDY

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Abstract

Background: Although the underlying hepatic illness and its effects are more likely to negatively affect the prognosis of patients with diabetes mellitus (DM), the clinical effect of diabetes on liver cirrhosis (LC) remains unclear. This study assessed the mortality and complications of patients with LC with and without co-existing DM, as well as the prevalence of DM in patients with LC in a tertiary hospital in South India. The aim is to assess the prevalence of type 2 diabetes among patients with liver cirrhosis and study its complications and outcomes. **Materials and Methods:** This prospective observational study included patients with LC who fulfilled the inclusion criteria. The patients were subjected to a detailed clinical history to ascertain the etiological diagnosis of DM with and without complications. The patients were categorised into two groups: group A consisted of patients with DM and LC, and group B consisted of patients with LC without DM to compare the outcomes, risk factors, and complications associated with it. **Result:** In total, 228 patients with cirrhosis were included, of whom 24% of the LC patients had DM and the remaining 76% of the LC patients had no history of DM. The mean age of the study patients was 48.5±13.4 years, and the majority of them (84%) were male. The predominant aetiologies of cirrhosis were alcohol intake (45%) and Hepatitis B Virus infection (26%). Univariate analysis revealed that diabetes mellitus was associated with an advanced age of > 60 years ($p<0.01$), body mass index (BMI) of ≥30 ($p<0.02$), and alcoholic aetiology ($p<0.02$). After adjusting for confounders during multivariate analysis, only age >60 years (HR, 2.20; 95% CI, 1.40–3.50) and BMI ≥ 30 (HR, 1.60; 95% CI, 1.05–2.40) were associated with DM. During a median follow-up period of 24 (8–26) months, the proportions of hepatic encephalopathy, variceal haemorrhage, and urinary tract infection were higher in patients with LC and diabetes than in those with LC alone. The mean length of hospitalization was higher in the diabetic group (19.5 days) than in the non-diabetic group (15.2 days). **Conclusion:** Diabetes mellitus accounts for one-fourth of all cirrhosis cases. Diabetes increases the likelihood of cirrhosis, complications, and hospital stays among patients with cirrhosis.

INTRODUCTION

Diabetes mellitus (DM) and liver cirrhosis (LC) have long been known to be related and significantly impair the quality of life.[1] Patients with cirrhosis have higher rates of DM than the general population, indicating that cirrhosis is a diabetogenic disease.[2] As per the recent study, the prevalence of DM in individuals with cirrhosis was about 31%, while 6.28% of the world's population was estimated to have DM.[2,3] About 80% of cirrhosis patients have impaired glucose tolerance, while 30% to 60% of patients with severe cirrhosis acquire diabetes.[4]

Diabetes that develops as a consequence of cirrhosis is called hepatogenous diabetes (HD). It is different from type 2 diabetes, in that, it frequently has no family history and is less frequently linked to obesity, has a higher frequency of hypoglycemic episodes,

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and has a lower incidence of micro and macrovascular problems.[4,5] While their significance in the aetiology of HD is uncertain, recent studies suggest that roles of hepatokines, adipokines, gut dysbiosis, hyperammonemia, sarcopenia and myosteatosis have emerged in the pathogenesis of metabolic disturbances in LC, including IR and glucose intolerance, and systemic inflammation all impact glucose control in cirrhosis patients.[6]

Although DM predisposes patients to the progression of liver disease and a higher risk of serious complications of LC, it is evident that LC contributes to dysglycaemia through a variety of mechanisms by interfering with insulin-glucose metabolism. It was assumed that patients with compensated LC and concurrent DM may be more susceptible to decompensating events.[7] In patients with LC, type 2 diabetes mellitus (T2DM), in particular, is linked to negative outcomes, such as a greater hospitalisation rate, a higher prevalence of HCC, and an elevated risk of death.[8]

Similar to DM, HD may be associated with a higher incidence of complications. Hepatic encephalopathy (HE), spontaneous bacterial peritonitis (SBP), variceal haemorrhage (VH), and renal failure are the major complications of cirrhosis associated with diabetes mellitus.[9] However, because the prognosis of patients who develop HD is more likely to be adversely influenced by the underlying hepatic disease and its consequences than by HD itself, the clinical impact of HD on LC has not yet been clarified. Moreover, there is a dearth of information from the Indian subcontinent on how DM affects different aspects of LC and its complications. Therefore, this prospective observational study was conducted to assess the prevalence of DM in patients as well as to investigate the complications and mortality of patients with LC with and without coexisting DM.[10]

MATERIALS AND METHODS

Study settings and selection of participants

This prospective observational study was conducted among patients with liver cirrhosis at the Department of Hepatology, Rajiv Gandhi Government General Hospital, Chennai, between April 2024 and September 2024, after obtaining approval from the institutional ethics committee (registration no. 07102024).

The inclusion criteria included patients with liver cirrhosis aged 18 years and above, who agreed to participate in this study and signed the written consent form. The exclusion criteria were as follows: chronic pancreatitis, hepatocellular carcinoma (HCC), or other neoplasms at the time of enrolment; chronic renal failure; chronic cardiorespiratory illnesses; recent binge alcohol drinking; ongoing steroid therapy; and current pregnancy.

Cirrhosis was diagnosed based on clinical features, imaging characteristics, liver stiffness measurements, and endoscopic findings. All patients were subjected to a detailed clinical history to ascertain the etiological diagnosis of T2DM with and without complications. Furthermore, the diagnosis of T2DM at the time of inclusion was based on the presence of two glycaemia values ≥126 mg/dL or anti-diabetic treatment. In patients with a prior diagnosis of T2DM, information was gathered regarding the onset of T2DM with the diagnosis of cirrhosis and the existence of risk factors for T2DM.

Sample size estimation

The estimated sample size was calculated by using the formula n = (Z 2 × P × (1-P)) / d 2 and factors and influence on survival of type 2 DM in liver cirrhosis done in Spain in 2019.10 With 95% confidence in with the expected prevalence (P) of 30% by Torner M et al,[11] which assessed the prevalence, associated interval and precision of 6% and P = 30%, q = 70%, α = 5% d = 6, 20% relative prevalence = 6%

Formula used:(Zα/2)2 Pq / d2 = (1.96)2x30x70 / 6x6 = 224, hence sample size = 224 cases

Categorisation of patients based on diabetes status

After the inclusion of the patients, two groups were formed: group A consisted of T2DM patients with liver cirrhosis, and group B consisted of liver cirrhosis patients without T2DM to compare the outcomes of survival, hospital stay, and risk factors for the complications associated with it.

Patients' evaluation and follow-up

Demographic information, including age, gender, comorbidities, aetiology of cirrhosis, duration of cirrhosis, and diabetes, and anthropometric parameters, such as hand grip dynamometer, weight, height, and mid-upper arm circumference, were collected at baseline. Estimates of dry weight were made for the corrected BMI calculation by deducting 5%, 10%, or 15% of the actual weight in the case of mild ascites if only ascites is present, moderate ascites with shifting dullness, or severe ascites, respectively, and an additional 5% in the case of pedal oedema.

Liver function tests, complete haemogram, kidney function tests, blood culture, urine culture, urine PCR, routine microscopy, ascitic culture, ascitic fluid analysis when needed clinically, coagulation profile, thyroid function tests, serum lipid profile testing, and fasting and postprandial blood sugar were performed in all patients. In addition, metabolic syndrome (Met-S) was diagnosed using the International Diabetes Federation criteria.10 HCC was diagnosed as per the Indian National Association for the Study of Liver guideline.[11]

Standard medical therapy was provided to all patients who followed up regularly in the outpatient clinic at intervals of 4–6 weeks for decompensated cirrhosis and 12 weeks for compensated cirrhosis, or as needed. Oral hypoglycaemic agents were used for cirrhosis up to Child-Pugh class B, and insulin was considered in all stages of cirrhosis. The rates of cirrhosis complications (HE, VH, SBP, and hepatorenal syndrome-acute kidney injury [HRS-AKI]) and outcomes of the study were assessed for 90 days for complications such as pneumonia, UTI,

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Cellulitis, Acute on chronic liver failure, and death during follow-up were recorded.

The West Haven criteria were used to diagnose the HE. SBP was diagnosed based on a polymorphonuclear leukocyte count in ascitic fluid of > 250 cells/mm3 and/or positive ascitic fluid culture in the absence of an intra-abdominal surgically treatable source. HRS-AKI was diagnosed according to the revised consensus recommendation of the International Club of Ascites.[12] Patients with a minimum follow-up of 3 months were included in the analysis.

Statistical analysis: The data were analysed using the Statistical Package for the Social Sciences (SPSS) software version 23. Categorical data are presented as numbers and percentages, whereas continuous data are presented as mean ± standard deviation. Fisher's exact test or the chi-square test was used to compare categorical variables. To identify independent correlates of the T2DM group about non-diabetes, multivariate regression analysis was performed. For every significant variable in the regression analysis, the odds ratio (OR) and 95% confidence interval (CI) were calculated. Statistical significance was set at $p < 0.05$.

RESULTS

Baseline characteristics: A total of 272 patients with cirrhosis presented during the study period. Of these, 44 patients were excluded before enrolment based on the broad exclusion criteria. Approximately 228 patients with LC were included. Among them, 24% of the LC patients had T2DM, and the remaining 76% of the LC patients had no history of T2DM. The demographic details of the study participants are shown in Table 1. The mean age of the LC patients was 48.5±13.4 years, and the majority of them (84%) were male. The aetiologies of cirrhosis were alcohol intake (45%), followed by hepatitis B virus (HBV) (26%), hepatitis C virus (HCV) (4%), cryptogenic cirrhosis (9%), non-alcoholic steatohepatitis (NASH) (12%), and others (4%). Among LC patients with T2DM, 17% had a family history of diabetes.

Table 1: Baseline demographic characteristics.

Variables	N= 228
Mean Age (±SD) in years	48.5±13.4
Gender, Male n (%)	192 (84)
Mean BMI (±SD) in kg/m2	21.12±3.32
Aetiology n (%)	
Alcohol intake	103 (45)
Hepatitis B	59 (26)
Hepatitis C	9 (4)
NASH	27 (12)
Cryptogenic cirrhosis	21 (9)
Others	9 (4)
Family history of diabetes n (%)	9 (17)

Comparison of clinical characteristics and risk factors between LC patients with T2DM and without T2DM

The mean age of the T2DM group of patients with LC was 46.5 years which was lower than that of the non-diabetic group (49.2 years) and was significant (p=0.004). The duration was also lower in the T2DM group (12.10 months) than in the non-diabetic group (15.2 months). Furthermore, the mean duration of diabetes was 64.26 months which was highly significant. Univariate analysis showed that diabetes mellitus was associated with an advanced age of > 60 years (p<0.01), BMI of ≥30 (p<0.02), and alcoholic aetiology (p<0.02). After adjusting for confounders during multivariate analysis, the only variables associated with T2DM were age >60 years (HR, 2.20; 95% CI: 1.40–3.50) and BMI ≥ 30 (HR, 1.60; 95% CI: 1.05–2.40).

Table 2: Univariate and multivariate logistic regression analyses show an association of risk factors among LC patients with T2DM

Variables	COR (95% CI)	AOR (95% CI)
Age > 60 years	2.15 (1.10–3.40)*	2.20 (1.40–3.50)*
BMI ≥ 30 kg/m2	1.53 (1.10–2.40)*	1.60 (1.05–2.40)*
Alcoholic Aetiology	0.46 (0.12-1.78)*	0.52 (0.13-2.06)

BMI- Body mass Index; COR, crude odds ratio; AOR, adjusted odds ratio; CI- Confidence interval; *Significant P-value (p <0.01)

No significant differences were observed in the clinical characteristics or laboratory investigations between the groups [Table 3]. The mean MELD score for the T2DM group of patients was 19.5, while it was 17.9 in the LC alone group. The mean Child-Pugh score among the T2DM group was 9.65 and 9.48 in the non-diabetic group, where there was a higher proportion of patients in the class C category in both groups. Similarly, there were no major differences in the proportion of oesophageal varices in each group, and the number of patients in the large (grade III-IV) category was higher in both groups (71% and 69%, respectively). Furthermore, the majority of HCC cases were in the non-diabetic group (11.8%) compared to the diabetic group (5.6%). The mean haemoglobin level was slightly lower (8.8 g/dl) in the

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diabetic group than in the non-diabetic group (9.8 g/dl).

Table 3: Comparison of clinical characteristics and risk factors between LC patients with T2DM and without T2DM

Variables	LC + T2DM group (n= 55)	LC alone group (n= 173)	P Value
Age (mean± SD, years)	46.5±10.6	49.2±9.2	0.004
Duration of cirrhosis	12.10±9.23	15.2±10.49	0.007
Duration of DM	64.26±42.36	0	0.0001
MELD scores (13)	19.5±6.2	17.9±7.4	0.487
Child-Pugh score (14)	9.65±2.4	9.48±2.8	0.469
Child-Pugh class (%)			
Class A	14.2	15.3	0.894
Class B	31.6	29.6	
Class C	54.2	55.1	
Oesophageal varices (%)			
None	7	6	0.913
Small (grade I-II)	22	25	
Large (grade III-IV)	71	69	
Ascites (%)	64	71	0.193
HCC (%)	5.6	11.8	0.246
Serum total bilirubin median (range) mg/dl	2.6 (0.3–24)	2.2 (0.6–22)	0.876
Serum albumin (mean± SD, mg/dl)	2.64±0.58	2.71±0.49	0.611
INR (mean± SD)	1.7±0.65	1.7±0.59	0.910
Serum AST median (range) U/L	68.2 (19–498)	69 (20–1958)	0.234
Serum ALT median (range) U/L	32 (7.1–1128)	36.2 (8–1647)	0.568
Serum total protein (mean± SD, mg/dl)	6.06±1.2	6.02±1.41	0.636
Haemoglobin (mean± SD, gm/dl)	8.8±3.3	9.8±4.12	0.568
Total leukocyte count (cells/mm3)	7861.36±2433.11	7451.78±4-620.24	0.696
Platelets count median (range) (x 103 per mL)	89 (24–189)	98 (160–486)	0.309
Ferritin (mean± SD, ng/ml)	232.62±156.71	208.91±183.34	0.256
Serum sodium (mean± SD, mEq/l)	131.2±5.3	132.6±9.2	0.816
Serum potassium (mean± SD, mEq/l)	4.32±1.1	4.2±0.8	0.602
Serum urea median (range) mg/dl	34 (12–142)	28 (8.2–168)	0.705
Creatinine median (range) mg/dl	0.82 (0.5–4.96)	0.86 (0.54–4.9)	0.803
TSH median (range) mIU/L	2.4 (0.06–66)	2.94 (0.4–22)	0.653
Total Cholesterol median (range) mg/dl	81.6 (36–194)	98.5 (49-238)	0.012
HDL-cholesterol median (range) mg/dl	17.5 (2.6–41)	20 (8–40)	0.587
Triglyceride median (range) mg/dl	66 (22–142)	74.7 (22.5–404)	0.182

Complications and outcomes

During a median follow-up period of 24 (8–26) months, the proportions of hepatic encephalopathy, variceal haemorrhage, and UTI were higher in patients with LC and T2DM than in those with LC alone. Variceal haemorrhage (p=0.001), UTI (p=0.001), and ACLF (p=0.004) were significant. No significant differences were observed between the LC+T2DM group and LC alone groups in terms of hepatic encephalopathy, SBP, pneumonia, cellulitis, HRS-AKI, and mortality. The mean length of hospitalisation was higher in the diabetic group (19.5 days) than in the non-diabetic group (15.2 days) [Figure 1].

Table 4: Complications of cirrhosis and mortality rates between LC patients with T2DM and without T2DM

Complications	LC + T2DM group	LC alone group	P Value
Hepatic encephalopathy	18 (32%)	48 (28%)	0.06
Variceal haemorrhage	26 (48%)	61 (35%)	0.001
SBP	3 (6%)	11 (6.4%)	0.04
UTI	10 (19%)	14 (8%)	0.001
Pneumonia	7 (12.6%)	24 (13.8%)	0.53
Cellulitis	6 (10.5%)	14 (8.2%)	0.46
HRS-AKI	9 (16.6%)	20 (11.6%)	0.89
ACLF	19 (34.2%)	57 (32.8%)	0.003
Mortality	14 (24.8%)	49 (28.2%)	0.63

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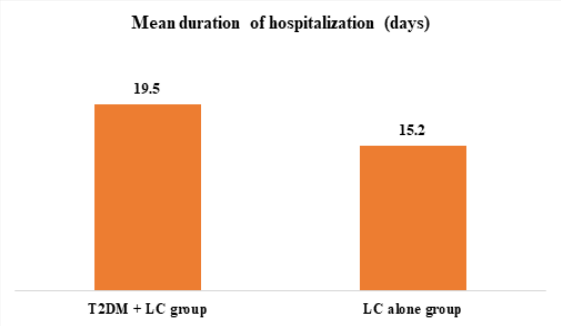

Figure 1: Duration of hospitalization among diabetic and non-diabetic LC patients

DISCUSSION

Diabetes puts people at risk for severe liver illnesses, while cirrhosis is known to cause dysglycaemia through several pathways. Various pathophysiological alterations in cirrhosis, such as neurohormonal changes, gut dysbiosis, systemic inflammation, hypovitaminosis D, hyperammonaemia, and sarcopenia, can affect body glucose regulation.[10] This correlation is more concerning than anticipated because DM has also been shown to significantly affect people with cirrhosis. Diabetes mellitus has been linked to an increased risk of complications and death in cirrhosis.[11]

In our study, DM was noted in nearly one-fourth of patients with cirrhosis. This was in line with a recent systematic analysis of 58 studies encompassing 9705 cirrhosis patients which found a prevalence of DM of about 31%.[2] Several other studies also highlighted the prevalence of diabetes in cirrhosis has been reported in the range of 12.3-57% and shown a high prevalence of liver diseases in diabetic patients and a high prevalence of diabetes in patients with liver disease.[1,12,13] It was noted that the majority of the patients were male and the predominant aetiology was alcohol intake which was similar to Maji T et al. where there were majority of male patients 80% and most of the patients had a history of alcohol intake.[14] Although diabetes mellitus was linked to advanced age (>60 years), BMI ≥30, and alcoholic aetiology when compared to the non-diabetic group, both conditions have similar negative effects on cirrhosis complications and outcomes. It is important to distinguish between HD and T2DM because their pathophysiological differences can have therapeutic implications. Furthermore, Child-Pugh class-C cirrhosis was more common in both the groups of our study which was similar to Maji T et al,[14] In a previous study, cirrhosis patients in Child-Pugh classifications A, B, and C had prevalences of DM of 20.5%, 56%, and 61%, respectively.[15]

In another study, patients with cirrhosis who had high Child-Pugh scores (OR = 1.43) also had diabetes.[16] In particular, there was no difference in the degree of cirrhosis between the diabetic and non-diabetic groups. This suggests that the pathophysiology of HD may be influenced by factors other than cirrhosis severity, as determined by traditional grading systems. The cause of cirrhosis also affects the prevalence of DM in patients with cirrhosis.

There were comparably lower mean albumin and platelet counts. This was similar to Ramachandran TM et al. where they also recorded lower albumin levels compared with the non-diabetic group.[17] This may be related to albuminuria in diabetes, which is more noticeable in cirrhosis.[18] In addition, there were increased levels of ferritin noted in diabetic patients which was also in line with Ramachandran TM et al.[17] As diabetes and cirrhosis were associated with noticeably elevated blood ferritin levels, numerous investigations have described elevated ferritin in diabetes.[19,20] Furthermore, ferritin levels were higher in individuals with chronic hepatitis C infection, according to Lecube A et al. and this was due to concurrent diabetes rather than the virus itself.[21] However, uncertainty surrounds the precise mechanism underlying elevated ferritin levels in diabetes. Ferritin levels are higher in chronic inflammatory illnesses where increased oxidative stress and inflammation are important mechanisms involved in the pathogenesis of T2DM and associated complications.[22]

In this study, a higher proportion of hepatic encephalopathy, variceal haemorrhage, and UTI were noted in patients with LC and T2DM than in those with LC alone. This was in line with previous studies, where a higher frequency of HE, variceal haemorrhage, and UTI was noted in patients with diabetes. In patients with cirrhosis, DM has been associated in multiple studies with an increased incidence, severity, and progression of HE.[23,24] The number of HE events in the HD and T2DM groups did not differ significantly, as shown in our study. In patients with cirrhosis, DM may cause HE through the induction of intestinal glutaminase and gut dysbiosis.[6]

In individuals with cirrhosis, hyperglycaemia can result in splanchnic hyperaemia and increased portal pressure, both of which can increase the risk of variceal haemorrhage.[25] The occurrence of UTI was significantly higher in the diabetic group than in the non-diabetic group, which is in line with a previous study.[17] In addition, ACLF was also slightly higher in the diabetes population than in the LC alone group. Other complications, including mortality, showed no significant differences between the groups. There were a majority of patients in the diabetic group had an increased length of hospitalization than the non-diabetic group which was noted in the previous study.[17] Although this study was among the few that assessed the comparison of prevalence and outcomes among diabetic and non-diabetic LC patients in an estimated sample size, certain limitations.

Limitation

This study was conducted within a limited time interval and captured the complications and outcomes within the desired period. Diabetes and cirrhosis are long-term diseases that require

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prolonged follow-up. Therefore, this study should be conducted in a larger subgroup with long-term follow-up to understand the progression of the disease.

CONCLUSION

The study found that 24% of patients with liver cirrhosis had T2DM. Patients with liver cirrhosis and T2DM were more likely to be older and have a higher BMI. While T2DM was associated with alcoholic cirrhosis aetiology in the univariate analysis, this association was not significant in the multivariate analysis. Patients with liver cirrhosis and T2DM experienced more variceal haemorrhage and UTIs and tended to have longer hospital stays. No significant differences were observed in other complications or mortality between the two groups. Furthermore, the results should be validated in a larger population over a longer term duration of the study period.

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