Journal of Cardiovascular Disease Research

ISSN:0975 -3583,0976-2833 VOL13, ISSUE 08, 2022

Original research article

Diabetes and risk of persistent non-alcoholic fatty liver disease

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Abstract

Background and Objectives: The purpose of research was to investigate the incidence of non-alcoholic fatty liver disease, also known as fatty liver, in diabetic patients.

Methods: The research was done from June 2019 to May 2020 at Department of General Medicine, Katuri Medical College and Hospital, Guntur, Andhra Pradesh, India. 85 type 1 and type 2 diabetics were examined. IPD and OPD patients participated. The study group included 20 males and 65 females, ages 19 to 73.

Results: 3 males (3.5%) had fatty liver and 30 females (35.3%). In 36 of 85 diabetics tested, fatty liver was detected. Patients with fatty liver and central obesity (waist-hip ratio >1): 25 (69.44%). Patients with fatty liver had >180 triglycerides 31 (86.1%). Overweight patients with fatty liver numbered 24 (66.67%). 12 fatty liver patients had normal BMIs (33.33%). Patients with fatty liver and high cholesterol (>200 mg %): 23 (69.69%). No change in serum protein or albumin globulin ratio.

Conclusion: Non-alcoholic fatty liver disease was 42.35% frequent, especially in type 2 diabetics. It affected women (35.3%) more than males. NAFLD is more common in extremely obese and centrally obese people. 90% of fatty liver sufferers had dyslipidaemia's (particularly hypertriglyceridemia).

Keywords: Type 2 diabetes, fatty liver disease, cholesterol, triglyceride, non-alcoholic steatohepatitis

Introduction

In India, the prevalence of diabetes is 3.8%. It is well knowledge that diabetes affects practically all organ systems and is a systemic illness. Today, more and more people are becoming aware of non-alcoholic fatty liver disease (NAFLD) as a type 2 diabetes complication that has the potential to be very devastating. Simple steatosis or steatosis with modest inflammation to severe non-alcoholic steatohepatitis are all on the NAFLD continuum (NASH) [1, 2]. Although there are still many unanswered questions regarding the pathogenesis and therapy, great progress has been made since the designations non-alcoholic steatohepatitis (NASH) and non-alcoholic fatty liver disease (NAFLD) were first coined in 1980 and 1986, respectively.

Blood sugar levels are tightly controlled within specific ranges. An individual's blood glucose level in the post-absorptive state is typically set between 4.5 and 5 mmol/L in humans and many other mammals. It may increase to 6.5-7.2 mmol/L following the consumption of a meal high in carbohydrates. 3.3 to 3.9 mmol/L are the amounts while fasting [2, 3, 4].

The passage through the cell membrane is the rate-limiting phase in the uptake of glucose in extra hepatic tissues since extra hepatic tissues, with the exception of the pancreatic islets, are somewhat impermeable to glucose (via the GLUT 2 receptors) while liver cells are totally permeable. An essential element that affects how quickly glucose is absorbed by the liver and extra hepatic tissues is the blood glucose concentration [4, 5, 6]. The liver's ability to absorb glucose is likely directly influenced by the concentration of important intermediates as well as the activity of specific enzymes. Blood sugar levels after a meal must be controlled by the enzyme glucokinase. Because glucose-6-phosphate inhibits the enzyme hexokinase, which is present in extra hepatic organs, there may be some feedback control over the uptake of glucose in these tissues. Because glucokinase is unaffected by glucose-6-phosphate, the liver is not confined by this restriction [6, 7, 8].

The liver seems to be the net generator of glucose when systemic blood glucose levels are normal (4.5-5.5 mmol/L). At higher levels, there is a net intake of glucose since the output of glucose stops as the glucose level rises. The control of blood sugar is mostly dependent on insulin. It is directly a result of the degree of hyperglycaemia and is produced by the B-cells of the islets of Langerhans in the pancreas. Through the GLUT 2 receptors, glucose can readily enter the islet cell. The flow through glycolysis, the citric acid cycle, and ATP synthesis are hence dependent on blood glucose levels [8, 9].

The B-cell membrane depolarizes as a result of increased calcium influx through voltage-sensitive calcium channels, which in turn stimulates insulin exocytosis. Increased ATP concentration inhibits the

ISSN:0975 -3583,0976-2833 VOL13, ISSUE 08, 2022

ATP-sensitive potassium channels that are responsible for depolarizing the B-cell membrane.

Material and Methods

From June 2019 to May 2020 the investigation was carried out at Department of General Medicine, Katuri Medical College and Hospital, Guntur, Andhra Pradesh, India, where the patients adapted the inclusion and exclusion parameter.

Inclusion criteria

1. The presence of diabetes mellitus (types 1 or 2) for whatever length of time.

Exclusion criteria

1. Drinking alcohol.
2. HIV ELISA seropositivity.
3. Anti-HCV antibody positivity.
4. People taking medications known to cause steatohepatitis, such as estrogen-containing medicines, oral contraceptives, amiodarone and steroids.

During this time, 85 diabetics of both types 1 and 2 were studied. The study comprised both inpatients and outpatients. The average age of the study group, which included around 20 men and 65 women, ranged from 19 to 73 years old. These patients had diabetes for an average of 5.17 years out of a possible total of 0 to 20 years.

The screening results for both HIV and HCV for all of the patients mentioned above were negative. These individuals underwent the following tests: random blood sugar, lipid profile, abdominal ultrasound, and liver function test. The patient's height, weight and BMI were all measured. A BMI of 25 or higher was deemed overweight. To check for central obesity, the waist to hip ratio was also assessed [9, 10].

NAFLD was presumed to be present if the ultrasonography revealed signs of fatty liver, whether or not transaminases were elevated. Data were gathered and examined for the following reasons: the frequency of NAFLD, Lipid profile and the occurrence of NAFLD are associated, core obesity and NAFLD's connection.

Results

Table 1: Fatty liver in patients

No. of patients with fatty liver	No. of patients without fatty liver
36 (42.35%)	49

36 patients (42.35%) of the 85 diabetics that were investigated had fatty liver.

Table 2: Gender distribution

Sex	Fatty liver present	Fatty liver absent
Male	3 (3.5%)	17
Female	30 (35.3%)	35

Males made up 3 (3.5%) of the population, while females made up 30 (35.3%).

Table 3: Waist hip ratio

Waist hip ratio >1	Waist hip ratio <1
25 (69.44%)	11 (30.5%)

There were 25 (69.44%) patients with fatty liver who also had central obesity (waist to hip ratio greater than 1). The waist-hip ratio of all patients with fatty liver was greater than one in men and greater than 0.85 in women.

Table 4: TGL in patients

No. of patients with ↑ TGL	No. of patients with normal TGL
31 (86.12%)	5

There were 31 individuals (86.12%) with fatty liver who had elevated triglycerides >180.

Table 5: BMI of patients

No. of patients with BMI >25	No. of patients with BMI <25
24 (66.67%)	12 (33.33%)

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There were 24 (66.67%) patients who were overweight out of the people whose livers were found to be fatty. There were 12 (33.33%) patients with normal BMI among those with fatty liver.

Table 6: Cholesterol in patients

Patients with cholesterol >200	Patients with cholesterol <200
23 (69.69%)	13 (39.39%)

23 (69.69%) of the individuals with fatty liver had elevated cholesterol (>200 mg %). Serum alkaline phosphatase and transaminases were very slightly increased in all of the patients with ultrasonography indications of fatty liver. Neither the serum protein ratio nor the albumin globulin ratio changed.

Discussion

The prevalence of fatty liver is 42.35%, according to this study. According to research by Daad H. Akbar, 55% of participants had NAFLD. Ultrasonography testing was used in another investigation by Gupta P *et al*. to estimate the prevalence of NAFLD to be 49%. Patients with NAFLD were more likely to be overweight (67.74%). Wanless and Lentz found mild to severe steatosis in 35% of individuals who were lean and roughly 70% of patients who were obese. According to Garcia Monzon *et al*., only 8% of patients had normal biopsy results, whereas 69% of patients had NASH, 22% had simple steatosis. The highest prevalence of NAFLD was seen in participants with a BMI of 30 +/- 5.5 kg/m2 in another investigation. In contrast, another study's NAFLD patients showed no statistically significant variation in body mass index. NASH may slowly develop in obese people over years with little symptoms before becoming obvious on its own, as a result of cirrhosis, or as a result of portal hypertension issues [10, 11, 12].

The prevalence of fatty liver was found to be 35.3% greater in women than in males. Women are more prone than males to acquire fatty livers, according to numerous research. NAFLD and NASH have been documented in patients who lacked the conventional risk factors of obesity, diabetes, and overt hyperlipidemia. It was mentioned in patients with central visceral obesity in a study by Bacon BR *et al*. A substantial link between visceral adipose tissue and fatty liver was discovered by another investigation. In this study, 100% of patients with fatty liver had central obesity, and 69.44% of patients had a waist to hip ratio larger than one. Individuals with increased triglycerides and cholesterol were present in 86.12 % and 69.9% more patients, respectively [12, 13, 14].

As was previously mentioned, one-third of those with hypercholesterolemia and two-thirds of those with hypertriglyceridemia have fatty liver. Another study found that 20% of patients with obesity and fatty liver disease also had previously identified hyperlipidemia. In other studies, fatty liver and the degree of dyslipidemia, particularly the level of triglycerides, were significantly correlated. Hypertriglyceridemia has been found to be a substantial risk factor for the onset of NAFLD and NASH and to be closely correlated with the histological severity of the disease [14, 15, 16].

The risk factors with the best documentation include type 2 diabetes and obesity, while hypertriglyceridemia and advanced age are indicators of the severity of underlying histologic changes. There is often truncal or central adiposity in slim people with fatty livers. Women are more likely than men to suffer from NAFLD [16, 17, 18]. Only 10% of obese individuals who underwent multiple evaluations had normal liver biopsy outcomes. Cirrhosis affects less than 5% of the population, but steatosis affects 85%. Third of the latter group have NASH.

Conclusion

The incidence of non-alcoholic fatty liver disorder was 42.3% and individuals with type 2 diabetes mellitus are mostly likely to have it. It was observed in women at a rate that was 35.3% higher than in men. Participants who were discovered to have a higher prevalence of non-alcoholic fatty liver disease were found to be those who were overweight or obese, as well as those who had central obesity. 92.3% of the patients diagnosed with fatty liver had dyslipidaemia's, most notably hypertriglyceridemia.

Funding support: Nil.

Conflict of interest: None.

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