Diabetes, Metabolic Syndrome and Obesity

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

High Prevalence of Fatty Liver in Bangladeshi Adolescents and Young Adults with Type 2 Diabetes: Key Predictors and Screening Recommendations

Kishore Kumar Shil[1], Mashfiqul Hasan[2], Nusrat Sultana[2], Sayad Bin Abdus Salam[2], Syeda Nazlee Mostafa[3], Muhammad Abul Hasanat[2]

[1]Department of Endocrinology, Khulna Medical College Hospital, Khulna, Bangladesh; [2]Department of Endocrinology, Bangladesh Medical University, Dhaka, Bangladesh; [3]Department of Radiology and Imaging, Bangladesh Medical University, Dhaka, Bangladesh

Correspondence: Kishore Kumar Shil, Department of Endocrinology, Khulna Medical College Hospital, Khulna, Bangladesh, Tel +8801723512712, Email kishorshil31039@gmail.com

Background: Obesity and insulin resistance are the key pathogenic factors for both metabolic dysfunction-associated steatotic liver disease (MASLD) and type-2 diabetes (T2DM), and their rising prevalence is driving an increase in MASLD cases.

Objective: To assess the presence of fatty liver disease by ultrasonography (USG) and to find out its predictors in newly diagnosed young-onset T2DM.

Methods: This cross-sectional study enrolled 137 newly diagnosed adolescents and young adult T2DM subjects (aged 10–34 years) by non-probability purposive sampling at a tertiary hospital in Bangladesh. Individuals with a history of alcoholism, pregnancy, infections, endocrinopathies, genetic disorders, or drugs that can cause fatty liver were excluded. Fatty liver was assessed by ultrasonography. C-peptide, anti-GAD65, and anti-IA2 antibodies were measured by chemiluminescent immunoassay, and anti-ZnT8 antibodies by ELISA.

Results: Among 137 newly diagnosed adolescents and young adult T2DM patients, 92 (68.1%) had fatty liver on ultrasonography—58 (63%) with grade 1 and 34 (37%) with grade 2. Most were overweight/obese (67.2%), had central obesity (88.3%), and reported low physical activity (68.6%). Fatty liver was significantly associated with older age, higher BMI, WHR, and a positive family history of diabetes. On multivariate analysis, age (OR 1.14, 95% CI 1.04–1.25, p=0.004), positive family history of DM (OR 3.21, 95% CI 1.29–8.22, p=0.012), acanthosis nigricans (OR: 5.05, 95% CI 1.59–16.04, p=0.006), and fasting C-peptide levels (OR 1.46, 95% CI 1.10–1.92; p = 0.007) were significant predictors of fatty liver.

Conclusion: Fatty liver is highly prevalent in young-onset T2DM at diagnosis. Age, a positive family history of diabetes, acanthosis nigricans, and elevated fasting C-peptide levels are strong predictors. Routine use of ultrasonography may facilitate early detection and management of liver disease in this population.

Keywords: fatty liver, risk factors, young onset T2DM

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) represents the most common chronic liver condition in individuals with type 2 diabetes (T2DM).[1] In the absence of secondary causes of steatosis or significant alcohol consumption, patients with liver disease ranging from hepatic steatosis to cirrhosis are considered to have MASLD. Independent of obesity status, insulin resistance serves as a primary driver of this condition.[2,3] Hence, MASLD has been recognized as the hepatic component of metabolic syndrome.[4] Hyperinsulinemia and insulin resistance contribute to impaired lipid metabolism, hepatic triglyceride (TG) accumulation in MASLD, and β-cell dysfunction in T2DM.[5–7] Hyperinsulinemia, a compensatory response to insulin resistance, plays a central role in the progression of Metabolic

Received: 5 February 2025
Accepted: 5 June 2025
Published: 24 June 2025

dysfunction-associated steatotic liver disease (MASLD). It drives hepatic fat accumulation through increased de novo lipogenesis, reduced fatty acid oxidation, and elevated FFA influx from insulin-resistant adipose tissue. These changes promote steatosis, oxidative stress, and inflammation, contributing to steatohepatitis (MASH) and fibrosis development. Additionally, hyperinsulinemia activates hepatic stellate cells and fibrogenic pathways, exacerbating liver injury.[8] It has emerged as a significant public health concern and is largely attributed to the widespread availability of high-calorie foods and the adoption of sedentary lifestyles.[9] According to estimates, individuals with MASLD who do not properly manage their comorbidities may be at risk for up to 66% of all-cause deaths and 83% of cardiovascular deaths.[10] The risk of MASLD is influenced by genetic factors, including single nucleotide polymorphisms in specific genes such as patatin-like phospholipase domain-containing protein 3 (PNPLA3), which has been associated with the regulation of triacylglycerol production. Ethnicity also plays a significant role, with Hispanics demonstrating a higher predisposition to the condition.[11] South Asians, who are predisposed to visceral adiposity, may also exhibit a higher prevalence of MASLD. The global pooled prevalence of MASLD among patients with T2DM adults is approximately 65.33%. Regionally, prevalence is highest in Eastern Europe (80.62%), followed by the Middle East (71.24%), and lowest in Africa (53.10%).[12] However, the rate of MASLD at diagnosis of young-onset T2DM is unclear. The prevalence of MASLD was 27.5% in children and adolescents with T1D.[13] A study conducted in Bangladesh in 2006 revealed that 36% of children and adolescents without diabetes mellitus had liver steatosis.[14] On the other hand, young-onset T2DM is characterized by a relatively aggressive disease course with rapid decline of beta-cell function and early onset of chronic complications. Hence it is expected that there might be a high burden of hepatic steatosis in this group which is also reflected by previous studies.[15] Early-onset MASLD is linked to higher risks of liver fibrosis, cirrhosis, and cardiovascular events. Both steatotic liver disease and MASH may progress to cirrhosis in up to 25% of cases over 10–20 years. Youth with biopsy-confirmed NAFLD face increased rates of MACE, including heart disease and heart failure.[16,17] As MASLD is typically an asymptomatic condition, its prevalence in people with T2DM remains largely undetected. Although liver biopsy is the gold standard for diagnosing MASLD, it is rarely performed unless clinically indicated. Transabdominal ultrasound, however, serves as a valuable tool for assessing hepatic steatosis. Given the increasing prevalence of obesity and T2DM among younger individuals over the past few decades in our country, a higher prevalence of MASLD is anticipated. So, Routine ultrasonography may be helpful for all newly diagnosed T2DM patients to enable early detection of hepatic steatosis. Consequently, the present study was designed to determine the frequency of steatotic changes in the liver among individuals with newly diagnosed adolescent and young adult T2DM and to identify predictors of these changes within this specific population group.

Materials and Methods

Study Participants and Study Design

This cross-sectional study was conducted in the Department of Endocrinology, Bangladesh Medical University (BMU), Dhaka, Bangladesh as part of an ongoing research initiative by the Study of Diabetes in Young (SODY) group, which focuses on evaluating insulin resistance, β-cell secretory capacity, autoantibodies, and genes associated with Maturity Onset Diabetes of the Young (MODY). In this study, abdominal ultrasonography (USG) was performed to assess pancreatic morphology, which also provided information regarding fatty changes in the liver. The present analysis included 137 newly diagnosed adolescents and young adult phenotypical T2DM patients enrolled by non-probability purposive sampling, diagnosed according to the American Diabetes Association (ADA) criteria (age range: 10–34 years). The sample size was determined using the following formula: $\boldsymbol{n} = \frac{z^2 pq}{d^2}$

Although there is no specific data on the prevalence of MASLD in young individuals with type 2 diabetes mellitus (T2DM), the global prevalence of MASLD among adults with T2DM has been reported as 65.3%.[12] Assuming this prevalence, with a 95% confidence level and a margin of error of 8%, the required sample size was calculated to be 137.

Individuals with a history of alcoholism, pregnancy, chronic infections (such as hepatitis C), chronic liver or kidney disease, endocrinopathies, genetic disorders, or the use of medications known to cause fatty liver or affect endogenous insulin and C-peptide levels were excluded from the study.

Study Procedure

A comprehensive history and detailed clinical evaluation were conducted for all participants. Height was measured using a stadiometer, while waist circumference (WC) was assessed to the nearest centimeter using a flexible steel tape. Blood pressure (BP) was recorded in millimeters of mercury using a standard sphygmomanometer. Demographic and clinical characteristics of the patients were systematically documented using a standardized, pre-tested structured datasheet. Participants or their eligible attendants were interviewed regarding past medical history and family history. Fasting blood samples were collected from each participant to measure C-peptide levels and islet autoantibodies, enabling the exclusion of type 1 diabetes (T1DM). The presence of autoantibodies was considered positive at the following thresholds: glutamic acid decarboxylase autoantibody (GAD Ab) ≥5 U/mL, zinc transporter 8 autoantibody (ZnT8 Ab) ≥15 U/mL, and islet antigen-2 autoantibody (IA2 Ab) ≥7 U/mL. C-peptide levels were categorized as low if <0.9 ng/mL and adequate if ≥0.9 ng/mL. Individuals with low C-peptide levels (<0.9 ng/mL) and positivity for two or more autoantibodies were diagnosed with type 1 diabetes mellitus (T1DM) and excluded from the study. MODY cases were excluded based on clinical criteria: Individuals with diabetes diagnosed under 25 years of age, who do not fully fit the phenotypes of T1DM (negative autoantibody and low C-peptide level) or T2DM (obesity and feature of insulin resistance) and who have a strong family history of diabetes (vertical transmission of disease in three generations (autosomal dominant mode of inheritance) with at least one family member with onset of disease <25 years of age).

Body mass index (BMI) for participants under 18 years of age was categorized based on the official Centers for Disease Control and Prevention (CDC) guidelines: BMI between the 5th and 85th percentile was classified as normal, the 85th to 95th percentile as overweight, and above the 95th percentile as obese.[18] BMI classification for participants aged 18 years or older followed the World Health Organization (WHO) criteria for adult obesity (Male: WC - ≥90 cm; Female: WC - ≥80 cm) specific to the Asian population. Abnormal WC for participants under 18 was defined as exceeding the 90th percentile for age and sex.[19] Abdominal obesity was defined as WHR ≥0.88 for males and ≥0.81 for females for adults. Abnormal WHR for participants <18 was defined as exceeding the 90th percentile for age and sex.

Real-time abdominal ultrasonography (USG) was conducted to diagnose fatty liver, utilizing a 3.5 MHz curvilinear transducer with SONO ACE 8000 and Sonoline Antares ultrasound machines following a 6-hour fasting period. On USG, a hyperechogenic ("bright liver") appearance indicated hepatic steatosis. The severity of hepatic steatosis was graded as 1, 2, or 3. Grade 1 was characterized by mildly increased liver echogenicity with normal visualization of vessels and no posterior attenuation. Grade 2 exhibited moderate echogenicity with partial vessel obscuration and early posterior attenuation. Grade 3 presented as a diffuse increase in echogenicity, complete vessel obscuration, and pronounced posterior attenuation.[20]

C-peptide, glutamic acid decarboxylase antibody (GAD65 Ab), and anti-IA2 antibody (Anti-IA2 Ab) levels were measured using a chemiluminescent immunometric assay on the MAGLUMI 2000 Plus analyzer (Snibe, China), with intra-assay coefficients of variation (CV) of 5%, 3.6%, and 3.4%, respectively. Serum ZnT8 antibody levels were determined using an enzyme-linked immunosorbent assay (ELISA) kit (RSR™ ZnT8 Ab™, RSR Ltd., Cardiff, UK), demonstrating 72% sensitivity and 99% specificity, with an intra-assay CV of 4.39%.

Statistical Analysis

Statistical analyses were performed by using IBM SPSS Statistics for Windows version 25.0. Quantitative data were expressed as mean ±SD or median and interquartile range (IQR), whereas qualitative data were expressed as frequency distribution and percentage. The association between categorical variables was analyzed by the χ^2 test and the continuous variable by Student's *t*-test. Multivariate logistic regression was done for the predictors of fatty liver. A P-value of less than 0.05 was considered statistically significant for all statistical tests.

Ethical Considerations

Voluntary informed written consent was taken from each participant and/or their legal guardian after thoroughly explaining the procedure and purpose of the study. Each participant had the right to participate, refuse, or withdraw from the study at any point without any repercussions. All individuals received appropriate medical care and guidance

regardless of their enrollment status. Information of the patients was kept confidential. Proper counseling was done before the collection of blood samples. Adequate safety measures were also taken in every step of sample collection. The project ran after approval from the Institutional Review Board (IRB), Bangladesh Medical University, Dhaka, Bangladesh (IRB approval ID 3823). Our study complies with the Declaration of Helsinki.

Results

The study enrolled 137 adolescents and young adult subjects with newly diagnosed phenotypic T2DM to assess the prevalence and predictors of fatty liver who were diagnosed based on ultrasonographic criteria. No one fulfilled the requirements of T1DM depending on the auto-antibody positivity and C-peptide level.

The clinical characteristics of the study participants are summarized in Table 1. The age of the participants ranged from 10 to 34 years, with a mean ± standard deviation (SD) of 25.0 ± 5.8 years. A substantial proportion of the participants exhibited elevated BMI and WHR, at 67.2% and 88.3%, respectively. Most participants reported light physical activity (68.6%). Fatty liver was identified in 92 participants (68.1%), of whom 63% had grade 1 fatty liver and 34 37% had grade 2 (Figure 1).

Participants with fatty liver had higher age (26.0±5.9 vs 23.0±5.1 years; p=0.002), WHR (0.94±0.14 vs 0.89±0.08; p=0.02], frequency of overweight/obesity (78.3% vs 44.5%; p<0.001), and a positive family history of DM (72.8% vs 40%; p<0.001). The fatty liver group had lower HbA1c levels (9.3±2.3 vs 10.7±3.8%; p=0.02) but higher fasting C-peptide levels (5.2±2.7 vs 3.3±2.3 ng/mL; p<0.001) (Table 2). There was no significant difference in lipid levels.

Table 1 Demographic and Clinical Characteristics of the Study Participants (n=137)

Variables	Value
Age (years; Mean ±SD)	25.0±5.8
Gender	
Male	62 (45.3%)
Female	75 (54.7%)
Family history[a]	
Present	85 (62%)
Absent	52 (38%)
Acanthosis Nigricans	
Present	74 (54%)
Absent	63 (46%)
WC (cm)	
Normal	53 (38.7%)
High	84 (61.3%)
Physical activity level	
Light	94 (68.6%)
Moderate	41 (29.9%)
Vigorous	2 (1.5%)
WHR	
Normal	16 (11.7%)
High	121 (88.3%)
BMI (Kg/m^2)	
Normal	45 (32.8%)
Overweight/Obese	92 (67.2%)

Notes: Mean ± SD for normally distributed data. Percentages are over column total. Family history[a]: Family history of of DM in 10 relatives.
Abbreviations: DM, diabetes mellitus of young; HC, Hip circumference; BMI, body mass index; C, Waist circumference; WHR, Waist-hip ratio.

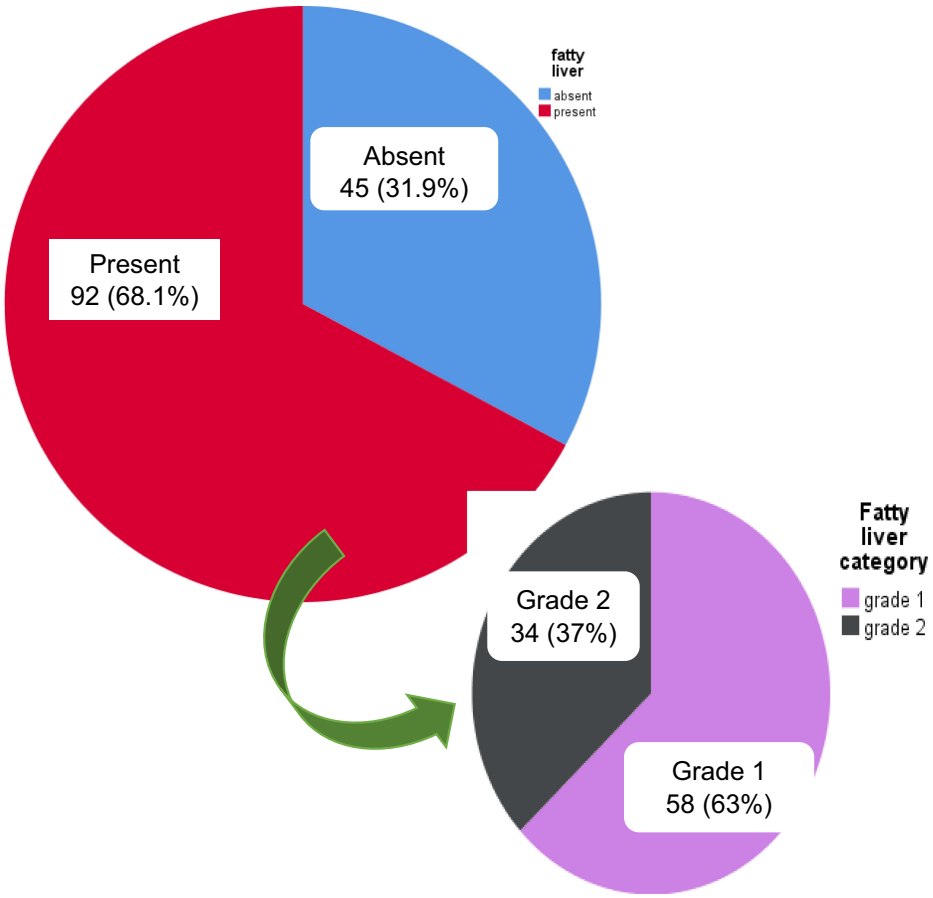

Figure 1 Prevalence of fatty liver and its severity among the study participants (n=137).

On Multiple logistic regression analysis, age (OR 1.14, 95% CI 1.04–1.25, p=0.004), positive family history of DM (OR 3.21, 95% CI 1.29–8.22, p=0.012), acanthosis nigricans (OR: 5.05, 95% CI 1.59–16.04, p=0.006), and fasting C-peptide levels (OR 1.46, 95% CI 1.10–1.92; p = 0.007) identified as significant predictors of fatty liver (Table 3).

Table 2 Clinical and Biochemical Characteristics of Fatty Liver Categories (N=137)

Characteristics	Group		p-value[a]
	Present (n=92)	Absent (n=45)	
Age (years; mean ±SD)	26.0±5.9	23.0±5.1	0.002
Age category (years)			
10–18	11 (11.95%)	10 (22.2%)	0.11
≥ 19	81 (88.92%)	35 (77.7%)	
Gender			
Male	44 (47.8%)	18 (40%)	0.38
Female	48 (52.17%)	27 (60%)	
BMI category; n (%)			
Normal	20 (21.7%)	25 (55.5%)	<0.001
Overweight/obese	72 (78.3%)	20 (44.5%)	
Waist-hip ratio (cm, mean ±SD)	0.94±0.14	0.89±0.08	0.02

(*Continued*)

https://doi.org/10.2147/DMSO.S520806

Table 2 (Continued).

Characteristics	Group		p-value[a]
	Present (n=92)	Absent (n=45)	
Acanthosis nigricans; n (%)			
Present	47 (51.0%)	27 (60%)	0.32
Absent	45 (49.0%)	18 (40%)	
[b]Family history of DM; n (%)	67 (72.8%)	18 (40%)	<0.001
Physical activity; n (%)			
Low	66 (71.7%)	28 (62.2%)	0.503
Middle	25 (27.1%)	16 (35.5%)	
High	1 (1.08%)	1 (1.08%)	
FPG (mmol/L, mean ±SD)	11.6±5.0	13.2±6.3	0.10
2h PG (mmol/L, mean ±SD)	18.9±6.8	20.7±7.8	0.17
HbA1c (%, mean ±SD)	9.3±2.3	10.7±3.8	0.02
TC (mg/dl; mean ±SD)	191.6±48.6	177.4±44.3	0.10
LDL-C (mg/dl; mean ±SD)	103.6±35.6	102.0±36.6	0.82
HDL-C (mg/dl; mean ±SD)	37.2±8.0	39.0±9.0	0.22
TG (mg/dl; mean ±SD)	277.1±215.5	226.1±131.4	0.14
Fasting C-peptide (ng/mL; mean ±SD)	5.2±2.7	3.3±2.3	<0.001

Notes: p-value[a] was measured by χ^2, Fisher's Exact test, and independent *t*-test where applicable. Percentages are over the column total if not otherwise mentioned. [b]Family history of diabetes- positive family history of diabetes in 1st-degree relatives.

Abbreviations: BMI, body mass index; FPG, fasting plasma glucose; 2h PG, 2 hours after 75 plasma glucose; HbA1c, glycated hemoglobin; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TG, triglyceride; MASLD, metabolic dysfunction associated steatotic liver disease.

Table 3 Multivariate Logistic Regression for the Predictors of Fatty Liver

Variables	OR	P	95% CI	
			Lower	Upper
Age (each year increase)	1.14	0.004	1.04	1.25
Family history of diabetes	3.21	0.012	1.29	8.22
Physical activity	0.78	0.63	0.28	2.14
BMI (kg/m^2)	1.12	0.12	0.97	1.29
Acanthosis nigricans	5.05	0.006	1.59	16.04
C-peptide (per ng/mL increase)	1.46	0.007	1.10	1.92
TG (per mg/dl increase)	1.00	0.36	0.99	1.00

Notes: R^2= 32.2–44.8%. OR- Exp(B); p <0.05 is significant.

Abbreviations: BMI, body mass index; TG, triglyceride; 95% CI, 95% confidence interval.

Discussion

With the rising frequency of obesity and metabolic syndrome, the prevalence of MASLD has sharply increased worldwide.[21] MASLD is becoming a public health concern. A wide range of illnesses, including advanced fibrosis, cirrhosis, hepatocellular carcinoma, and simple steatosis, are included in MASLD. These illnesses significantly increase the morbidity and mortality associated with liver disease.[22,23] The prevalence of MASLD in the general population varies between 15–40% in Western countries and between 9–40% in Asian countries.[24] By imaging, the global pooled prevalence of fatty liver is 25.2% among the general population. The prevalence is high among diabetic subjects due to genetic and environmental influences. The relationship between MASLD and T2DM seems to be bidirectional; an

increase in T2DM prevalence may lead to a rise in MASLD cases, and conversely, an increase in MASLD may also elevate the prevalence of T2DM. This correlation suggests the possibility of a genetic connection, along with obesity and insulin resistance, serving as potential predictors for both T2DM and MASLD.[25] In India, the prevalence of MASLD in individuals with T2DM varies greatly, from 45.0% to 72.0%.[26] The global prevalence of MASLD among adults with type 2 diabetes is approximately 65.33%, with the highest rates in Eastern Europe (80.62%) and the lowest in Africa (53.10%).[12] In South Asia, the prevalence of NAFLD in adults with type 2 diabetes ranges from 50% to 75%. Compared to Europeans, South Asians are more vulnerable to metabolic syndrome, T2DM, and MASLD due to differences in body composition, particularly higher adiposity and lower muscle mass. Lifestyle changes are a major factor driving the increasing rates of fatty liver disease, obesity, and T2DM in South Asia. Although South Asians may not appear as overtly obese as other ethnic groups, they tend to be more metabolically obese.[27] In our study, two-thirds of the participants with young-onset T2DM had fatty liver. This result emphasizes how critical it is to assess and treat MASLD as soon as possible.

According to a study by Yi et al, men were more likely than women to have MASLD.[28] According to our research, female patients with T2DM had a greater prevalence of MASLD.[29] Several studies have shown that the prevalence of MASLD is increasing in the elderly due to an increase in obesity and insulin resistance, with a higher proportion of patients falling between the ages of 40 and 60.[28] Additionally, our investigation also revealed that increasing age is a significant predictor of fatty liver. One of the risk factors for the onset of MASLD is dyslipidemia. Numerous investigations have revealed that 20–92% of individuals suffer from hyperlipidemia, elevated cholesterol, elevated triglycerides, or both.[30] We did not observe any significant difference in lipid profiles between participants with or without fatty change in liver. We have recruited only young subjects, and their eating habits may also have contributed to this variation in lipid profiles. According to Gupte et al, 65%, 12.5%, and 92.5% of people with T2DM had mild, moderate, or severe MASLD.[31] Banerjee et al demonstrated that among patients with T2DM, fatty alterations were seen in 43% of cases, cirrhosis in 20%, and NASH in 40%.[31] Also, we found that 34 (37%) and 58 (63%) had grade 2 and grade 1 fatty livers, respectively. There is a well-established correlation between MASLD and T2DM, which may be attributed to factors such as obesity, sedentary lifestyle, insulin resistance, compensatory hyperinsulinemia, impaired lipid metabolism, and hepatic triglyceride (TG) accumulation.[5–7]

In our study, we also found that obesity, physical inactivity, insulin resistance and compensatory hyperinsulinemia are the major contributors to fatty liver in young T2DM subjects. As, majority of the participants' BMI and WHR were high and most of the participants' physical activity was light. WHR and positive family history of DM had a significant association with the MASLD group, and fasting C-peptide was significantly high among the fatty liver categories. In multiple logistic regression, age, positive family history of DM, acanthosis nigricans, and fasting C-peptide levels were significant predictors of fatty liver. While obesity and insulin resistance are primary contributors to both diabetes and fatty liver, secondary causes of fatty liver also exist. In our study, these were excluded based on clinical history alone. We did not assess dietary intake (eg, high-calorie or high-fructose diets) or liver inflammation markers (ALT, AST, CRP), as this study was done as part of the young diabetes study. Although the cross-sectional design is suitable for evaluating prevalence and associated risk factors, it limits causal interpretation and does not capture disease progression. Future prospective studies are needed to track MASLD progression in young-onset T2DM. In summary, the high prevalence of fatty liver disease in young-onset diabetes warrants urgent attention, and routine screening at diagnosis is recommended to detect fatty liver early and promote better liver health in these patients.

Conclusion

Prevalence of fatty liver is alarmingly high in newly diagnosed young-onset T2DM subjects in our country. Rapidly rising prevalence of obesity, physical inactivity, genetic background (positive family history) and insulin resistance may lead to high prevalence of T2DM as well as MASLD. Age, positive family history of DM, acanthosis nigricans, and fasting C-peptide are significant predictors of fatty liver. Therefore, early screening and management of fatty liver in individuals with T2DM should be a growing priority to reduce the burden of liver-related complications.

Acknowledgment

The authors acknowledge the contribution of the Department of Microbiology, BMU for their technical help and the Study on Diabetes in Young (SODY) group, BMU for their overall support.

Funding

This work was supported as a research grant by Research and Development, BMU, and SODY group, BMU, Dhaka, Bangladesh.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Ludwig J, Viggiano TR, McGill DB, Oh BJ. Nonalcoholic steatohepatitis: mayo clinic experiences with a hitherto unnamed disease. *Mayo Clin Proc*. 1980;55(7):434–438. doi:10.1016/S0025-6196(24)00530-5
2. Harrison SA, Gawrieh S, Roberts K, et al. Prospective evaluation of the prevalence of non-alcoholic fatty liver disease and steatohepatitis in a large middle-aged US cohort. *J Hepatol*. 2021;75(2):284–291. doi:10.1016/j.jhep.2021.02.034
3. Castera L, Cusi K. Diabetes and cirrhosis: current concepts on diagnosis and management. *Hepatology*. 2023;77(6):2128–2146. doi:10.1097/HEP.0000000000000263
4. Rohini I, Aarthi N. A study of prevalence of non alcoholic fatty liver in type 2 diabetes mellitus patients. *Int J Contemporary Med Res*. 2019;6(12):12. doi:10.21276/ijcmr.2019.6.12.48
5. Younossi ZM, Gramlich T, Matteoni CA, Boparai N, McCullough AJ. Nonalcoholic fatty liver disease in patients with type 2 diabetes. *Clin Gastroenterol Hepatol*. 2004;2(3):262–265. doi:10.1016/S1542-3565(04)00014-X
6. Targher G, Bertolini L, Rodella S, et al. Nonalcoholic fatty liver disease is independently associated with an increased incidence of cardiovascular events in type 2 diabetic patients. *Diabetes Care*. 2007;30(8):2119–2121. doi:10.2337/dc07-0349
7. Parving HH, Gall MA, Skøtt P, et al. Prevalence and causes of albuminuria in non-insulin-dependent diabetic patients. *Kidney Int*. 1992;41(4):758–762. doi:10.1038/ki.1992.118
8. Samuel VT, Shulman GI. Nonalcoholic fatty liver disease, insulin resistance, and ceramides. *N Engl J Med*. 2019;381(19):1866–1869. doi:10.1056/NEJMcibr1910023
9. Huang DQ, Noureddin N, Ajmera V, et al. Type 2 diabetes, hepatic decompensation, and hepatocellular carcinoma in patients with non-alcoholic fatty liver disease: an individual participant-level data meta-analysis. *Lancet Gastroenterol Hepatol*. 2023;8(9):829–836. doi:10.1016/S2468-1253(23)00157-7
10. Paik JM, Deshpande R, Golabi P, Younossi I, Henry L, Younossi ZM. The impact of modifiable risk factors on the long-term outcomes of non-alcoholic fatty liver disease. *Aliment Pharmacol Ther*. 2020;51(2):291–304. doi:10.1111/apt.15580
11. William CD, Stengel J, Asike MI, et al. Prevalence of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among a largely middle-aged population utilizing ultrasound and liver biopsy: a prospective study. *Gastroenterology*. 2011;140(1):124–131. doi:10.1053/j.gastro.2010.09.038
12. Younossi ZM, Golabi P, Price JK, et al. The global epidemiology of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among patients with type 2 diabetes. *Clin Gastroenterol Hepatol*. 2024;22(10):1999–2010. doi:10.1016/j.cgh.2024.03.006
13. Maffeis C, Piona C, Morandi A, et al. Glycaemic control metrics and metabolic dysfunction–associated steatotic liver disease in children and adolescents with type 1 diabetes. *Diabetes Obesity Metab*. 2024;26(12):5896–5905. doi:10.1111/dom.15961
14. Shelim R, Mohsin F, Begum T, Baki MA, Mahbuba S, Islam R. Hepatic steatosis among obese children and adolescents. *J Bangladesh College Physicians Surgeons*. 2016;34(2):57–63. doi:10.3329/jbcps.v34i2.32290
15. Hu C, Cioana M, Saini A, et al. The prevalence of non-alcoholic fatty liver disease in pediatric type 2 diabetes: a systematic review and meta-analysis. *Front Adolescent Med*. 2024;2:1303375. doi:10.3389/fradm.2024.1303375
16. Parsa AA, Azama KA, Vawer M, Ona MA, Seto TB. Prevalence Study of MASLD in adolescent and young adult pacific islanders and asians living in Hawaii. *J Endocrine Soc*. 2024;8(3):bvad165. doi:10.1210/jendso/bvad165
17. Chung GE, Yu SJ, Yoo JJ, et al. Metabolic dysfunction-associated steatotic liver disease increases cardiovascular disease risk in young adults. *Sci Rep*. 2025;15(1):5777. doi:10.1038/s41598-025-89293-6
18. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. *CDC Growth Charts*. United States advance data from vital and health statistics, no. 314. Hyattsville, MD: National Center for Health Statistics; 2000:11183293.
19. Xi B, Zong XN, Kelishadi R, et al. International waist circumference percentile cutoffs for central obesity in children and adolescents aged 6 to 18 years. *J Clin Endocrinol Metab*. 2020;105(4):e1569–83. doi:10.1210/clinem/dgz195
20. Saadeh S, younossi ZM, Remer EM, et al. The utility of radiological imaging in nonalcoholic fatty liver disease. *Gastroenterology*. 2002;123(3):745–750. doi:10.1053/gast.2002.35354
21. Younossi Z, Anstee QM, Marietti M, et al. Global burden of MASLD and NASH: trends, predictions, risk factors and prevention. *Nat Rev Gastroenterol Hepatol*. 2018;15(1):11–20. doi:10.1038/nrgastro.2017.109
22. Leite NC, Villela-Nogueira CA, Pannain VL, et al. Histopathological stages of nonalcoholic fatty liver disease in type 2 diabetes: prevalences and correlated factors. *Liver Int*. 2011;31(5):700–706. doi:10.1111/j.1478-3231.2011.02482.x
23. Targher G, Bertolini L, Rodella S, et al. Non-alcoholic fatty liver disease is independently associated with an increased prevalence of chronic kidney disease and proliferative/laser-treated retinopathy in type 2 diabetic patients. *Diabetologia*. 2008;51(3):444–450. doi:10.1007/s00125-007-0897-4

24. Amarapurkar D, Kamani P, Patel N, et al. Prevalence of non-alcoholic fatty liver disease: population based study. *Ann Hepatol*. 2007;6(3):161–163. doi:10.1016/S1665-2681(19)31922-2
25. Younossi ZM, Kalligeros M, Henry L. Epidemiology of metabolic dysfunction-associated steatotic liver disease. *Clin Mol Hepatol*. 2024;31 (Suppl):S32. doi:10.3350/cmh.2024.0431
26. Sinha A, Bankura B. Prevalence of nonalcoholic fatty liver disease in type 2 diabetes mellitus patients from the Eastern region of India. *Diabetes Epidemiol Manag*. 2023;12:100161. doi:10.1016/j.deman.2023.100161
27. Pati GK, Singh SP. Nonalcoholic fatty liver disease in South Asia. *Euroasian J Hepato-Gastroenterol*. 2016;6(2):154. doi:10.5005/jp-journals -10018-1189
28. Yi M, Chen RP, Yang R, Chen H. Increased prevalence and risk of non-alcoholic fatty liver disease in overweight and obese patients with type 2 diabetes in South China. *Diabetic Med*. 2017;34(4):505–513. doi:10.1111/dme.13174
29. Kalra S, Vithalani M, Gulati G, et al. Study of prevalence of nonalcoholic fatty liver disease (MASLD) in type 2 diabetes patients in India (SPRINT). *J Assoc Physicians India*. 2013;61(7):448–453.
30. Reid AE. Nonalcoholic steatohepatitis. *Gastroenterology*. 2001;121(3):710–723. doi:10.1053/gast.2001.27126
31. Gupte P, Amarapurkar D, Agal S, et al. Non-alcoholic steatohepatitis in type 2 diabetes mellitus. *J Gastroenterol Hepatol*. 2004;19(8):854–858. doi:10.1111/j.1440-1746.2004.03312.x