

Correlation between Fatty Liver Index and Obesity on the Incidence of Prediabetes, Type-II Diabetes, and Non-alcoholic Fatty Liver Disease

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ABSTRACT

Our goal was to investigate the relationship between obesity and the fatty liver index and the prevalence of prediabetes, Type II diabetes, and non-alcoholic fatty liver disease. Participants in this study had their ALT and GGT levels evaluated, and questionnaires, blood pressure readings, and venous blood samples were used to determine their body composition. The participants were subsequently separated into groups according to their FLI status and BMI. The results were almost the same as those of primary research. The effects of being overweight or obese were evaluated independently in comparison to those with a BMI of less than 25 kg/m² or less than 30 kg/m². The features of the participants were compared using chi-square testing. Relationships between risk factors and future outcome groups were assessed using linear regression with age and sex adjustments. The study discovered that all risk factors, including body mass index (BMI) and adiposity indicators, were associated with ultrasound-diagnosed type II diabetes and non-alcoholic fatty liver disease (NAFLD), and that there were notable gender variations in BMI. Higher BMI was linked to a higher risk of prediabetes in people who were overweight or obese, but it was not substantially associated with prediabetes risk in those who were normal weight. We concluded that the probability of occurrence prediabetes or type II diabetes linked to being overweight or obese is considerably modulated by the degree of elevation of the FLI.



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1. INTRODUCTION

A growing number of studies are examining the relationship between obesity and the Fatty Liver Index (FLI) in relation to prediabetes, Type-II diabetes, and non-alcoholic fatty liver disease (NAFLD), as these illnesses are becoming more commonplace globally [1]. A diagnostic technique called the Fatty Liver Index uses clinical characteristics such as body mass index (BMI), waist circumference, triglyceride levels, and gamma-glutamyl transferase (GGT) levels to evaluate the risk of non-alcoholic fatty liver disease (NAFLD). It is a useful marker for comprehending the development of metabolic illnesses because it can be used as a non-invasive predictor of the formation of liver fat [2]. A substantial risk factor for the onset of metabolic disorders such as prediabetes, Type-II diabetes, and non-alcoholic fatty liver disease (NAFLD) has long been identified as obesity, especially central obesity [3]. One characteristic of these disorders is insulin resistance, which is exacerbated by excessive fat buildup, particularly in visceral fat [4]. Because the liver plays a crucial role in controlling metabolic processes, there is a particularly significant correlation between obesity and fatty liver disease. Hepatic steatosis, or the buildup of fat in the liver, frequently coexists with insulin resistance and decreased glucose metabolism, both of which may ultimately culminate in prediabetes and Type-II diabetes [5]. Type 2 diabetes and non-alcoholic steatohepatitis (NASH) are clearly correlated; the prevalence of type 2 diabetes is estimated to be 22.5% and 43.6%, respectively, among those with NAFLD and NASH, while the prevalence of NAFLD (liver steatosis and fibrosis) is higher in those with type 2 diabetes than in those who are age and BMI matched [6], [7].

Using metabolic investigations showing hepatic and peripheral insulin resistance in 30 individuals with biopsy-proven NAFLD, as opposed to 10 healthy controls and 10 individuals with type II diabetes, a study initially proposed that NAFLD was the liver-related manifestation of the syndrome known as metabolic syndrome [9]. Nevertheless, when it was shown that high ALT, a biochemical proxy of NAFLD, linked to elevated hepatic glucose production and hepatic insulin resistance, could indicate the onset of type 2 diabetes, the role of fatty liver in incident type 2 diabetes was first identified [10]. According to a number of studies, people who have a higher Fatty Liver Index—which indicates a higher risk of NAFLD—are also more likely to develop Type II diabetes and insulin resistance. Furthermore, it has been demonstrated that NAFLD itself accelerates the development of diabetes and prediabetes, resulting in a vicious circle of metabolic dysfunction [11], [12]. Although normal serum ALT concentrations do not rule out the occurrence of NAFLD [14], it is noteworthy that even ALT levels within the upper normal range were linked to greater incidence type 2 diabetes rates [13]. This has led to calls to update the normal reference range of serum ALT concentrations [15]. Even while serum ALT levels typically range from 6 to 40 IU/L, there is a linear dose-response association between them and the risk of metabolic syndrome [16].

According to a comprehensive analysis of all the research evaluating the relationship between ALT levels and type 2 diabetes, there was a 16% higher chance of incident type 2 diabetes for every 5 IU/L increase in serum ALT [17]. Bedogni et al. previously developed a straightforward algorithm (based on BMI, waist circumference, serum triglycerides (TGs), and gamma-glutamyl transferase [GGT]) for the prediction of fatty liver/hepatic steatosis (as detected by ultrasonography) in the general population using data from the Dionysos Nutrition & Liver Study. Recipient operating characteristics area under the curve (ROC-AUC) for the algorithm, known as the fatty liver index (FLI), was 0.85 (95% CI 0.81–0.88). For assessment, selecting people for intensive management counseling, or epidemiological investigations, a FLI of less than 30 can be

used to rule out hepatic steatosis (sensitivity of 87%) or to rule in hepatic steatosis when FLI of greater than 60 (specificity of 86%) [18]. We next used proton magnetic resonance spectroscopy (1H-MRS) to quantitatively assess liver fat in order to validate the FLI as a measure of hepatic steatosis [19]. Importantly, as the seeds of disease are sown much earlier in life, it is crucial to take into account the tight connection between NAFLD and insulin resistance and its cardiometabolic load from childhood [20- 22]. For the purpose of directing preventative actions, early interventions, and individualized treatment plans meant to lessen the burden of these interconnected diseases, the relationship between the FLI, obesity, and the incidence of prediabetes, Type-II diabetes, and NAFLD is essential. Thus, investigating the connection between obesity and the Fatty Liver Index in relation to metabolic diseases like prediabetes, Type II diabetes, and non-alcoholic fatty liver disease (NAFLD) offers important insights into the pathophysiology of these conditions and emphasizes the necessity of integrated approaches to their management and prevention. Thus, our goal was to investigate the relationship between obesity and the fatty liver index and the prevalence of prediabetes, Type II diabetes, and non-alcoholic fatty liver disease.

2. RESEARCH METHODOLOGY

The present cross-sectional study was conducted in multi-settings e.g., Department of Internal Medicine, CMH Institute of Medical Sciences Bahawalpur, Pakistan; Department of Medicine, Dow University of Health Sciences (DUHS) Karachi, Pakistan; and Department of Medicine, King Edward Medical University/Mayo Hospital, Lahore, Pakistan during the period of January 2024 to February 2025. The study was supervised by Aichi Medical College, Demra, Dhaka, Bangladesh; Emergency Department, Yeti Hospital, Pvt. Ltd., Nepal; and Al-Amiri Hospital Kuwait. Participants whose ALT and GGT levels were tested were included in the sample for this research. Participants who had type 2 diabetes at baseline ($n = 8$), type 1 diabetes mellitus ($n = 14$), or were pregnant at either time ($n = 48$) were not included. Local ethics committees approved the study, and participants provided documented knowledgeable permission. Questionnaires, blood pressure, and body composition: Measurements were taken of waist circumference, height, and weight. Three blood pressure readings were averaged for the purpose of evaluation. Questionnaires were used to ask about drinking and smoking habits. Following a 12-hour fast, venous blood samples were drawn from the right antecubital vein. Serum glucose and TG concentrations were measured using a clinical analyzer and an enzymatic method (Olympus System Reagent; Olympus Diagnostica GmbH, Hamburg, Germany). An automated analyzer was used to measure the enzymatic levels of serum ALT and GGT. For ALT and GGT, the mean inter-assay coefficient of variation was 3.7% and 2.1%, respectively. Based on their BMI and FLI status at both time points, participants were further divided into groups. FLI was classified as either high (≥ 60) (FLI+) or low (< 60) (FLI-). Using cut-offs for high FLI as the ≥ 85 th, ≥ 80 th, and 75th percentiles for FLI-value, we conducted sensitivity analyses. The outcomes were nearly identical to those of the primary studies. The effects of being overweight (BMI ≥ 25 kg/m²) or obese (BMI ≥ 30 kg/m²) (BMI+) in comparison to having a BMI < 25 kg/m² or a BMI < 30 kg/m² (BMI-) were assessed by independent studies. Studies using ultrasound were conducted. Adult abdominal transducers operating at 4.0 MHz were used to scan the liver fat. Five generally recognized criteria for fatty liver were used by a qualified sonographer to score the liver fat status from the ultrasonography pictures [20], [21]. Chi-square tests were used as necessary to compare participant characteristics. To evaluate relationships between risk factors and future outcome groups (pre-diabetes and type 2 diabetes), linear regression with age and sex adjustments was employed. Using a logistic regression model adjusted for age and sex, odds ratios and 95% CIs were computed. The statistical analysis was conducted using SPSS 23.0. For statistical significance, a p value of less than 0.05 was used.

3. RESULTS AND DISCUSSION

Table 1 displays participant characteristics by gender distribution and by type 2 diabetes/pre-diabetes status.

Serum glucose, liver enzyme concentrations (serum ALT and GGT), and TG concentrations all showed significant gender differences in BMI (p always $<.0001$). All risk factors, comprising adiposity indicators (waist circumference and BMI) and FLI, were linked to ultrasound-diagnosed NAFLD and type II diabetes, according to our study of the connection between NAFLD risk factors and type II diabetes in Table 2. In predicting prediabetes, Table 3a displays the odds ratio anticipates (OR 95% CI) for the following groups: normal weight and low-FLI (BMI $-$, FLI $-$) (reference group), normal weight and high-FLI (BMI $-$, FLI $+$), overweight but low-FLI (BMI $+$, FLI $-$), and overweight/obesity and high-FLI (BMI $+$, FLI $+$). Higher FLIs were not significantly associated with raise the risk of prediabetes in those participating of normal weight compared with individuals who had reduced FLIs; nevertheless, among individuals who were overweight or obese, a higher FLI was linked to a substantially greater likelihood of prediabetes than those with lower FLIs. The odds ratio estimates (OR 95% CI) for type 2 diabetes prediction are displayed in Table 3b for the reference group, which consists of individuals with normal weight and low-FLI (BMI $-$, FLI $-$), those who are overweight or obese but have low-FLI (BMI $+$, FLI $-$), and those who are overweight or obese and have a high-FLI (BMI $+$, FLI $+$). There were not enough normal-weight persons with high-FLI (BMI $-$, FLI $+$) to do analysis. A higher FLI did not raise the risk of type 2 diabetes in persons of normal weight. However, compared to persons with a lower FLI, those with a higher FLI who were overweight or obese had a significantly increased risk of type 2 diabetes.

Table 1: Characteristics of Study Population

Parameters	Male	Female	Incident prediabetes	Incident T2D	p-Value
Age (years)	5.0	4.9	4.9	4.6	.011
BMI (kg/m ²)	4.0	4.6	4.3	6.2	$<.0001$
Waist circumference (cm)	10.7	11.2	11.7	14.7	$<.0001$
ALT (mmol/L)	10.3	5.3	9.2	10.6	$<.0001$
GGT (U/L)	26.2	12.9	19.1	24.0	$<.0001$
TG (mmol/L)	1.02	0.70	1.05	0.86	$<.0001$
FLI (units)	40.1 (5.1–92.7)	19.7 (2.3–76.2)	34.9 (3.3–90.2)	64.2 (4.8–98.1)	$<.0001$
Glucose (mmol/L)	0.58	0.45	0.32	0.48	$<.0001$

ALT: alanine aminotransferase; BMI: body mass index; FLI: fatty liver index (95% confidence intervals); GGT: gamma-glutamyl transferase; TG: triglycerides.

Table 2: Independent association between risk factors in NAFLD and Type 2 diabetes (T2D)

Risk factor	NAFLD p Value	T2D p Value
Waist circumference (cm)	$<.0001$	.01
BMI	$<.0001$	.009
(log)FLI	$<.0001$	.017
(log)ALT	$<.0001$	.05
(log)GGT	$<.0001$	.31
(log)TG	$<.0001$	.78

Table 3a: Odds ratio (OR) and 95% confidence intervals (CI) for incident prediabetes according to body mass index (BMI) and fatty liver index (FLI) status

	%	OR (95% CI)	p Value
BMI < 25 kg/m²			
(BMI−, FLI−)	62.3	Reference	Reference
(BMI−, FLI+)	2.5	2.06 (0.50–8.41)	.31
(BMI+, FLI−)	23.7	1.68 (1.33–2.14)	<.0001
(BMI+, FLI+)	11.5	2.12 (1.57–2.85)	<.0001
BMI < 30 kg/m²			
(BMI−, FLI−)	81.3	Reference	Reference
(BMI−, FLI+)	9.0	1.87 (1.26–2.77)	<.0001
(BMI+, FLI−)	1.7	4.47 (2.17–9.23)	<.0001
(BMI+, FLI+)	8.0	1.87 (1.28–2.72)	<.0001

Table 3b: Odds ratio (OR) and 95% confidence intervals (CI) for according to body mass index (BMI) and fatty liver index (FLI) status

	OR (95% CI)	(p Value)
BMI < 25 kg/m²		
(BMI−, FLI−)	Reference	Reference
(BMI−, FLI+)	--	--
(BMI+, FLI−)	0.99 (0.42–2.28)	Insignificant
(BMI+, FLI+)	10.3 (5.69–18.4)	<.0001
BMI < 30 kg/m²		
(BMI−, FLI−)	Reference	Reference
(BMI−, FLI+)	1.08 (0.25–4.76)	Insignificant
(BMI+, FLI−)	1.62 (0.21–12.4)	Insignificant
(BMI+, FLI+)	8.94 (4.86–16.5)	<.0001

Furthermore, higher FLI values elevated the risk of type 2 diabetes by more than 10 and 15 times in individuals who were overweight or obese when compared to similar BMI-matched individuals with lower FLI. Higher FLI values also increased the risk of prediabetes by more than 3 times in participants who were overweight or obese. This demonstrates a risk stratification for occurrence prediabetes and type 2 diabetes based on weight classification and the existence or absence of NAFLD, an indicator of liver fat accumulation [22]. But as a quick and reliable indicator of hepatic steatosis in the general population, we employed the FLI. FLI was initially created utilizing ultrasonography, as the authors initially proposed. and we next used proton magnetic resonance spectroscopy (1H-MRS) to confirm its application in predicting the risk of NAFLD in healthy controls and insulin-resistant obese people [24]. Previous research has shown that type 2 diabetes is associated with greater FLI [25– 27]. In a different study that looked at the relationships between incident type 2 diabetes and fatty liver, the HR of incident type 2 diabetes increased steadily across FLI categories of 10, increasing by 1.8% for every unit rise in FLI [28]. According to the same FLI groupings—≤30, 30–≤ 60, or > 60—a similar association with FLI was observed in a follow-up study of Korean people. The odds ratio for new-onset type 2 diabetes for FLI < 30 vs. 30–59 vs. ≥ 60 was 1.87 (1.05, 3.33) and 2.84 (1.4, 5.75) [29]. Remarkably, a new investigation showed that the risk of incident type II diabetes is influenced by changes in NAFLD status over time, as assessed by serial FLI and abdominal ultrasonography: the risk with cured NAFLD was not substantially distinct from that with no NAFLD [30]. By investigating the collaborative risk between weight status (overweight/obese) and the presence/absence of NAFLD, this study aims to provide additional understanding. In contrast, those who were obese and MUO with a high FLI had an approximately twofold increased risk of incident type 2 diabetes [31]. The risk of incident type II diabetes was not significantly higher than that of the MHO non-

obese. In a study examining the dosage response between liver fat and metabolic end points, the mechanism behind this association between liver fat, metabolic syndrome, and type II diabetes was examined [32]. Skeletal muscle insulin resistance, hypertriglyceridemia, and low HDL-cholesterol become noticeable when liver fat accumulation surpasses $6 \pm 2\%$, which is marginally higher than the upper limit of normal liver fat in the general population (5.56%), as established by the Dallas Heart Study [33]. Others have observed a link between type 2 diabetes and liver fibrosis in addition to steatosis. Bjorkstrom et al. conducted a retrospective analysis of 396 patients with biopsy-proven non-alcoholic fatty liver disease (NAFLD) and discovered that patients with fibrosis stages 3–2 were more likely to be diagnosed with type 2 diabetes than those with stages 0–2, while patients with fibrosis stages 0–2 had a higher risk of type 2 diabetes based on their fat score [34]. One of the study's shortcomings is that only a tiny percentage of patients went on to acquire type 2 diabetes.

4. CONCLUSION

We concluded that the probability of occurrence prediabetes or type II diabetes linked to being overweight or obese is considerably modulated by the degree of elevation of the FLI. In people who are overweight or obese, an elevated FLI dramatically raises the risk of incident prediabetes, type II diabetes, and non-alcoholic fatty liver disease (NAFLD), underscoring the part that liver fat buildup plays in the pathogenesis of prediabetes and type 2 diabetes.

AUTHOR'S CONTRIBUTION

Shahriar Ahmed: “Contributions of a significant nature to the conception of the work; or the acquisition, collection, analysis, or interpretation of data for the work; AND Final approval of the version that is for publication.”

Roshni Lamichhane: “drafting the work or conducting a critical evaluation of it to identify significant intellectual content; AND obtaining final approval of the version that will be included in the publication.”

Naheed Akhter: “The conception or design of the work; the gathering, examination, or interpretation of facts for the work; or any combination of these.”

Nargis Khan: “Substantial contributions to the conception or design of the work; or the acquisition.”

Gull Hassan Sethar: “drafting the work or conduct a critical review of draft to identify significant intellectual substance.”

Imran Mahfooz Khan: “The process of drafting the work or conducting a critical review of it to identify significant intellectual substance.”

Rabia Zulfiqar: “Creating a draft of the work or conducting a critical evaluation of it to identify significant intellectual substance; It is agreed upon that one will be responsible for all parts of the job, including ensuring that any questions concerning the accuracy or integrity of any portion of the work are appropriately examined and handled.

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