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Hepatogenous Diabetes and Impaired Glucose Tolerance in Cirrhosis: A Hospital-Based Cross-Sectional Study

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Abstract

Background: Diabetes mellitus is frequently associated with liver cirrhosis as hepatogenous diabetes. Glucose metabolism abnormalities often remain undetected in compensated cirrhosis due to normal fasting glucose levels. Early identification using oral glucose tolerance testing (OGTT) may help detect subclinical disease and assess its relationship with severity of liver dysfunction ***Objective:*** To study the prevalence of impaired glucose tolerance (IGT) and diabetes mellitus (DM) in patients with liver cirrhosis and to evaluate their association with the severity of liver disease. ***Methods:*** This hospital-based cross-sectional observational study was conducted on 60 patients with liver cirrhosis admitted to Sri Guru Ram Das Institute of Medical Sciences and Research, Amritsar, over one year. Diagnosis of cirrhosis was based on clinical, biochemical, and ultrasonographic findings. All patients underwent OGTT and were classified according to ADA 2014 criteria. Severity of cirrhosis was assessed using the Modified Child–Pugh score. Statistical analysis was performed using SPSS version 16. ***Results:*** The mean age of patients was 53.40 ± 13.60 years, with a male predominance (83.3%). Alcoholic liver disease was the most common etiology (81.67%). Based on OGTT results, 41.67% of patients had impaired glucose tolerance and 25% had diabetes mellitus. Overall, 66.67% of patients exhibited abnormal glucose tolerance. A statistically significant association was observed between Child–Pugh class and glucose intolerance ($p < 0.001$), with higher prevalence in advanced cirrhosis. ***Conclusion:*** Glucose metabolism abnormalities are highly prevalent among patients with liver cirrhosis. OGTT is an effective tool for detecting subclinical glucose intolerance, particularly in advanced disease. Routine screening may facilitate early diagnosis and improve clinical outcomes.

Keywords: Liver cirrhosis, Hepatogenous diabetes, Impaired glucose tolerance, Oral glucose tolerance test, Child–Pugh score

INTRODUCTION

Diabetes mellitus is one of the most common chronic diseases in the world affecting about 6.4% of the world population[1]. It comprises a group of common metabolic disorders that share phenotype of hyperglycemia. Many distinct types of diabetes mellitus exist which are caused by complex interactions of genetic, environmental factors and life style. The metabolic dysregulation associated with DM causes secondary pathophysiological changes in multiple organ systems. The vascular complications of DM are retinopathy, neuropathy, nephropathy (microvascular) and coronary heart disease, peripheral arterial disease, cerebrovascular disease (macrovascular)[2].

The term cirrhosis was first introduced by Laennec in 1826. It is a consequence of chronic liver disease characterized by replacement of liver tissue by fibrosis, scar tissue and regenerative nodules, leading to loss of liver function[3].

Modified CHILD PUGH SCORE[4]
The Child Pugh score was originally developed by Child and Turcotte in 1964.

Factor	Units	1	2	3
Serum Bilirubin	mg/dl	<2.0	2.0-3.0	>3.0
Serum Albumin	g/dl	>3.5	3.0-3.5	<3.0
Prothrombin Time	Seconds Prolonged	0-4	4-6	>6
Ascites		None	Easily controlled	Poorly controlled
Hepatic Encephalopathy		None	Minimal	Advanced

Child A = 5–6 points, Child B = 7–9 points, Child C = 10-15 points. This modified Child pugh score quickly became the gold standard tool for predicting the likelihood of survival in patients with cirrhosis.

An association between diabetes mellitus (DM) and liver cirrhosis was first described by Bohan[5]and named as hepatogenous diabetes by Magyesi [5,6]. The liver has an important role in carbohydrate metabolism.Diabetes that develops as a complication of cirrhosis of liver is known as 'Hepatogenous Diabetes[7].

The pathophysiology of hepatogenous diabetes is complex and not precisely known. Insulin resistance in peripheral tissues (adipose and muscular tissue) plays a central role in the glucose metabolism disturbance [8,9,10,11-14]. It has also been proposed that reduced insulin extraction by the damaged liver and portosystemic shunts result in hyperinsulinemia which is potentiated by raised levels of contra-insulin hormones (glucagon, growth hormone, insulin-like growth factor, free fatty acids and cytokines)[10,12,14] due to cirrhosis.

DM in patients with compensated liver cirrhosis may be subclinical, since fasting serum glucose levels may be normal. In these cases, it is necessary to perform an oral glucose tolerance test (OGTT) to detect an impairment of glucose metabolism[15]. In conclusion, Diabetes is commonly associated with liver cirrhosis as hepatogenous diabetes, often remaining undetected in compensated cases due to normal fasting glucose levels. Since impaired glucose metabolism may worsen complications and influence disease severity, this study aims to assess glucose abnormalities in cirrhosis and their relationship with the severity of liver dysfunction.

MATERIALS AND METHODS

Study Design

This was a hospital-based cross-sectional observational study conducted to evaluate abnormalities of glucose metabolism in patients with liver cirrhosis and their association with the severity of liver disease.

Study Setting and Duration

The study was carried out in the Department of Medicine at Sri Guru Ram Das Institute of Medical Sciences and Research (SGRDIMSAR), Sri Amritsar, over a period of one year (specify exact months/year if required). All eligible patients admitted to the medical wards during the study period were screened for inclusion.

Ethical Considerations

Prior to commencement, the study protocol was reviewed and approved by the Institutional Ethics Committee of SGRDIMSAR, Amritsar. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Written informed consent (ICF) was obtained from all participants before enrollment in the study. Confidentiality of patient data was strictly maintained, and participation was voluntary.

Sample Size

A total of 60 patients diagnosed with liver cirrhosis were enrolled after applying predefined inclusion and exclusion criteria.

Study Population

Inclusion Criteria

- Patients admitted in medical wards with a confirmed diagnosis of liver cirrhosis.

Exclusion Criteria

- HbA1c > 6.5%
- Known cases of diabetes mellitus
- Known cases of HIV, HCV, or HBsAg positivity
- Patients receiving insulin, oral hypoglycemic agents, corticosteroids, thiazides, or phenytoin
- Pregnant women

These criteria were applied to eliminate confounding factors that could independently influence glucose metabolism.

Clinical Assessment

All enrolled patients underwent:

- Detailed history taking using a structured proforma (including demographic details, duration of liver disease, alcohol intake, drug history, and associated comorbidities).
- Comprehensive general physical examination.
- Systemic examination with emphasis on signs of chronic liver disease such as jaundice, ascites, splenomegaly, and hepatic encephalopathy.

Diagnosis of Liver Cirrhosis

The diagnosis of liver cirrhosis was established based on a combination of:

1. Clinical features suggestive of chronic liver disease.
2. Biochemical abnormalities on liver function testing (elevated bilirubin, hypoalbuminemia, prolonged prothrombin time).
3. Ultrasonographic findings consistent with cirrhosis (coarse echotexture, nodular liver surface, features of portal hypertension, splenomegaly, ascites).

Laboratory Investigations

All patients were subjected to the following investigations:

1. Complete Blood Count (Hb, TLC, DLC)
2. Prothrombin Time Index (PTI)
3. Liver Function Tests (bilirubin, AST, ALT, ALP, serum albumin)
4. Total Serum Protein

5. Viral markers (HBsAg, Anti-HCV, HIV)
6. Ultrasonography (USG) abdomen
7. Oral Glucose Tolerance Test (OGTT)

Oral Glucose Tolerance Test (OGTT)

Procedure

- Patients were advised overnight fasting for 8–10 hours.
- Fasting plasma and urine samples were collected.
- A 75 g oral glucose load dissolved in 300 ml of water was administered over 5 minutes.
- Blood and urine samples were subsequently collected at 30 minutes, 1 hour, 2 hours, and 2.5 hours post-glucose load (minimum at 1 and 2 hours).
- Timing of sample collection was documented accurately.
- Plasma glucose estimation was performed using Trinder's enzymatic method.

Interpretation (ADA 2014 Criteria)

- <140 mg/dl (2-hour value): Normal glucose tolerance
- 140–199 mg/dl: Impaired glucose tolerance
- ≥200 mg/dl: Provisional diagnosis of diabetes mellitus

Patients were classified accordingly.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 16.

- Continuous variables were expressed as mean ± standard deviation (SD).
- Categorical variables were expressed as frequencies and percentages.
- Chi-square test and Student's t-test were applied where appropriate.

A p-value <0.05 was considered statistically significant

RESULTS

Baseline Characteristics

Age Distribution

A total of 60 patients with liver cirrhosis were included in this cross-sectional period prevalence study. The mean age of the study population was **53.40 ± 13.60 years**. The majority of patients (63.34%) belonged to the 41–65 years age group. The age distribution of participants is shown in **Table 1**.

TABLE 1: Age distribution of participants

Age group	No. of cases	Percentage
25-40	11	18.33
41-65	38	63.34
66-85	11	18.33
Total	60	100.00

Gender Distribution

Of the 60 patients, **50 (83.3%) were males** and **10 (16.7%) were females**, showing a marked male predominance. The gender distribution is illustrated in **Figure 1**.

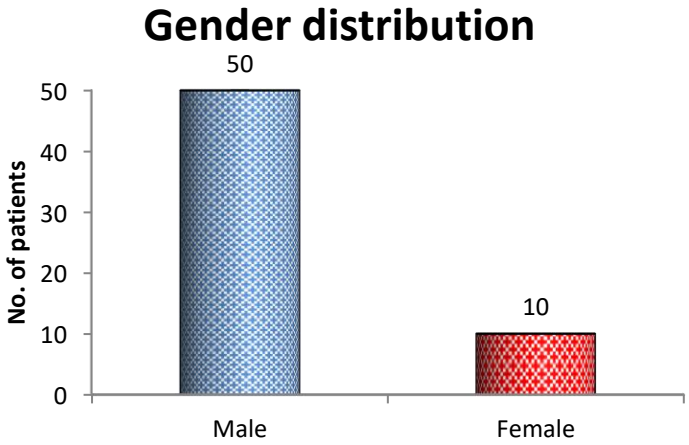

Figure 1: Gender distribution of participants

Etiology of Cirrhosis

Alcoholic liver disease (ALD) was the most common etiology, accounting for **49 patients (81.67%)**. Ten patients (16.67%) had cirrhosis of other/unknown causes, while only one patient (1.67%) had NASH-related cirrhosis. The distribution of glucose tolerance status according to etiology is presented in **Table 2**.

Table 2: Distribution of Glucose Tolerance Status According to Etiology

	NGT	IGT	DM	Total
ALD	17 (28.33%)	21 (35%)	11 (18.33%)	49(81.67%)
NASH	1(1.67%)	0	0	1(1.67%)
Others	2 (3.33%)	4 (6.67%)	4 (6.67%)	10(16.67%)
Total	20	25	15	60

Severity of Cirrhosis

Patients were categorized according to the Modified Child–Pugh Score into three classes.

- Class A: 16 patients (26.67%)
- Class B: 23 patients (38.33%)
- Class C: 21 patients (35%)

The largest proportion belonged to Child–Pugh Class B. The distribution is shown in **Figure 2**.

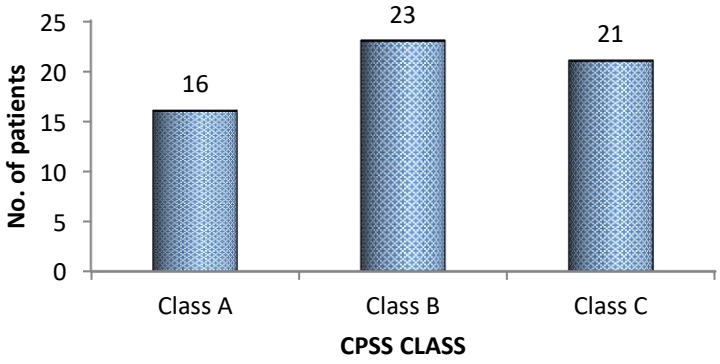

Figure 2: Distribution of Patients According to Modified Child–Pugh Class

Prevalence of Glucose Intolerance

Based on Oral Glucose Tolerance Test (OGTT) results:

- **Normal Glucose Tolerance (NGT):** 20 patients (33.33%)
- **Impaired Glucose Tolerance (IGT):** 25 patients (41.67%)
- **Diabetes Mellitus (DM):** 15 patients (25.00%)

Thus, **40 patients (66.67%)** had abnormal glucose tolerance (IGT + DM). The distribution is shown in **Table 3**.

Table 3: Distribution of Participants Based on OGTT Results

OGTT	Number of patients	Percentage
NGT	20	33.33%
IGT	25	41.67%
DM	15	25%
Total	60	100%

Association Between Child–Pugh Class and Glucose Tolerance

For analytical purposes, patients were divided into:

- **Group 1:** Child–Pugh Class A (n = 16)
- **Group 2:** Child–Pugh Class B and C combined (n = 44)

Similarly, glucose tolerance was categorized as:

- Normal glucose tolerance
- Abnormal glucose tolerance (IGT + DM)

The distribution is shown in **Table 4**.

A statistically significant association was observed between Child–Pugh class and glucose intolerance ($p < 0.001$), with abnormal glucose tolerance being more frequent in advanced cirrhosis (Class B and C).

Table 4: Distribution of Glucose Tolerance According to Child–Pugh Class

OGTT	Group 1 (Child Pugh Class A)		Group 2 (Child Pugh Class B + Class C)		Total	
	No. of cases	%age	No. of cases	%age	No. of cases	%age
Normal glucose tolerance	13	21.66	7	11.66	20	33.33
Abnormal glucose tolerance (Patients with IGT and with DM)	3	5.00	37	61.67	40	66.37
Total	**16**	**26.66**	**44**	**73.33**	**60**	**100.00**

DISCUSSION

The relationship between chronic liver disease and abnormalities of carbohydrate metabolism has long been recognized. The liver occupies a central role in glucose homeostasis, and hepatic dysfunction leads to insulin resistance, impaired glucose uptake, and altered insulin metabolism, resulting in glucose intolerance and diabetes mellitus8. 7,8,16 Hepatogenous diabetes is now considered a distinct clinical entity occurring as a complication of cirrhosis and differs from classical type 2 diabetes mellitus.17,18

In the present cross-sectional study, 60 cirrhotic patients (mean age 53.40 ± 13.60 years; 83.3% males) were evaluated using the oral glucose tolerance test (OGTT). The prevalence of impaired glucose tolerance (IGT) was found to be 41.67%, while 25% of patients were diagnosed with overt diabetes mellitus. Thus, 66.67% of the study population demonstrated abnormal glucose metabolism. These findings are consistent with earlier reports that indicate that 30–60% of cirrhotic patients develop overt diabetes and up to 80% may exhibit glucose intolerance. 12,19-21

The prevalence of diabetes in our study (25%) is comparable to that reported by Kobashi-Margáin et al.22, who observed diabetes in 23.2% of cirrhotic patients. Similarly, García-Compeán et al.23 reported IGT and DM prevalence of 38.5% and 21.5%, respectively, which closely approximate the findings of the present study. Müller et al.24 documented IGT in 36% and diabetes in 37% of cirrhotic patients. Although the prevalence of diabetes in their study was higher than ours, the proportion of IGT remains comparable.

In contrast, Obang et al.25 reported a lower prevalence of diabetes (12.9%) among chronic liver disease patients. Sukumaran et al.26 observed IGT in 34% and diabetes in only 11% of cases. These variations may be explained by differences in study design, patient selection, etiological spectrum, and diagnostic methods employed.

On the other hand, Ennaifer et al.27 reported a higher prevalence of diabetes (42.8%) with lower IGT (26%) among cirrhotic patients. Such discrepancies across studies likely reflect regional differences, underlying etiologies of cirrhosis, and heterogeneity in study populations.

Alcoholic liver disease (ALD) constituted 81.67% of cases in our study. Chronic alcohol consumption has been shown to increase the risk of diabetes in patients with liver disease. 28,29 Alcohol contributes to hepatic insulin resistance and may also induce pancreatic β-cell dysfunction, thereby aggravating glucose metabolism abnormalities.7 These mechanisms may partly explain the high prevalence of glucose intolerance observed in our cohort.

An important observation of this study is the high detection rate of glucose abnormalities using OGTT. Previous studies have demonstrated that fasting plasma glucose alone may fail to identify a significant proportion of cirrhotic patients with impaired glucose tolerance.30 Nishida et al. showed that OGTT is superior in detecting subclinical glucose abnormalities and also serves as a predictor of prognosis in cirrhotic patients.15 Therefore, OGTT remains a sensitive and valuable diagnostic tool in evaluating carbohydrate metabolism disturbances in cirrhosis.

The pathophysiology of hepatogenous diabetes is complex and multifactorial. It involves peripheral insulin resistance, reduced hepatic insulin extraction, hyperinsulinemia, and impaired pancreatic β-cell response.12,13, Unlike classical type 2 diabetes mellitus, hepatogenous diabetes is less frequently associated with microvascular complications but is closely linked to hepatic dysfunction and disease progression.16,31

Furthermore, in our study, abnormal glucose tolerance increased with advancing Child–Pugh class ($p < 0.001$), indicating that worsening hepatic dysfunction correlates with increasing impairment of glucose metabolism. Similar findings were reported by Mukherjee et al.32 and Sukumaran et al.26, who observed a higher prevalence of hepatogenous diabetes in advanced stages of cirrhosis. Nishida et al. also demonstrated poorer survival in

cirrhotic patients with diabetes compared to those with normal glucose tolerance.15

Overall, the present study demonstrates a high prevalence of impaired glucose tolerance and diabetes mellitus among patients with liver cirrhosis. These findings support routine screening using OGTT for early detection of hepatogenous diabetes, particularly in patients with advanced liver disease. Early identification and management of glucose metabolism abnormalities may have important implications for clinical outcomes in cirrhotic patients.

Limitations

The present study has certain limitations that should be acknowledged. It was conducted at a single tertiary care center, which may limit the generalizability of the findings to the broader population. The relatively small sample size of 60 patients may reduce the statistical power and precision of prevalence estimates. As the study design was cross-sectional, it does not allow for establishment of causal relationships between severity of cirrhosis and abnormalities in glucose metabolism. Additionally, long-term follow-up data were not available to evaluate the prognostic impact of impaired glucose tolerance or diabetes mellitus on morbidity and mortality. Insulin levels and indices of insulin resistance, such as HOMA-IR, were not assessed, thereby limiting deeper pathophysiological interpretation. Furthermore, exclusion of patients with viral hepatitis may restrict the applicability of results to settings where viral etiologies constitute a major proportion of cirrhosis.

Future Directions

Future research should involve larger multicentric studies to validate these findings across diverse populations and etiological profiles of cirrhosis. Prospective longitudinal studies are needed to assess the long-term prognostic significance of hepatogenous diabetes and impaired glucose tolerance in relation to liver-related complications and survival outcomes. Detailed evaluation of insulin resistance markers and pancreatic β-cell function may provide further insight into the mechanisms underlying glucose metabolism disturbances in cirrhosis. Comparative studies evaluating fasting plasma glucose, HbA1c, and oral glucose tolerance test in cirrhotic patients could help refine optimal screening strategies. Additionally, interventional studies exploring whether early detection and appropriate glycemic management improve clinical outcomes in cirrhotic patients would be valuable. Integration of glucose metabolism parameters into existing prognostic scoring systems may also enhance risk stratification and clinical decision-making.

CONCLUSION

The present study was conducted on 60 cases (50 males and 10 females) of cirrhosis of liver of various etiologies, majority (49 cases) of which were of alcoholic liver disease. The prevalence of impaired glucose tolerance was found to be 41.67% and that of diabetes mellitus was 25%. Moreover, there was a significant correlation between child pugh scores and results of OGTT defining the role of glucose tolerance in prognosticating cases of cirrhosis of liver.

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