“A PROSPECTIVE STUDY TO IDENTIFY THE PREVALENCE OF IMPAIRED GLUCOSE TOLERANCE IN PREVIOUS UNDIAGNOSED DIABETES IN CIRRHOSIS PATIENTS USING ORAL GLUCOSE TOLERANCE TEST”

BY

DR.VEERAUMREDDY OBUL REDDY

Dissertation submitted to

BLDE (Deemed to be University) Vijayapur, Karnataka

In partial fulfillment of the requirements for the degree of

DOCTOROFMEDICINE IN

GENERAL MEDICNE

Under the guidance of

Dr. VJAYAKUMAR WARAD

PROFESSOR

DEPARTMENTOFGENERAL MEDICNE

BLDE (Deemed to be University)

SHRIB.M.PATILMEDICALCOLLEGE

HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

KARNATAKA

2020

"A PROSPECTIVE STUDY TO IDENTIFY THE PREVALENCE OF IMPAIRED GLUCOSE TOLERANCE IN PREVIOUS UNDIAGNOSED DIABETES IN CIRRHOSIS PATIENTS USING ORAL GLUCOSE TOLERANCE TEST"

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

LIST OF ABBREVIATIONS USED

OGTT	-	Oral Glucose Tolerance Test
IGT	-	Impaired Glucose Tolerance
NGT	-	Normal Glucose Tolerance
IDDM	-	Insulin Dependent Diabetes Mellitus
NIDDM	-	Non-Insulin Dependent Diabetes Mellitus
GLP 1	-	Glucagon Like Peptide 1
GDM	-	Gestational Diabetes Mellitus
FPG	-	Fasting plasma glucose
H/o	-	History of
CLD	-	Chronic Liver Disease
ICMR	-	Indian Council of Medical Research
DPP-4	-	Serum dipeptidyl peptidase-4
IFG	-	Impaired Fasting Glucose
AGE	-	Advanced glycation End Products
FFA	-	Free Fatty Acids
CHD	-	Congenital Heart Disease
WHO	-	World Health Organisation
SD	-	Standard deviation
DCCT	-	Diabetes Control and Complications Trial

ABSTRACT

The importance of undiagnosed diabetes mellitus (DM) or impaired glucose tolerance in the pathogenesis of hyperglycemia in liver disease is still not well-established. Diabetes mellitus is a global health problem that may affect the prognosis by interfering with the various metabolic function of the body. Hence it is important to know the glycemic status of the patients with liver cirrhosis to anticipate and treat the complications associated with it which in turn will help in the prognosis.

The objective of my study is to perform fasting glucose value and 2-hour OGTT in cirrhosis patients and determine their glycemic status. This is a prospective cross-sectional study. Cases are taken from liver cirrhosis patients between ages 20-80 years, and selected from patients attending inpatient and outpatient departments at our hospital. The sample size is 85. The period of study was from January 2021 to June 2022.

Patients were tested with an oral glucose tolerance test and results along with various laboratory values and demographic details were recorded. The results show a statistically significant increase of 17.6% in impaired glucose tolerance and diabetes mellitus in the 85 patients with liver cirrhosis which is significant.

Our study concluded that performing OGTT to find the prevalence of impaired glucose tolerance and diabetes mellitus in liver cirrhosis patients will help in improving the prognosis of the disease. The glycemic status of the patient may help in deferring the complications associated with poor glucose control. Incidence of the potential complications of Diabetes mellitus can be reduced by prompt identification and treatment.

TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

The liver is a key component of the body's glucose metabolism. The liver plays a crucial role in the consumption of glucose during the post-prandial period. Glucose enters the hepatocyte and is phosphorylated to glucose 6-phosphate. The pentose phosphate pathway, the hexosamine pathway, and the glycogen synthesis pathway are a few examples of metabolic activities where glucose 6-phosphate enters. production of fatty acids, etc. [1]

The human liver not only uses glucose but also makes glucose from alanine, lactate, and glycerol to release into the bloodstream. Glycogenolysis is the name given to this process. Therefore, changes to the design and function of the liver will affect how the body processes glucose. [2]

Since the liver is a key location for glucose metabolism, changes in carbohydrate/glucose metabolism are seen in cirrhotic patients. Hepatogenous diabetes (HD) is the term used to describe diabetes mellitus (DM) brought on by chronic liver disease (CLD) (HD). Chronic liver disease frequently masks diabetes and poor glucose tolerance.[3]

Normal fasting glucose levels are typical in patients with hepatogenous diabetes (HD), but the oral glucose tolerance test result is abnormal, which is necessary for the diagnosis. Glycated haemoglobin will not help in the initial diagnosis of hepatogenous diabetes since it is frequently deceptively low in cirrhotic patients. HD is linked to a higher likelihood of cirrhosis complications, 5-year survival rate, and a higher incidence of hepatocellular cancer. Hepatic encephalopathy (HE), spontaneous bacterial peritonitis (SBP), sepsis, esophageal variceal bleeding, and renal failure are the main consequences of cirrhosis linked with HD.

Peripheral insulin resistance and -cell dysfunction in cirrhotic individuals are the result of a variety of causes. Hyperinsulinemia is one of the contributing factors. In cirrhotic patients, the liver extracts less insulin due to a reduction in liver cell mass and the existence of portosystemic collaterals, which causes systemic hyperinsulinemia. Hyperinsulinemia is also a result of an excessive insulin secretion, which has recently been seen in cirrhotic individuals as a result of pancreatic islet hypertrophy. Insulin resistance can result from hyperinsulinemia by reducing the activity of target cells' insulin receptors. [4]

The two incretin hormones that are produced naturally are glucagon like peptide (GLP-1). Glucagon secretion is suppressed while insulin secretion is stimulated by the incretin hormone GLP-1. Impairment in glucose tolerance will result from GLP inactivation. The hydrolysis of GLP-1 by serum DPP-4 renders it inactive. DPP-4 is upregulated in cirhhotic patients, which inhibits GLP and lessens the effects of incretins.

The primary catabolic site for these advanced glycation end products (AGEs), which are produced as a result of hyperglycemia, is the liver. Serum levels of AGEs are significantly higher in cirrhosis patients and are correlated with the higher risk of the liver disease. The AGE's are believed to cause -cell damage and also insulin resistance. [5]

Numerous investigations have revealed that diabetic cirrhotic patients have a bad prognosis. Therefore, it is crucial to detect diabetes or decreased glucose tolerance in cirrhotic patients in order to anticipate how well the condition would progress. [6]

The main goal of this study is to use an oral glucose tolerance test to determine the prevalence of impaired glucose tolerance and previously undetected diabetes in people with cirrhosis of the liver.

AIMS AND OBJECTIVES

AIM:

The aim is to identify the prevalence of impaired glucose tolerance in cirrhosis patients using oral glucose tolerance test (OGTT).

OBJECTIVE:

To perform fasting glucose value and 2 hour OGTT in cirrhosis patients and determine their glycemic status.

REVIEW OF LITERATURE

Type 2 diabetes is a serious condition that affects people all over the world and is currently experiencing an epidemic in both industrialized and developing nations. According to the International Diabetes Federation, 463 million persons worldwide already have diabetes.

Diabetes mellitus: an overview

Diabetes mellitus are characterized by hyperglycemia caused by deficiencies in insulin secretion, insulin action. When one of the three requirements is satisfied, the American Diabetes Association Expert Panel advises a diagnosis of diabetes mellitus.

A pandemic of diabetes exists worldwide. Globally, the prevalence of diabetes has increased due to changing lifestyles and rising obesity. 425 million people worldwide had diabetes in 2017. About 10% of Americans had diabetes in 2015, according to the International Diabetes Federation (IDF). Of these, 7 million patients lacked a diagnosis. As people age, diabetes becomes more common. In people over 65, diabetes affects about 25% of the population. [7]

Criteria for the diagnosis of Diabetes Mellitus [6]

1) Diabetes symptoms along with an unexpected plasma glucose level of more than 200 mg/dl.

Casual is defined as occurring at any time of day, regardless of when one lasts had a meal. Diabetes is characterised by polyuria, polydipsia, and unexplained weight loss.

2) A 126 mg/dl fasting plasma glucose level. A minimum of 8 hours without consuming any calories is considered a fast.

3) During an oral glucose tolerance test, 2 hours plasma glucose 200 mg/dl.

DIABETES MELLITUS CLASSIFICATION

The new classification separates diabetes mellitus into four categories. 8 Pregnancy-related diabetes, Type 1, Type 2, and "Other Specific Types"

Type 1: (Formerly IDDM/Juvenile Onset DM) is defined by the complete lack of insulin caused by -cell death brought on by an autoimmune mechanism.

Type 2: (NIDDM/Adult onset) is characterised by peripheral insulin resistance and a malfunction in the insulin secretory pathway in the beta cells.

Individuals with hereditary impairments of beta-cell activity (MODY, or Maturity Onset Diabetes Mellitus), or with problems of insulin action, are included in the category of other specific types. individuals with endocrinopathies and illnesses of the exocrine pancreas.

SECONDARY

- Pancreatic disease
- Hormonal abnormalities
- Genetic diseases
- Ingestion of certain drugs or chemical compounds.

Pathophysiology

Beta-cell dysfunction and insulin resistance are both symptoms of T2DM. Initially, an increase in insulin secretion occurs as compensation, which maintains glucose levels within normal ranges. Hyperglycemia results from beta cells shifting as the disease progresses and insulin production failing to keep glucose homeostasis. Most T2DM patients are overweight or have a higher body fat percentage, which is mostly focused in the abdominal region. Increased FFA release and dysregulated adipokines are just two of the inflammatory processes by which adipose tissue causes insulin resistance.

The likelihood of developing T2DM is increased by inactivity, prior GDM in people with hypertension or dyslipidemia, and prior GDM in people with hypertension or dyslipidemia. Adipokine dysregulation, inflammation, altered incretin biology (decreased levels of incretins such glucagon-like peptide-1 (GLP-I) or incretin resistance), hyperglucagonemia, increased renal glucose reabsorption, and changes in the gut flora are also factors.

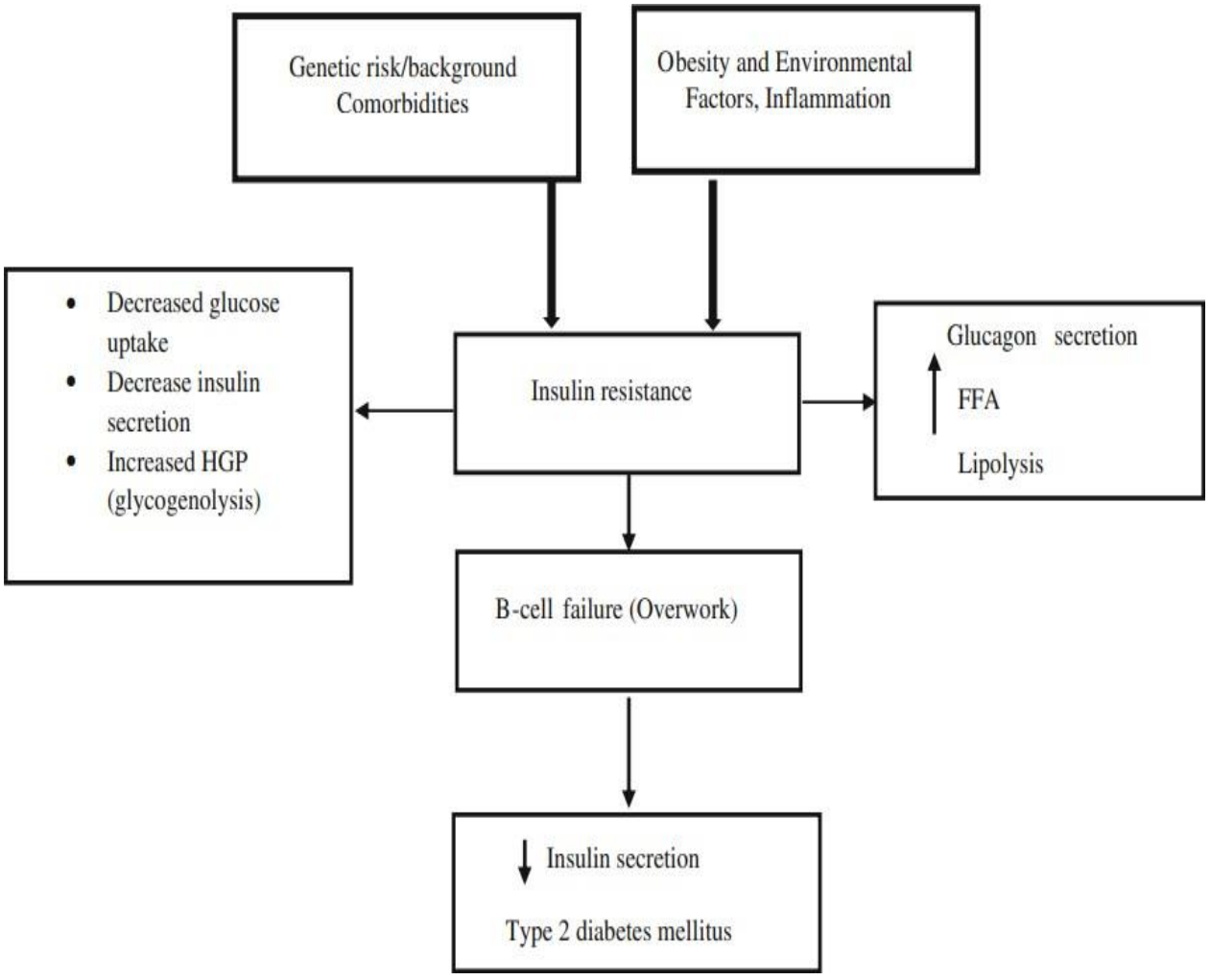

Figure 1: Mechanism of genetic predisposition and obesity induced insulin resistance and T2DM

For beta cells to release insulin, glucose transfer into the cell is necessary, which is mostly handled by the glucose transporter 2. (GLUT-2). A genetically modified mouse model that altered the expression of GLUT-2 resulted in mice with glucose intolerance; comparable changes in GLUT-2 could be produced in normal mice fed a high-fat diet, suggesting a possible mechanism linking a high-fat diet to the development of diabetes.

T2DM is characterized by

- Adipocyte insulin resistance causes an increase in plasma free fatty acids. Hepatic insulin resistance causes an increase in glucose production. Muscle insulin resistance causes a decrease in glucose uptake. Increased insulin resistance stimulates the release of adipocytokines. Progressive beta-cell failure.
- Increased hepatic sensitivity to glucagon Hyperglucagonemia Impaired incretin effect (GLP-1 and GIP).
- Increased renal glucose reabsorption Brain neurotransmitter dysfunction leading to failure of appetite suppression resulting in weight gain

Risk factors

- Obesity
- Fat distribution
- Inactivity
- Family history
- Ethnicity and race
- Hypertension
- Dyslipidemia
- Old age

- Pre-diabetes
- Pregnancy related risk (history of GDM)
- Insulin resistance

Table 1: Major causes for insulin resistance
Inherited states of target cell resistance
Leprechaunism (insulin-receptor mutations)
Rabson-Mendenhall syndrome (insulin-receptor mutations)
Type A syndrome of insulin resistance (insulin-receptor mutations in some, unknown signaling defect in most)
Some lipodystrophies
Secondary insulin resistance
Obesity (free fatty acids and adipocytokines may contribute)
Stress, infection due to excess counterregulatory hormones (cortisol, catecholamines, growth hormone, glucagon)
Medications (eg, glucocorticoids, HIV antiretrovirals, oral contraceptives)
Inactivity
Pregnancy (placental lactogen)
The immune system (anti-insulin antibodies, anti-insulin receptor antibodies in type B syndrome)

Miscellaneous (starvation, uremia, cirrhosis, ketoacidosis)
Consequences of insulin resistance
Most cases of type 2 diabetes mellitus
Cardiovascular disease, hypertension
PCOS
Metabolic syndrome
Obesity-related cancers

- An excessive amount of circulating fatty acids is a crucial early factor in the development of insulin resistance (Fig. 422-2). The majority of serum albumin bound free fatty acids come from adipose tissue's intracellular lipolytic triglyceride reserves. Lipoprotein lipase, which catalyzes the breakdown of triglyceride-rich lipoproteins in tissues, also produces fatty acids. Insulin is involved in stimulating lipoprotein lipase and inhibiting lipolysis in adipose tissue. Increased lipolysis causes more fatty acids to be released as insulin resistance increases, thus decreasing insulin's antilipolytic impact. Excess fatty acids affect downstream signalling to boost substrate supply while simultaneously producing insulin resistance. While the liver increases glucose synthesis and triglyceride formation, Fatty acids accumulate as triglycerides in skeletal and cardiac muscle and prevent insulin-mediated glucose uptake.
- Another putative pathophysiologic cause for the metabolic syndrome is leptin resistance. Leptin increases energy expenditure, boosts insulin sensitivity, and decreases hunger. Through a nitric oxide-dependent mechanism, leptin can also have an impact on vascular and cardiovascular health. On the other hand, obesity causes hyperleptinemia, which then causes inflammation, insulin resistance,

hyperlipidemia, and a variety of cardiovascular diseases, such as CHD, atherosclerosis, hypertension, and heart failure.

- The oxidative stress theory can be used to explain both the tendency toward metabolic syndrome and ageing. A lack of mitochondrial oxidative phosphorylation is linked to the accumulation of triglycerides and other associated lipid molecules in muscle, according to studies of insulin resistant individuals with obesity or type 2 diabetes, children of type 2 diabetes patients, and the elderly.

Oral glucose tolerance test (OGTT)

Oral glucose tolerance test (OGTT) is test used to determine the body capacity to tolerate the oral glucose load which might help to diagnose the person with diabetes mellitus or impaired glucose tolerance.

Procedure

The OGTT is carried out meticulously, and patients are given instructions. Patients are asked to arrive at 8 am so that a blood sample can be taken while they are fasting. A sample of urine is also taken. This is a sampling from the first hour.

75g of anhydrous glucose (82.5g of glucose monohydrate) in 250–300ml of water is the dosage for a glucose load. The user is instructed to drink slowly and finish within 5 minutes in order to prevent vomiting. It is also possible to flavour the solution to lessen the likelihood of nausea.

According to the WHO, two samples should be taken at the 0th and 2nd hours after a glucose load.

INDICATIONS FOR ORAL GLUCOSE TOLERANCE TEST

- During pregnancy
- Excessive weight gain or weight loss

- History of miscarriage in females
- Insulin resistance clinical signs
- Reactive hypoglycemia

CONTRAINDICATIONS FOR ORAL GLUCOSE TOLERANCE TEST:

- If person is confirmed diabetes mellitus
- Follow-up of diabetes mellitus
- Acutely ill patients

Preparation of patient

- The patient is instructed to have good carbohydrate diet for 3 days prior to the test. further, The evening before the test, eat a meal with between 30-50 gms of carbohydrates.
- Patients should abstain from smoking throughout the examination, and strenuous exercise the day before should be avoided.
- Patients should avoid medicines likely to affect blood glucose levels for at least 2 days before to the testing.
- Patients shouldn't consume the food after 8 p.m. the night before. you shouldn't eat breakfast.
- The patient is asked to stay in the hospital for the whole. two-hour waiting time without engaging in any strenuous activity.

Criteria

Table 2: American diabetes association criteria for the diagnosis of diabetes

1. HBA1C ≥6.5%. The test should be carried out in a lab with an NGSP-certified methodology and standardized to the DCCT assay. **OR**
2.126 mg/dL (7 mmol/L) FPG. Having no calorie intake for at least eight hours is considered fasting **OR**
3. During an OGTT, the 2-hour plasma glucose was under 200 mg/dL (11.1 mmol/L). The test should be performed with a glucose load equivalent to 75 g of anhydrous glucose dissolved in water, according to the World Health Organization.
4. When a patient has the typical hyperglycemia symptoms and signs, a random plasma glucose level of less than 200 mg/dL (11.1 mmol/L) is considered to be hyperglycemia.

FPG between 5.6 and 6.9 mmol/L (100 to 125 mg/dL) - IFG
140 to 199 mg/dL (7.8 to 11.0 mmol/L) with the 75 g OGTT at two hours after the load - IGT
5.7 to 6.4% (39–46 mmol/mol) HBA1C

Factors affecting the glucose tolerance test

- Insulin levels

- Carbohydrate starvation
- Exercise
- Liver disease
- Acute infections
- Hyperthyroidism

Impaired oral glucose tolerance in Liver cirrhosis [9]

Impaired glucose tolerance (IGT) and resistance of insulin are prevalent in people with chronic liver disease, it has been known since the late 1960s. 15% to 30% of people with liver cirrhosis have frank glycosuria, overt diabetes, and fasting hyperglycemia. [10] ,[11]

Hepatogenous diabetes, in which diabetes is brought on by chronic liver illness, and type 2 diabetes mellitus (DM), in which diabetes is brought on by or coexists with chronic liver disease. Two different chronic liver diseases are associated with diabetes. The pathophysiology of non-alcoholic steatohepatitis, which results in cirrhosis, has recently been linked to DM. Additionally, the use of corticosteroids, interferons, and other medications to address specific disorders. [9,12] In addition, hyperglycemia may result from the use of corticosteroids, interferons, and other medications to treat chronic hepatitis. [9] IGT and chronic liver disease are thought to interact, one of which worsens the other's condition over time. [13]

Mechanism of glucose regulation in liver cirrhosis

By storing glucose and generating endogenous glucose from the liver's glycogen stores, the liver plays a crucial part in controlling blood sugar levels. Skeletal muscle plays a crucial role in maintaining the balance of glucose in the body. Postprandial hyperglycemia in type 2 diabetes patients is typically brought on by increased endogenous glucose synthesis and decreased skeletal muscle glucose deposition. Over the course

of the illness, hepatocyte mass and skeletal muscle are lost, and early-stage hepatogenous diabetes patients with liver cirrhosis are characterised by severe postprandial hyperglycemia and enhanced insulin resistance. [9], [14], [15]

Numerous investigations have demonstrated that although insulin-stimulated glucose absorption into skeletal muscle is reduced in people with liver cirrhosis, enhanced insulin activity still restricts hepatic glucose synthesis. [16] Additionally, a portosystemic shunt or a reduced hepatocyte bulk in individuals with cirrhosis can promote hyperinsulinemia and the development of insulin resistance through insulin receptor downregulation. Increased insulin resistance eventually leads to overt diabetes, which raises the demand for pancreatic insulin secretion. Although the actual molecular explanation of cirrhotic individuals' lower insulin activity in their skeletal muscles is unknown, it may be influenced by their level of liver function.[18], [19]

Loss of skeletal muscle and impairment of liver function are two common pathophysiological symptoms of liver cirrhosis. The clinical values of patients with CLD are negatively impacted by skeletal muscle loss, particularly due to malnutrition. [20]

The precise method of action is unknown, though. [21] In people with liver cirrhosis, a lack of glucose absorption into both hepatic tissue and skeletal muscle leads to impaired glucose homeostasis. Furthermore, because of anomalies in the metabolism of glucose in skeletal muscle, type 2 diabetes is known to be a risk factor for chronic liver disease [22] [23]. In the early stages of the disease, cirrhosis and diabetes are both asymptomatic; hence, they can reinforce one another in a vicious cycle that decreases glucose metabolism and liver function during the usual course of liver cirrhosis. In cirrhotic people with overt DM, it is challenging to distinguish between conventional and hepatogenous diabetes. [9]

Due to the higher red blood cell turnover caused by hypersplenism, Kanda et al.24 found that individuals with cirrhosis and diabetes had lower HbA1c values than those with type 2 diabetes. Because HbA1c is a poor predictor of diabetes in individuals with cirrhosis, the American Diabetes Association advises that only

blood glucose criteria be used to diagnose diabetes in people with conditions linked to rapid red cell turnover. Indeed, we recently discovered that diabetic cirrhotic individuals had an average HbA1c level of 5.7 percent, which is within the normal range and emphasises the tendency for HbA1c to be lower in cirrhotic patients.

However, multiple articles have criticised the use of the 126 mg/dL FPG cutoff as the gold standard for diagnosing diabetes in cirrhotic patients. As we shown in 1999, the FPG level is insufficient for identifying diabetes in persons with cirrhosis. Cirrhotic individuals' skeletal muscles and liver have been proven to develop insulin resistance, which may cause postprandial glucose levels to rise more sharply. Due to the higher red blood cell turnover caused by hypersplenism, patients with cirrhosis and diabetes exhibited lower HbA1c values than those with type 2 diabetes.

Because HbA1c is a poor predictor of diabetes in individuals with cirrhosis, the American Diabetes Association advises that only blood glucose criteria be used to diagnose diabetes in people with conditions linked to rapid red cell turnover. Indeed, we recently discovered that diabetic cirrhotic individuals had an average HbA1c level of 5.7 percent, which is within the normal range and emphasizes the tendency for HbA1c to be lower in cirrhotic patients. Hyperglycemia associated with liver cirrhosis is included in the diagnostic criteria for gestational diabetes, as well as the classification and diagnosis of diabetes based on the same FPG cutoff threshold. [9,13]

HbA1c was introduced by Koga et al., [26] who used the average of measured HbA1c and glycated albumin/3 to quantify chronic liver disease. Matsumoto et al., [27] recently found that hypoalbuminemia and increased ICGR-15 levels were independent risk factors for DM in cirrhotic patients with FPG values of 126 mg/dL.

This study raises the possibility that cirrhotic people' glucose tolerance may be underestimated when utilising the conventional FPG criteria or HbA1c, which are the gold standards for diagnosing diabetes. Without a thorough blood test and additional examinations, patients with compensated liver cirrhosis are often treated as healthy individuals since they do not exhibit any symptoms. Therefore, if postprandial hyperglycemia or

hypoalbuminemia are suspected in cirrhotic people with lower FPG values (110 mg/dL), an OGTT is advised. [9]

Although Holstein et al., [38] found no evidence of cardiovascular mortality, during the course of an average follow-up of five years, 52 percent of cirrhotic patients passed away due to cirrhotic sequelae. Therefore, it is questionable if these criteria are effective at correctly detecting diabetes in cirrhotic people. For the diagnosis of diabetes in cirrhotic people, additional variables like prognosis or diabetic sequelae may be used.

With a huge number of undiagnosed cases who are still living as pre diabetics makes the scenario all more worry some.

REVIEW OF OTHER STUDIES

Elichi Imano et al., (1999) had done a study on significance of OGTT for detection of Diabetes Mellitus in patients with liver cirrhosis and had concluded that the patients with occult liver disease should undergo screening for diabetes for early diagnosis and treatment and to improve the prognosis of cirrhosis. [14]

Diego Garcia Compean et al., (2014) had done a study on regarding impaired glucose tolerance that is subclinical as a predictor of death in liver cirrhosis and concluded that subclinical abnormal glucose tolerance has been associated with poor survival rate of patients with liver cirrhosis. [29]

Kayo Taguchi et al., (2014) had done a study on liver dysfunction's early warning symptom is insulin resistance in liver cirrhosis and concluded that insulin resistance in both liver and peripheral tissue is the early etiology of metabolic abnormality in the patients with liver cirrhosis. All LC patients had insulin resistance in both peripheral (skeletal and adipose) and hepatic tissues as measured by HECGL, although no significant link was found between the degree of glucose intolerance and the severity of hepatic dysfunction. Insulin resistance in both the liver and peripheral tissues is the first symptom of LC in patients. [30]

Laure Elkrief et al., (2016) had done a review on diabetes mellitus in patients with cirrhosis clinical implications and management and observed that DM is present in 30% of patients with cirrhosis and it is associated with poor prognosis in them. Diabetes is an independent predictive factor in people with CHC and cirrhosis. Improving diabetes management may improve cirrhosis outcomes. [31]

Pavan Hanchanale et al., (2017) had done a study on prevalence of glucose intolerance in cirrhotic and risk factors predicting its progression to diabetes mellitus and concluded that the Cirrhotic patients have a much higher incidence of glucose tolerance, and a 2-hour GGT reveals a considerable percentage of cirrhotic patients with normal fasting glucose and glycated haemoglobin. [32]

Kesavan K et al., (2019) had done a study on impaired glucose regulation in cirrhosis of liver the utility of oral glucose tolerance test and they had concluded that CLD was more common in males and there was a significant percentage of IGT/DM diagnosed in patients using OGTT. Their study showed significant percentage of patients with glucose metabolism disorders and correlation of them with the severity of the disease as the disease progressed. [5]

Huan Li et al., (2021) conducted a clinical study of abnormal glucose metabolism and insulin resistance in the patients with liver cirrhosis. The fasting insulin level of patients with cirrhosis of Child-Pugh grade C was considerably higher than that of Child-Pugh grade B patients, but FBG, PBG, and ISI did not differ significantly from those of Child-Pugh grade B patients. The greater the fasting and postprandial blood glucose levels, the higher the FIN with worsening liver function deterioration. Insulin resistance varied among patients with liver cirrhosis. Clinicians can take proactive steps to avoid the development of hepatogenic diabetes mellitus. [33]

MATERIALS AND METHODS

The study comprises patients with liver cirrhosis who are admitted as inpatients or patients who visit the outpatient clinic at the Shri B. M. Patil Medical College Hospital and Research Centre in Vijayapura who have been diagnosed with liver cirrhosis.

Study Design: Prospective Cross sectional study

Study Period: One and a half year from January 2021 to June 2022

Patient who are diagnosed cases of liver cirrhosis will be selected for study. The diagnosis of cirrhosis was made by clinical, radiological and/or laboratory evidence. Patient with radiological signs of cirrhosis, clinical signs of decompensated liver function (like ascites, portal hypertension, esophageal varices in upper GI endoscopy, hepatic encephalopathy and others) included in the study.

Patients with established liver cirrhosis irrespective of the underlying cause (ethanol consumption, Hepatitis B, Hepatitis C, autoimmune hepatitis etc).

Patients were deemed to have impaired glucose tolerance if their fasting plasma glucose ranged between 100 and 125 mg/dl and their two-hour plasma glucose ranged between 140 and 199 mg/dL. [34]

Each patient's information was gathered using a pre-tested proforma that matched the study's goals. Patients were clearly informed of the study's purpose before their agreement was obtained.

Clinical history, physical examination, and investigations described under investigations were used to obtain data. The proforma contained the information. The first and second hour values of the OGTT were recorded.

Sample collection:

Prior to the procedure, the individuals provided written consent. The patients were instructed to fast for 12 hours the night before the OGTT. Prior to the OGTT, the baseline plasma glucose levels was determined. After that, the patients received an oral glucose load of 75g of anhydrous glucose in 300ml of water, and after one and two hours, the plasma glucose levels were determined by drawing 3ml of blood into a sodium fluoride container. Impaired glucose tolerance was determined by a 2 hour glucose level between 140 and 199 mg/dl, while diabetes mellitus in cirrhotic patients was determined by a value over 200 mg/dl. [32]

METHOD OF COLLECTION OF DATA

Study population.

- Patients with liver cirrhosis
- Detailed physical and clinical examination was done.

Inclusion Criteria:

- Any patient with liver cirrhosis diagnosed clinically and laboratorically and/or radiologically.
- Non diabetic patients
- Patient age >20 and <80 years
- Viral hepatitis patients (Hepatitis B)

Exclusion Criteria:

- Any patient who does not give consent
- Overt diabetes patients
- Pregnant patients

Diagnostic Criteria:

- Patients were considered to have impaired glucose tolerance if their fasting blood sugar was between 100 and 125 mg/dl and/or their two-hour blood sugar was between 140 and 199 mg/dl after an oral glucose challenge. [34]
- Patient with 2nd hour OGTT value above 200mg/dl was taken as Diabetes Mellitus.

Sample Size: With an expected proportion of DM among patients with liver cirrhosis as diagnosed by the OGTT proportion rate of 35%13, a sample size of 85 patients would be needed for the study to have a 95% level of confidence and a 10% absolute precision.

The formula is N = Z 2/P*qd2.

Z=Z statistic at level of significance, where Absolute error = d2

P = Rate of Proportion q= 100-p

List of investigations

1 Complete blood count

2 Peripheral smear

3 LFT

4 Serum electrolytes

5 USG Abdomen (Hepato - biliary system)

6 HCV, HbsAg

7 PT/INR

8 URINE – Albumin, sugar, epithelial cells, pus cells, casts.

9 Upper GI endoscopy (if required)

10 OGTT

STATISTICAL ANALYSIS:

All of the collected data were entered into a Microsoft Excel spreadsheet, and statistical analysis was carried out using the social sciences statistical package (Version 20). Tables, figures, bar diagrams, and pie charts were used to show the results, which were summarised as Mean (Median) SD, counts, and percentages. Unpaired t-tests and chi-square tests were used to determine the mean difference between the continuous and categorical variables. A p-value <0.05 was considered statistically significant.

RESULTS

Table:1 Gender wise distribution of study

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	M	85	100.0	100.0	100.0

GRAPH:1 Genderwise distribution of the study

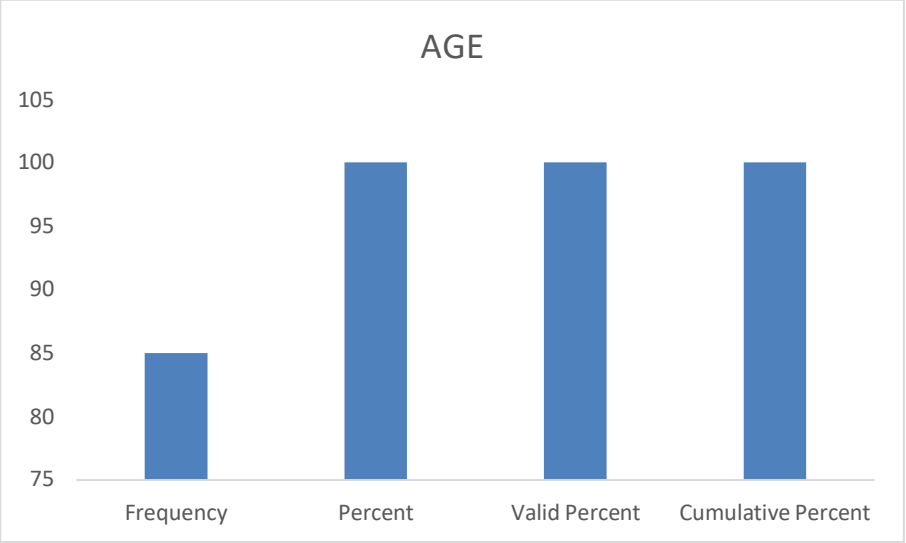

Table:2 Age wise distribution of study

Age (Years)	No. of patients	Percentage
< 30	2	2.4
30 – 39	19	22.4
40 – 49	32	37.6
50 – 59	21	24.7
60+	11	12.9
Total	85	100.0

The patients under the age of 30 years were 2%, between the age of 30-39, 40-49, 50-59, 60 were 22.4%, 37.6%, 24.7%, 12.9% respectively.

GRAPH- 2 Age wise distribution of the study

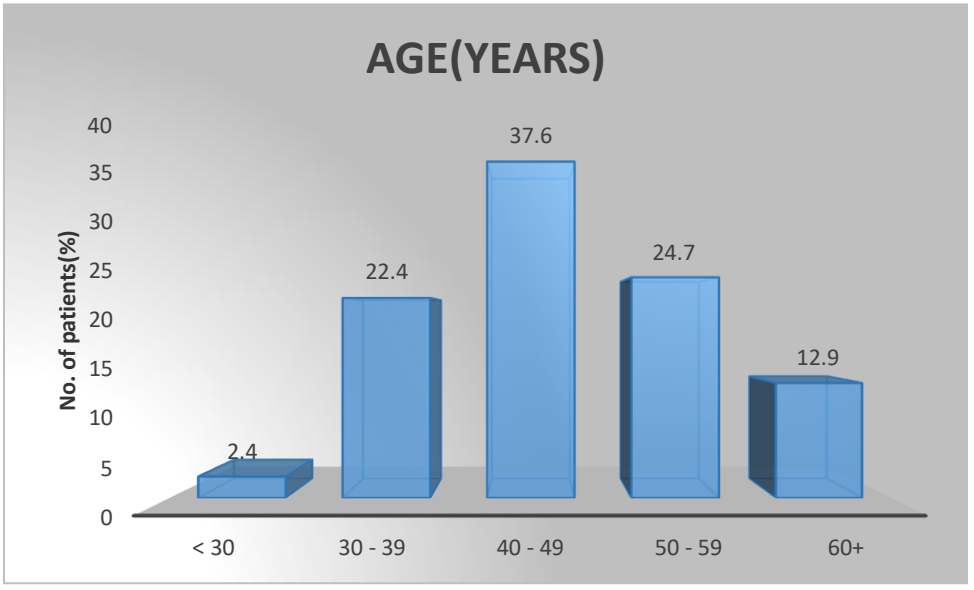

Table:3 The incidence of normal glucose tolerance

NGT	No. of patients	Percentage
Yes	29	34.1
No	56	65.9
Total	85	100.0

Out of the 85 patients included in the study, 29 patients were found to be having normal glucose tolerance which is 34.1%.

Graph -3 The incidence of normal glucose tolerance

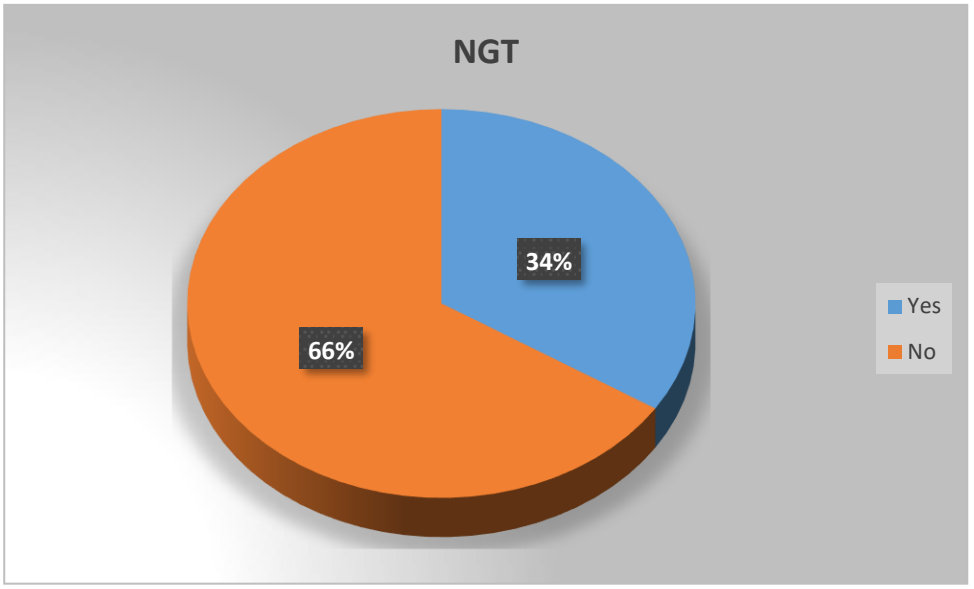

Table:4 The incidence of impaired glucose tolerance in study group

IGT	No. of patients	Percentage
Yes	15	17.6
No	70	82.4
Total	85	100.0

17.6% of the 85 individuals who were a part of the study had impaired glucose tolerance

GRAPH 4: The incidence of impaired glucose tolerance

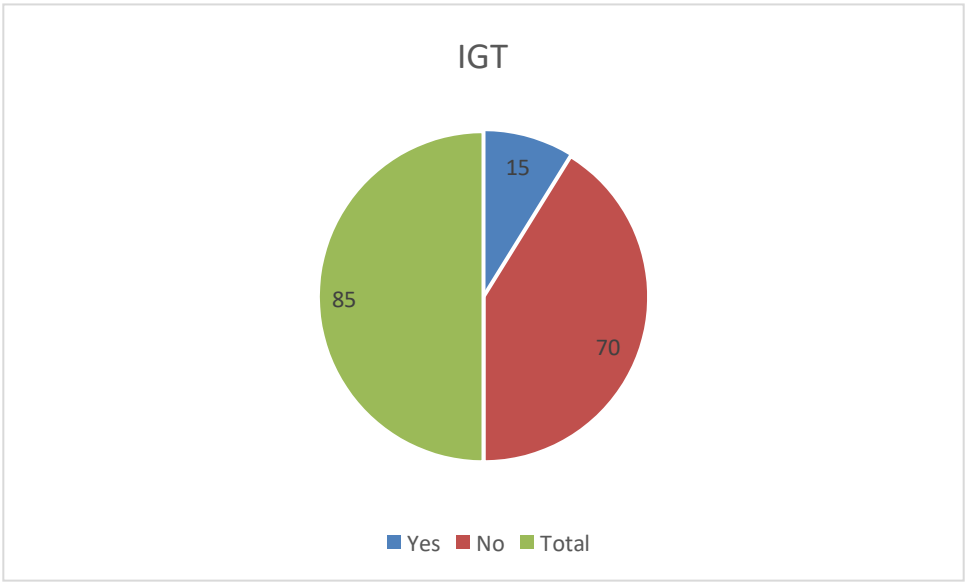

Table:5 The incidence of diabetes in study group

DIABETES	No. of patients	Percentage
Yes	15	17.6
No	70	82.4
Total	85	100.0

17.6% of the 85 study participants were found to have diabetes.

GRAPH 5: The incidence of diabetes

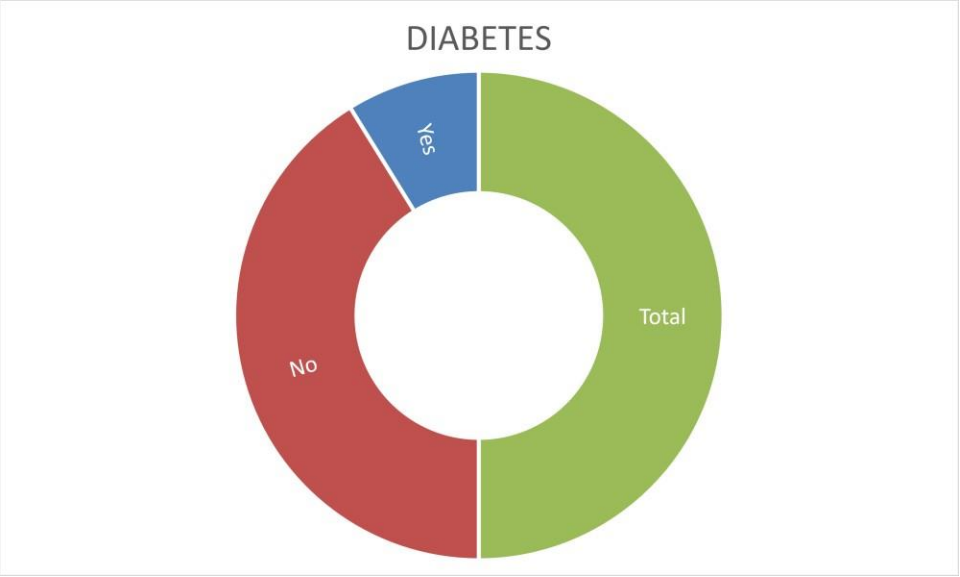

Table:6 The etiology involved in the study group

ETIOLOGY	No. of patients	Percentage
ALCOHOL	64	75.3
HEPATITIS B	1	1.2
HEPATITIS C	20	23.5
Total	85	100.0

75.3% of the 85 study participants were known to the researchers. case of alcoholic liver cirrhosis, 1.2% and 23.5% of patients were known cases of Hepatitis B&C infection.

GRAPH 6: Etiology in the study group

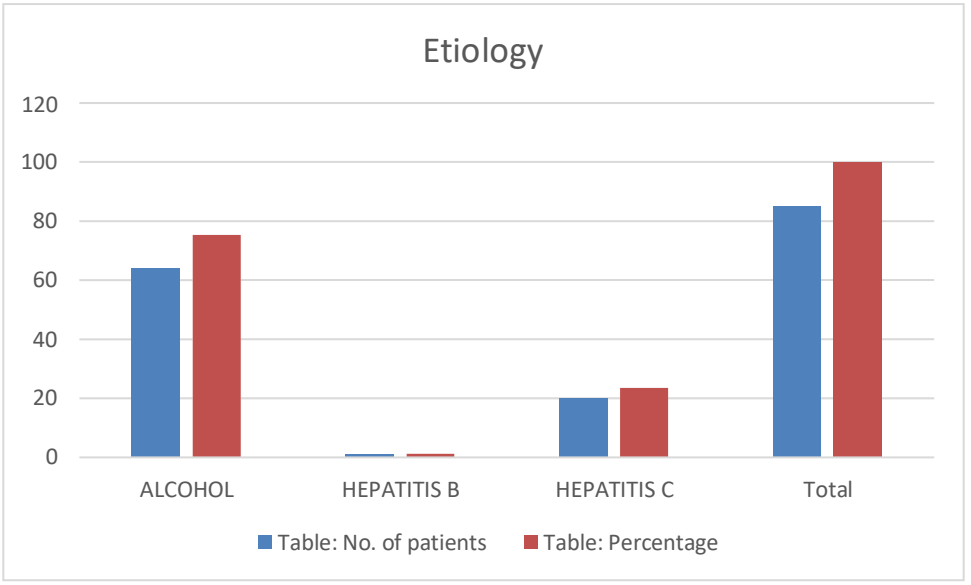

Table :7 The incidence of impaired glucose tolerance and diabetes

Table: **IGT VS DIABETES**

IGT	DIABETES				OR(95%CI)	Chi square test
	Yes		No			
	N	%	N	%	1.273(1.126-1.438)*	0.048*
Yes	0	0	15	100		
No	15	21.4	55	78.6		
Total	15	17.6	70	82.4		
*: Statistically significant						

Table 7: THE DESCRIPTIVE STATISTICS OF IMPAIRED GLUCOSE TOLERANCE AND NORMAL PATIENTS

Descriptive statistics of IGT & Normal Patients	Mean	Std. Deviation	95% Confidence Interval for Mean	
			Lower Bound	Upper Bound
HEMOGLOBIN				
IGT	8.53	1.506	7.70	9.37
Normal	8.29	2.266	7.75	8.83
Total	8.33	2.146	7.87	8.79
PLATELET COUNT				
IGT	123133.33	48761.616	96130.05	150136.62
Normal	123828.57	67140.124	107819.57	139837.57
Total	123705.88	64024.888	109896.03	137515.73
INR				
IGT	2.40	.828	1.94	2.86
Normal	1.86	.856	1.65	2.06
Total	1.95	.872	1.76	2.14
CREATININE				
IGT	1.47	.516	1.18	1.75
Normal	1.34	.478	1.23	1.46
Total	1.36	.484	1.26	1.47
TOTAL BILIRUBIN				
IGT	3.20	1.424	2.41	3.99
Normal	2.91	1.432	2.57	3.26
Total	2.96	1.426	2.66	3.27
SGPT				
IGT	40.67	13.075	33.43	47.91
Normal	39.80	13.658	36.54	43.06
Total	39.95	13.484	37.04	42.86
ALBUMIN				
IGT	2.00	.756	1.58	2.42
Normal	2.13	.867	1.92	2.34
Total	2.11	.845	1.92	2.29

Table:8 THE DEMOGRAPHIC AND VARIABLES OF THE STUDY

		N	Mean	Std. Deviation	95% Confidence Interval for Mean	
					Lower Bound	Upper Bound
HEMOGLOBIN	1.00	15	8.53	1.506	7.70	9.37
	2.00	70	8.29	2.266	7.75	8.83
	Total	85	8.33	2.146	7.87	8.79
PLATELET COUNT	1.00	15	123133.33	48761.616	96130.05	150136.62
	2.00	70	123828.57	67140.124	107819.57	139837.57
	Total	85	123705.88	64024.888	109896.03	137515.73
INR	1.00	15	2.40	.828	1.94	2.86
	2.00	70	1.86	.856	1.65	2.06
	Total	85	1.95	.872	1.76	2.14
CREATININE	1.00	15	1.47	.516	1.18	1.75
	2.00	70	1.34	.478	1.23	1.46
	Total	85	1.36	.484	1.26	1.47
TOTAL BILIRUBIN	1.00	15	3.20	1.424	2.41	3.99
	2.00	70	2.91	1.432	2.57	3.26
	Total	85	2.96	1.426	2.66	3.27
SGPT	1.00	15	40.67	13.075	33.43	47.91
	2.00	70	39.80	13.658	36.54	43.06
	Total	85	39.95	13.484	37.04	42.86
SODIUM	1.00	15	118.20	11.503	111.83	124.57
	2.00	70	123.33	12.887	120.26	126.40
	Total	85	122.42	12.741	119.68	125.17
ALBUMIN	1.00	15	2.00	.756	1.58	2.42
	2.00	70	2.13	.867	1.92	2.34
	Total	85	2.11	.845	1.92	2.29

Table :9 THE VARIABLES OF STUDY GROUP

		AGE	HEMOGLOBIN	PLATELET COUNT	INR	CREATININE
Mean		46.66	8.33	123705.88	1.95	1.36
Std. Deviation		10.309	2.146	64024.888	.872	.484
Percentiles	25	39.50	7.00	78000.00	1.00	1.00
	50	45.00	9.00	110000.00	2.00	1.00
	75	55.00	10.00	148000.00	2.00	2.00

Table :10 THE PERCENTAGE OF VARIABLES OF STUDY

		TOTAL BILIRUBIN	SGPT	SODIUM
Mean		2.96	39.95	122.42
Std. Deviation		1.426	13.484	12.741
Percentiles	25	2.00	28.00	111.00
	50	3.00	41.00	122.00
	75	4.00	52.00	132.00

DISCUSSION

Despite the fact that it has long been understood that DM and liver cirrhosis are concomitant conditions, the scope of the problem is typically understated. Due to the interaction of different factors in the aetiology and pathophysiology of both diseases, the relationship between DM and LC is quite complex.
Insulin resistance, steatosis, and diabetes mellitus have been linked to the HCV core protein, but overweight people with chronic HCV liver disease are more likely than non-obese patients to develop these conditions. Patients with asymptomatic liver disease will have trouble being diagnosed because of diabetes. Patients with liver cirrhosis.

This study sought to prove that it is common practice to ignore and underappreciate glycemic state in cirrhotic individuals. The outcome for these patients depends greatly on it. Numerous metabolic changes, primarily catabolic to muscle tissue, have been documented in liver cirrhosis. It is not yet understood how insulin resistance causes reduced glucose tolerance or overt diabetes mellitus.st receptor dysfunction probably exists in chronic liver disease that might be explained by the following factors:

1. Modified membrane lipid composition and elevated free fatty acid levels
2. Persistent insulin resistance
3. Elevated plasma concentrations of hormones that block the action of insulin, including growth hormone, glucagon, catecholamines, and maybe cytokines
4. Deficiency in insulin-like growth factors I and II, liver-derived humoral factors with insulin-like action.

In Impaired insulin sensitivity and subsequent changes in glucose metabolism (as shown by the high prevalence of insulin resistance and glucose intolerance) are unquestionably acquired in chronic liver diseases like cirrhosis. About 80% of cirrhotic individuals have glucose intolerance, 60% to 80% of them are insulin resistant, and 20% go on to develop overt diabetes mellitus. Long-standing theories suggest that chronic hyperinsulinemia causes or worsens insulin resistance.

Hepatogenous diabetes is the term used to describe diabetes mellitus related to LC (HD). Despite the fact that it is widely acknowledged that LC is a diabetogenic condition. The American Diabetes Association (ADA) and the World Health Organization do not recognise HD (WHO). Hemochromatosis, HCV, and alcohol-related cirrhosis are more frequently linked to HD than other etiologies.

In our study, out of the 85 patients enrolled, 17.6% were found to have impaired glucose tolerance and 17.6% to have diabetes. The patients involved in the study were all male. Because of this, there is a strong correlation between liver cirrhosis and diabetes, and OGTT is a useful test for determining the glycemic state of these individuals.

CONCLUSION

CONCLUSION

Both type 1 and type 2 diabetes are becoming more common, but type 2 is becoming more common more quickly. In type 2 diabetes individuals, dyslipidemia, abnormalities in the liver's enzymes, and nonalcoholic fatty liver disease (NAFLD) are common. The progression of fatty liver into cirrhosis and chronic liver disease can be prevented by adequate glycemic control and regular monitoring of FLP, LFT, and fatty liver. We draw the conclusion that patients with liver cirrhosis are more likely to have impaired glucose tolerance and diabetes mellitus. The OGTT is a trustworthy test that should be used to diagnose IGT and DM, providing the chance to recognise and stop the consequences of hyperglycemia.

SUMMARY

SUMMARY

This randomized cross section study was titled as "A PROSPECTIVE STUDY TO IDENTIFY THE PREVALENCE OF IMPAIRED GLUCOSE TOLERANCE IN PREVIOUS UNDIAGNOSED DIABETES IN CIRRHOSIS PATIENTS USING ORAL GLUCOSE TOLERANCE TEST" was carried out from January 2021 to July 2022 at the B.L.D.E (DU) Shri. B. M. Patil Medical College and Hospital, in the department of General medicine, Vijayapura.

It was designed to perform Oral glucose tolerance test in patients with liver cirrhosis and record the glucose values at 2 hour interval and interpret the glycemic status of the patient and comment about the prevalence. The objective was to perform fasting glucose value and 2 hour OGTT in cirrhosis patients and determine their glycemic status.

The study population of 85 with age and sex matched was randomly selected patients with liver cirrhosis between the age of 20 years to 60 years of both inpatient and out patient.

The observations and results were analysed statistically and were as follows:

Out of the 85 individuals who were involved in the study, 85 were male patients. 17.6% were found to have impaired glucose tolerance and 17.6% were found to be diabetic.

Thus there is a high prevalence of Impaired glucose tolerance and diabetes mellitus in patients with liver cirrhosis.

BIBLIOGRAPHY

LIST OF REFERENCES

1. Tsochatzis EA, Bosch J, Burroughs AK. Liver cirrhosis. Lancet. 2014;383(9930):1749–61.

2. Schuppan D, Afdhal NH. Liver cirrhosis. Lancet. 2008;371(9615):838–51.

3. Bianchi G, Marchesini G, Zoli M, Bugianesi E, Fabbri A, Pisi E. Prognostic significance of diabetes in patients with cirrhosis. Hepatology. 1994;20(1):119–25.

4. Kumar R. Hepatogenous diabetes: an underestimated problem of liver cirrhosis. Indian J Endocrinol Metab. 2018;22(4):552–7.

5. L'heveder R, Nolan T. International diabetes federation. Diabetes Res Clin Pract. 2013;101(3):349–51.

6. Classification and Diagnosis of Diabetes. Diabetes Care. 2016;39(Supplement 1):S13 LP-S22.

7. Carrillo-Larco RM, Barengo NC, Albitres-Flores L, Bernabe-Ortiz A. The risk of mortality among people with type 2 diabetes in Latin America: A systematic review and meta-analysis of population-based cohort studies. Diabetes Metab Res Rev. 2019;35(4):e3139.

8. Mayfield J. Diagnosis and Classification of Diabetes Mellitus: New Criteria. Am Fam Physician. 1998;58(6):1355–62.

9. Nishida T. Diagnosis and Clinical Implications of Diabetes in Liver Cirrhosis: A Focus on the Oral Glucose Tolerance Test. J Endocr Soc. 2017;1(7):886–96.

10. Hickman IJ, Macdonald GA. Impact of diabetes on the severity of liver disease. Am J Med. 2007 Oct;120(10):829–34.

11. Muting D, Wohlgemuth D, Dorsett R. Liver cirrhosis and diabetes mellitus. Geriatrics. 1969

Jan;24(1):91–9.

12. Falchuk KR, Fiske SC, Haggitt RC, Federman M, Trey C. Pericentral hepatic fibrosis and intracellular hyalin in diabetes mellitus. Gastroenterology. 1980 Mar;78(3):535–41.

13. Imano E, Nishida T, Shibata M, Kanda T. Significance of oral glucose tolerance test for the diagnosis of diabetes mellitus in patients with liver cirrhosis. Vol. 38, Internal medicine (Tokyo, Japan). Japan; 1999. p. 918–22.

14. Imano E, Kanda T, Nakatani Y, Motomura M, Arai K, Matsuhisa M, et al. Impaired splanchnic and peripheral glucose uptake in liver cirrhosis. J Hepatol. 1999 Sep;31(3):469–73.

15. Roden M, Bernroider E. Hepatic glucose metabolism in humans--its role in health and disease. Best Pract Res Clin Endocrinol Metab. 2003 Sep;17(3):365–83.

16. Proietto J, Alford FP, Dudley FJ. The mechanism of the carbohydrate intolerance of cirrhosis. J Clin Endocrinol Metab. 1980 Nov;51(5):1030–6.

17. Shmueli E, Walker M, Alberti G, Record CO. Normal splanchnic but impaired peripheral insulin-stimulated glucose uptake in cirrhosis. Hepatology. 1993 Jul;18(1):86–95.

18. Shanik MH, Xu Y, Skrha J, Dankner R, Zick Y, Roth J. Insulin resistance and hyperinsulinemia: is hyperinsulinemia the cart or the horse? Diabetes Care. 2008 Feb;31 Suppl 2:S262-8.

19. Kolaczynski JW, Carter R, Soprano KJ, Moscicki R, Boden G. Insulin binding and degradation by rat liver Kupffer and endothelial cells. Metabolism. 1993 Apr;42(4):477–81.

20. Dasarathy S, Merli M. Sarcopenia from mechanism to diagnosis and treatment in liver disease. J Hepatol. 2016 Dec;65(6):1232–44.

21. Davuluri G, Krokowski D, Guan B-J, Kumar A, Thapaliya S, Singh D, et al. Metabolic adaptation of

skeletal muscle to hyperammonemia drives the beneficial effects of l-leucine in cirrhosis. J Hepatol. 2016 Nov;65(5):929–37.

22. El-Serag HB, Tran T, Everhart JE. Diabetes increases the risk of chronic liver disease and hepatocellular carcinoma. Gastroenterology. 2004 Feb;126(2):460–8.

23. Shulman GI, Rothman DL, Jue T, Stein P, DeFronzo RA, Shulman RG. Quantitation of muscle glycogen synthesis in normal subjects and subjects with non-insulin-dependent diabetes by 13C nuclear magnetic resonance spectroscopy. N Engl J Med. 1990 Jan;322(4):223–8.

24. Kanda T. Clinical significance of HbA1c, serum fructosamine and 1, 5-AG in diabetic patients complicated with liver cirrhosis. J Japan Diab Soc. 1993;36:847–54.

25. Panzer S, Kronik G, Lechner K, Bettelheim P, Neumann E, Dudczak R. Glycosylated hemoglobins (GHb): an index of red cell survival. Blood. 1982 Jun;59(6):1348–50.

26. Koga M, Kasayama S, Kanehara H, Bando Y. CLD (chronic liver diseases)-HbA1C as a suitable indicator for estimation of mean plasma glucose in patients with chronic liver diseases. Diabetes Res Clin Pract. 2008 Aug;81(2):258–62.

27. Matsumoto N, Arase Y, Seko Y, Imai N, Kawamura Y, Sezaki H, et al. Prevalence and predictive factors of diabetes in hepatitis virus positive liver cirrhosis with fasting plasma glucose level of <126 mg/dL. Hepatol Res. 2012 Jun;42(6):558–63.

28. Holstein A, Hinze S, Thiessen E, Plaschke A, Egberts E-H. Clinical implications of hepatogenous diabetes in liver cirrhosis. J Gastroenterol Hepatol. 2002 Jun;17(6):677–81.

29. García-Compeán D, Jáquez-Quintana JO, Lavalle-González FJ, González-González JA, Muñoz-Espinosa LE, Villarreal-Pérez JZ, et al. Subclinical abnormal glucose tolerance is a predictor of death in liver cirrhosis. World J Gastroenterol WJG. 2014;20(22):7011.

30. Taguchi K, Yamanaka-Okumura H, Mizuno A, Nakamura T, Shimada M, Doi T, et al. Insulin resistance as early sign of hepatic dysfunction in liver cirrhosis. J Med Investig. 2014;61(1.2):180–9.

31. Elkrief L, Chouinard P, Bendersky N, Hajage D, Larroque B, Babany G, et al. Diabetes mellitus is an independent prognostic factor for major liver-related outcomes in patients with cirrhosis and chronic hepatitis C. Hepatology. 2014;60(3):823–31.

32. Hanchanale P, Venkataraman J, Jain M, Srinivasan V, Varghese J. Prevalence of glucose intolerance in cirrhotics and risk factors predicting its progression to diabetes mellitus. J Diabetol. 2017;8(1):12.

33. Li H, Sun F, Yang W, Huang M, Pan C, Lin C. Clinical study of abnormal glucose metabolism and insulin resistance in patients with liver cirrhosis. Am J Transl Res. 2021;13(4):3522–8.

34. Badiu C. Williams textbook of endocrinology. Acta Endocrinol. 2019;15(3):416–20.

ANNEXURE – I

ETHICAL CLEARANCE CERTIFICATE

ANNEXURE – II

INFORMED CONSENT FORM

TITLE OF RESEARCH: A PROSPECTIVE STUDY TO IDENTIFY THE PREVALENCE OF IMPAIRED GLUCOSE TOLERANCE IN CIRRHOSIS PATIENTS USING ORAL GLUCOSE TOLERANCE TEST

GUIDE : DR VIJAYKUMAR.G.WARAD

P.G. STUDENT : DR. OBUL REDDY

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to identify the prevalence of impaired glucose tolerance and diabetes in cirrhosis patients using oral glucose tolerance test.

PROCEDURE:

I understand that I will undergo detailed history and clinical examination and investigations.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved in this study and I may experience some pain during the above mentioned procedures.

BENEFITS:

I understand that my participation in this study will help Dr. VeeramReddy Obul Reddy to identify theprevalence of impaired glucose tolerance in cirrhosis patients using oral glucose tolerance test.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulation of hospital. If the data is used for publication the identity will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

INJURY STATEMENT:

I understand in the unlikely event of injury to me during the study I will get medical treatment but no further medical compensation.

_____

(Signature of patient)

STUDY SUBJECT CONSENT FORM:

I confirm that Dr. VeeramReddy Obul Reddy has explained to me the purpose of this research, thestudy procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all above in detail in my own language and I understand the same. I agree to give my consent to participate as a subject in this research project.

SIGNATURE OF PARTICIPANT DATE

ANNEXURE IX

PROFORMA

Name of the patient: OP / IP. NO:

Age in years: Sex: Religion:

Address: Occupation: Annual Income:

Presenting Complaints:

Past history:

Personal history:

1. Diet-Veg/Mixed
2. Sleep
3. Appetite
4. Bowel and Bladder Habits
5. Habits

Alcoholism – Duration of consumption (in years)

Ethanol consumption per day (in grams)

Family history: Any H/O T2 DM or Chronic liver disease in family or Ischemic heart disease or TB or hypertension in the family

GENERAL PHYSICAL EXAMINATION:

Built and Nourishment:

Ht (Cm):

Wt (Kg): BMI:

HEAD TO TOE PHYSICAL EXAMINATION (including signs of liver cell failure) :

a) Anaemia
b) Cyanosis
c) Jaundice
d) Palmar erythema
e) Flapping tremors
f) Spider naevi
g) Skin pigmentation
h) Loss of weight
i) Others (if any)

Vital parameters: a. Pulse:

b. BP:

c. Respiratory Rate:

d. Temperature:

SYSTEMIC EXAMINATION:

ABDOMEN EXAMINATION

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM

CENTRAL NERVOUS SYSTEM

INVESTIGATIONS	
1) Complete Blood Count	
2) Peripheral smear	
3) PT, INR	
3) LFT	
5) Serum electrolytes	
6) Urine examination	
7) HbsAg, HCV	
8) USG Abdomen	
9) ABG (if necessary)	
10) Upper GI Endoscopy (if done)	

OGTT:

S.NO	FASTING PLASMA GLUCOSE	2^{ND} HOUR PLASMA GLUCOSE

AGE	GENDER	ETIOLOGY	HEMOGLOBIN	PLATELET COUNT	INR	CREATININE	TOTAL BILIRUBIN	SGPT	SODIUM	ALBUMIN	NGT	IGT	DIABETES
45	M	ALCOHOL	11	136000	1.4	0.8	1.6	19	128	1.8	N		
50	M	ALCOHOL	9.2	128000	2	1.2	2.4	24	132	1.6	N		
57	M	ALCOHOL	8.6	100000	1.6	0.9	1.9	26	130	2.4	N		
30	M	HEPATITIS C	9.4	145000	1.2	1	1.4	35	129	2.3	N		
43	M	HEPATITIS C	8.4	150000	3.9	1.6	3.6	46	110	3		I	
45	M	ALCOHOL	8.9	145000	3.4	1.4	5.5	29	119	1	N		
63	M	ALCOHOL	9.2	155000	2.4	1.9	6	32	109	1.3	N		
35	M	HEPATITIS C	10.2	165000	1.6	2	3.9	56	110	1.8		I	
70	M	HEPATITIS B	8.6	110000	2	0.9	2.8	54	109	1.9	N		
50	M	ALCOHOL	9.8	55000	2.6	1.5	2.4	42	122	1.1		I	
40	M	HEPATITS C	7.4	89000	1	1.32	3.6	51	132	1.3	N		
56	M	ALCOHOL	8.4	98000	3.6	0.8	3.9	23	135	2.3	N		D
46	M	ALCOHOL	9.8	55000	3.1	0.9	4.5	28	106	1		I	
38	M	ALCOHOL	10.2	66000	3.7	1.2	3	56	110	1		I	
30	M	HEPATITS C	8.4	120000	4.1	1.7	6	43	120	2.4	N		
32	M	ALCOHOL	7.2	145000	2	1.4	7	34	124	1.9		I	
41	M	ALCOHOL	10.6	190000	1	1.3	1.2	29	127	2.8	N		
62	M	ALCOHOL	8.2	110000	2.2	2	3.2	35	123	3.3		I	
30	M	HEPATITS C	8.6	234000	2.4	1.8	2.6	54	134	1.6		I	
45	M	ALCOHOL	9.2	330000	1	1.2	2.5	58	146	1.1			D
48	M	ALCOHOL	7.4	98000	1.6	1.6	3.1	44	129	2.6	N		
69	M	ALCOHOL	6.1	90000	1.3	1.5	3	41	132	2.9	N		
36	M	ALCOHOL	8.4	145000	2.5	1.3	2.8	28	110	3.1		I	
42	M	HEPATITS C	9.2	123000	4	1.2	2.3	38	105	3.3	N		
45	M	ALCOHOL	8.6	110000	1	1.8	2.8	31	108	2.4			D
34	M	ALCOHOL	8.2	100000	1.2	1.9	1.9	24	134	1.7	N		
65	M	ALCOHOL	9.1	99000	1.6	2	4.1	18	127	1.5		I	
47	M	ALCOHOL	9.4	450000	4	0.9	4.2	62	123	1.1	N		
38	M	ALCOHOL	7.6	55000	1.3	1	2.9	54	110	1	N		
44	M	ALCOHOL	7.8	65000	2.1	1.6	1.8	48	140	2.2	N		
49	M	ALCOHOL	9.6	89000	2	1.2	2	44	139	2.7			D
50	M	ALCOHOL	8	145000	1.2	1.4	3	41	110	1	N		
38	M	HEPATITS C	10	165000	1.4	1.1	4.1	49	105	1.9		I	
36	M	ALCOHOL	10.4	75000	2	2.4	1.9	57	102	2.7	N		
56	M	ALCOHOL	10.1	45000	1.8	0.8	1.6	52	100	1.4	N		
65	M	HEPATITS C	7.1	146000	1.3	1.6	2.4	50	140	1			D
30	M	ALCOHOL	6.3	138000	2	0.9	2.2	26	120	2.5		I	
31	M	ALCOHOL	10.1	245000	1.4	1.8	1	20	122	1.5	N		
29	M	ALCOHOL	6.4	68000	1.6	1.2	1.6	36	132	1	N		
41	M	ALCOHOL	6.8	78000	3	2	1.8	29	131	1.3		I	
44	M	ALCOHOL	5.1	89000	2.4	0.8	5	18	106	1.9			D
54	M	ALCOHOL	5	98000	2.8	1	4.3	47	116	1.2	N		
45	M	ALCOHOL	9.8	165000	3.6	1.6	3.2	52	134	1.8	N		
55	M	ALCOHOL	10.6	55000	1.4	1.2	3.6	56	139	2.3	N		
43	M	ALCOHOL	8.1	178000	1.2	0.8	1.9	32	120	2.2	N		
55	M	HEPATITS C	8.5	76000	1.6	1.3	2.2	56	119	2			D
55	M	HEPATITIS C	11	124000	2	1.1	5	46	114	3.3	N		
61	M	ALCOHOL	7	154000	3.5	1.2	4.3	34	112	3.4	N		
43	M	HEPATITS C	9	132000	2.1	0.8	3.3	24	145	1.7	N		
43	M	ALCOHOL	10	110000	1.7								
42	M	ALCOHOL	8.4	58000	1.8								
56	M	ALCOHOL	9.6	78000	2.1								
29	M	ALCOHOL	9.8	160000	1.9								
41	M	HEPATITIS C	10.7	232000	2.4								
52	M	ALCOHOL	10.9	120000	1.5								
33	M	ALCOHOL	11	110000	1.49								
42	M	ALCOHOL	12	145000	2.4								
36	M	HEPATITIS C	6	155000	2.3								
43	M	ALCOHOL	5.4	60000	1.8								
62	M	ALCOHOL	4.4	84000	1.6								
43	M	ALCOHOL	9.4	230000	1.3								
44	M	ALCOHOL	12.1	34000	2.5								
30	M	HEPATITIS C	5.5	78000	1								
53	M	ALCOHOL	5.8	89000	1.4								
44	M	HEPATITIS C	8.9	65000	1.6								
65	M	ALCOHOL	9.7	98000	2.3								
56	M	ALCOHOL	12.4	120000	2.2								
65	M	ALCOHOL	7	110000	1.8								
64	M	HEPATITIS C	6	132000	1.5								
47	M	ALCOHOL	8.5	145000	1.2								
39	M	ALCOHOL	5.3	150000	2.7								
46	M	ALCOHOL	4.2	80000	2.4								
38	M	ALCOHOL	3	70000	1.9								
40	M	ALCOHOL	9	76000	1.7								
52	M	ALCOHOL	8.8	98000	1.5								
55	M	HEPATITIS C	5	65000	1.4								
47	M	ALCOHOL	4.3	54000	2								
58	M	ALCOHOL	9	78000	1.2								
55	M	ALCOHOL	8.4	89000	1								
39	M	ALCOHOL	4.4	156000	2								
48	M	ALCOHOL	3.6	145000	1.3								
50	M	HEPATITIS C	11	198000	1.4								
54	M	ALCOHOL	10.4	230000	1.7								
49	M	ALCOHOL	9.8	89000	2								

1.1	2	54	102	3.2		I	
0.9	1.4	44	110	2.6	N		
1.3	2.3	21	112	2	N		
0.9	2.2	17	118	1.8			D
1.8	1.9	56	128	1.1	N		
0.7	1.8	46	142	2.2	N		
1.3	3.4	52	130	3.4	N		
0.9	3.6	24	108	3.2	N		
1.6	4.4	61	102	1.1	N		
1	6	59	100	1			D
0.9	1.7	44	136	2.6	N		
2.4	2.9	42	139	3.5	N		
1.7	3	36	125	3.4	N		
0.8	4.4	39	137	3.9	N		
1.1	6.7	24	154	1			D
1.6	6	25	144	2.5	N		
0.9	4.2	51	123	2.3	N		
1.5	2.9	62	110	2.7	N		
1.7	1.2	70	112	1.2	N		
0.8	1.1	55	139	2.5		I	
1	2.7	41	116	2.2			D
1.5	2.8	57	129	1.5	N		
1.3	3.1	56	120	1	N		
0.9	3	45	124	2.6			D
0.8	1.5	43	148	1.5	N		
2	1.9	31	128	2	N		
2.3	2	29	116	2.2	N		
2.1	2.5	26	114	2.3			D
1.7	4.5	21	112	1.5	N		
0.9	5	35	130	1.7	N		
0.7	1.4	41	122	3.6			D
1	2	50	117	2.5	N		
1.4	1.5	19	114	3.7	N		
1.6	1.3	24	118	2.9			D
0.8	1	46	134	2	N		