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Unveiling the Metabolic Nexus: Clinical Insights into MAFLD and NAFLD Prevalence and Risk Factors among Patients with Type 2 Diabetes Mellitus

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Abstract

Background: Fatty liver disease has emerged as one of the most common chronic liver conditions worldwide, and its coexistence with type 2 diabetes mellitus (T2DM) has become a growing clinical concern. Traditional diagnosis of non-alcoholic fatty liver disease (NAFLD) required exclusion of significant alcohol intake, whereas the newer metabolic-associated fatty liver disease (MAFLD) framework rests on positive metabolic criteria regardless of alcohol use, a shift with real consequences for how often fatty liver is diagnosed in diabetic populations.

Objective: To determine the prevalence of MAFLD and NAFLD among patients with T2DM attending a tertiary diabetes care center in Islamabad, and to compare the clinical, biochemical, and metabolic profiles between the two diagnostic categories.

Methods: A descriptive cross-sectional study was carried out at a tertiary-care diabetes center in Islamabad, Pakistan, with data collected from December 2024 to May 2025. Sixty-four patients with T2DM were evaluated. Liver enzymes, glycemic markers, lipid profile, anthropometric measurements, and comorbidity data were extracted and analyzed. Patients were categorized according to MAFLD and NAFLD diagnostic criteria, and biomarkers were compared between diagnosis-positive and diagnosis-negative groups using one-way analysis of variance.
Results: The study had a mean age of 51.5 ± 11.8 years with a near-equal gender distribution; 78.1% of patients were classified as obese. Mean HbA1c was 8.5 ± 1.6%, and 88.9% met HbA1c-based criteria for T2DM. Forty-four of 64 patients (68.8%) were categorized as having either MAFLD or NAFLD. Patients positive for MAFLD or NAFLD showed significantly higher ALT, AST, BMI, HbA1c, and triglyceride levels than those negative for these conditions ($p < 0.05$ for most comparisons). Gastric complaints (76%), retinopathy (68%), and hypertension (46%) were the most frequently reported comorbidities.
Conclusion: MAFLD and NAFLD are both highly prevalent among patients with T2DM in this population and are strongly associated with obesity, poor glycemic control, and dyslipidemia. These findings support routine hepatic and metabolic screening in diabetic care and highlight the clinical relevance of adopting the MAFLD framework in local practice.

Introduction

Type 2 diabetes mellitus continues to place a heavy burden on health systems around the world, and its prevalence keeps rising alongside urbanization, sedentary living, and increasing rates of obesity[1]. Among the many conditions that travel alongside T2DM, fatty liver disease has drawn particular attention because of how often the two occur together and how much they seem to drive one another. Non-alcoholic fatty liver disease, long recognized as the hepatic manifestation of the metabolic syndrome, is now understood to affect a substantial share of the general population, with global prevalence estimates varying widely depending on the diagnostic method and population studied[2,3].

For many years, the diagnosis of NAFLD depended on ruling out other causes of liver disease, most importantly excessive alcohol intake. This exclusion-based definition, while useful, was seen by many hepatologists as an incomplete description of a condition that is, at its core, driven by metabolic dysfunction rather than the simple absence of alcohol use. In 2020, an international panel of experts proposed a new term, metabolic-associated fatty liver disease, built around positive diagnostic criteria rather than exclusion[4,5]. Under this framework, a patient can be diagnosed with MAFLD when hepatic steatosis is present together with overweight or obesity, T2DM, or at least two markers of metabolic dysregulation, regardless of how much alcohol they drink[6].

The relationship between fatty liver and diabetes does not run in a single direction. Insulin resistance appears to sit at the center of both conditions, and it is often difficult to tell whether it is the cause or the consequence of hepatic fat accumulation. Hepatic lipotoxicity, oxidative stress, and alterations in gut microbiota have all been proposed as mechanisms linking fatty liver disease with impaired glucose metabolism[7].

Because T2DM is such a strong predictor of hepatic steatosis, studies have consistently reported that a large share of diabetic patients already have some degree of fatty liver at the time of diagnosis. A cross-sectional analysis of patients with NAFLD found a high burden of prediabetes and undiagnosed diabetes even among those without a prior T2DM diagnosis, underscoring how tightly the two conditions are intertwined[8]. More recent work using pooled global data has estimated that the combined prevalence of T2DM among patients with either MAFLD or NAFLD sits close to a quarter of all cases, with incidence rates that vary depending on which diagnostic definition is applied[9]. The same body of evidence has shown that reanalyzing existing datasets under MAFLD

criteria instead of NAFLD criteria can raise fatty liver diagnosis rates among diabetic patients by nearly 69%, a figure that raises genuine questions about the balance between improved detection and possible over-diagnosis[9].

In Pakistan, where rates of obesity and T2DM are climbing quickly, a handful of studies have looked at NAFLD prevalence in diabetic populations, generally reporting figures well above global averages, with central obesity, elevated HbA1c, and dyslipidemia standing out as the strongest associated factors[10].

Beyond glycemic and hepatic outcomes, fatty liver disease in diabetic patients has also been linked to a wider set of complications. A broad review of NAFLD and cardiovascular disease highlighted the shared pathophysiology between hepatic steatosis and atherosclerotic risk, arguing for a more integrated approach to managing both conditions together rather than separately[11]. Some authors have gone further, suggesting that a metabolic dysfunction-associated steatotic liver disease label may offer an even better fit than MAFLD for certain patient groups, particularly those with a lean body habitus who would otherwise be missed by obesity-centered criteria[12]. Others have focused on the practical side of this debate, examining how MAFLD-specific metrics could refine risk stratification and guide future liver research[13], and the terminology itself continues to evolve, with ongoing discussion about whether steatotic liver disease should eventually replace both older labels altogether[14]. The pattern is also not limited to type 2 diabetes, since similar metabolic liver changes have been documented in patients with type 1 diabetes as well, which further supports the idea that metabolic dysfunction itself, rather than diabetes subtype alone, drives hepatic fat accumulation[27].

Diagnostic guidelines have had to keep pace with these conceptual shifts. Long-standing practice guidelines for NAFLD emphasized liver enzyme testing and imaging as the backbone of diagnosis[15], and ultrasonography in particular remains a widely used, low-cost, and reasonably reliable tool for detecting hepatic steatosis in both adults and children[16].

Given the rising burden of both T2DM and fatty liver disease locally, and the lack of Pakistani data comparing MAFLD and NAFLD within the same cohort, this study was designed to examine the prevalence of both conditions among patients attending a specialized diabetes center in Islamabad. Beyond simple prevalence, the study also set out to compare the biochemical, anthropometric, and comorbidity profiles between patients who met MAFLD criteria and those who met NAFLD criteria, with the aim of generating locally relevant evidence that could help guide screening and management decisions in everyday diabetic care.

Materials and Methods

Study design

This study utilized a descriptive cross-sectional design to determine the association of MAFLD and NAFLD with type 2 diabetes mellitus.

Study setting

The study was conducted at a tertiary care hospital in Islamabad, Pakistan, which provides specialized services for diabetic patients.

Study duration

The data was collected from December 2024 to May 2025.

Ethical approval

The protocol which was set for this study was reviewed by the ethical committee of the National Skill University, Islamabad before the commencement of data collection. All the patients were informed about the nature and purpose of the study and informed consent was obtained from each patient before including them in the study. The data of

patients remain confidential throughout the research according to Helsinki 1964 standards.

Sample size calculation

The study population consisted of adult patients, aged 20 years and above, diagnosed with T2DM and attending the center. Using an estimated NAFLD prevalence of 20% among T2DM patients, a 95% confidence interval, and a 5% margin of error, the required sample size was calculated as 246 using a standard WHO sample size formula[23]. Sixty-four complete patient records meeting the inclusion criteria were available for detailed biochemical and comorbidity analysis at the time of this report.

Parameter	Value
Level of significance	5%
Confidence interval	95%
Anticipated population proportion (P)	20%
Absolute precision (margin of error, d)	5%
Calculated sample size	246

Sampling technique

A consecutive sampling technique was applied. Patients attending the center who met the inclusion criteria were enrolled after obtaining informed consent, until the calculated sample size was completed.

Inclusion criteria

Patients were included if they had a confirmed diagnosis of T2DM, were aged 20 years or older, and had liver enzyme results consistent with possible NAFLD, with written informed consent on file.

Exclusion criteria

Patients were excluded if they had type 1 diabetes or other secondary forms of diabetes, a history of alcohol consumption exceeding the threshold used to diagnose NAFLD, chronic liver disease from causes other than fatty infiltration such as viral hepatitis, autoimmune hepatitis, Wilson's disease, or hemochromatosis, a history of liver transplantation or advanced cirrhosis, use of hepatotoxic medication, pregnancy or lactation, or severe comorbid or terminal illness that could confound the results.

Data collection

Patients coming with complications, after signing the informed consent, had blood drawn aseptically for laboratory analysis.

Statistical analysis

All the data was analyzed by IBM SPSS (Statistical Package for Social Sciences) version 23. Data were summarized as means with standard deviations for continuous variables and as frequencies with percentages for categorical variables. One-way analysis of variance was used to compare biochemical parameters between MAFLD-positive and MAFLD-negative groups, and separately between NAFLD-positive and NAFLD-negative groups. A p-value of less than 0.05 was considered statistically significant throughout the analysis.

Results

Baseline characteristics

The overall study had a mean age of 51.5 ± 11.8 years, with a near-equal gender distribution. Mean BMI was 30.8 ± 5.1 kg/m², mean HbA1c was 8.5 ± 1.6%, and mean triglycerides were markedly elevated at 223.7 ± 146.1 mg/dL, alongside a low mean HDL of 37.6 ± 8.1 mg/dL. Hypertension was present in 50.9% of patients, and 89.5% were on diabetic medication. Of the full study, 44 patients (68.8%) met criteria for either MAFLD or NAFLD (Table 1).

Table 1. Baseline characteristics of the entire patient cohort (N = 64)

Characteristic	Value
Total N	64
N (male / female)	26 / 28
Age (years)	51.5 ± 11.8
BMI (kg/m²)	30.8 ± 5.1
HbA1c (%)	8.5 ± 1.6
Total cholesterol (mg/dL)	194.8 ± 49.0
LDL (mg/dL)	99.8 ± 37.9
HDL (mg/dL)	37.6 ± 8.1
Triglycerides (mg/dL)	223.7 ± 146.1
ALT (U/L)	36.8 ± 19.4
AST (U/L)	26.5 ± 12.8
ALP (U/L)	230.9 ± 110.6
Total bilirubin (mg/dL)	0.5 ± 0.5
Hypertension, % (n)	50.9% (29)
On diabetic medication, % (n)	89.5% (51)
Coexisting liver disease, % (n)	10.5% (6)
NAFLD diagnosis, % (n)	67.2% (43)
MAFLD or NAFLD diagnosis, % (n)	68.8% (44)

Glycemic status

Of the 64 patients included in the analysis, 56 (88.9%) met criteria for T2DM based on HbA1c values, while 8 (9.5%) were classified as prediabetic. A one-way analysis of variance reached statistical significance (F = 12.5, p = 0.001) as shown in Table 02.

Table 2. One-way ANOVA for blood glucose by gender

Source of Variation	DF	Sum of Squares	Mean Square	F-statistic	P-value
Gender	1	15,200	15,200	12.5	0.001
Error	61	74,420	1,220	–	–
Total	62	89,620	–	–	–

Anthropometric findings

Most patients fell into the obese category, accounting for 78.1% (n = 50) of the study, followed by overweight at 15.6% (n = 10). Only 4.7% (n = 3) had a normal BMI, and 1.6% (n = 1) were underweight. The average BMI within the obese group was 33.9 kg/m², as shown in Table 03.

Table 3. Distribution of patients by BMI category

BMI Category	Number of Patients	Percentage	Average BMI (kg/m²)
Underweight	1	1.6%	18.0
Normal	3	4.7%	22.7
Overweight	10	15.6%	26.8
Obesity	50	78.1%	33.9
Total	64	100.0%	–

Comparison of MAFLD-positive and MAFLD-negative patients

When the 44 patients with confirmed fatty liver disease were further divided by diagnostic category, 25% were MAFLD-positive but NAFLD-negative, 22% were MAFLD-negative but NAFLD-positive, 22% met criteria for both conditions, and the remaining 31% were negative for both.

Patients who were MAFLD-positive had significantly higher ALT, AST, HbA1c, BMI, total cholesterol, LDL, and triglycerides compared with MAFLD-negative patients ($p < 0.05$ for all). ALP and HDL did not differ significantly between the two groups as shown in table 04.

Table 4. Comparison of biochemical parameters between MAFLD-positive and MAFLD-negative patients

Biomarker	MAFLD+ Mean (SD)	MAFLD- Mean (SD)	F-statistic	DF	P-value
ALT (U/L)	45.1 (18.2)	29.5 (13.5)	12.5	1,56	< 0.001
AST (U/L)	30.5 (12.1)	21.8 (9.8)	9.0	1,56	0.004
ALP (U/L)	255.3 (120.4)	210.1 (105.7)	2.8	1,56	0.099
HbA1c (%)	8.8 (1.2)	7.5 (1.0)	25.1	1,56	< 0.001
BMI (kg/m²)	34.5 (4.5)	28.5 (3.2)	35.8	1,56	< 0.001
Total cholesterol (mg/dL)	215.8 (45.1)	180.2 (30.5)	15.2	1,56	< 0.001
LDL (mg/dL)	115.6 (35.7)	95.2 (25.1)	10.1	1,56	0.002
HDL (mg/dL)	35.1 (8.2)	38.9 (7.5)	2.1	1,56	0.155
Triglycerides (mg/dL)	280.5 (120.3)	165.7 (60.1)	20.5	1,56	< 0.001

A similar pattern emerged for the NAFLD-positive group, which showed significantly higher ALT, AST, ALP, HbA1c, BMI, and triglycerides relative to NAFLD-negative patients. Total cholesterol, LDL, and HDL did not reach statistical significance in this comparison as shown in table 05.

Table 5. Comparison of biochemical parameters between NAFLD-positive and NAFLD-negative patients

Biomarker	NAFLD+ Mean (SD)	NAFLD- Mean (SD)	F-statistic	DF	P-value
ALT (U/L)	50.2 (15.8)	29.8 (14.0)	18.0	1,56	< 0.001
AST (U/L)	35.1 (10.5)	22.5 (10.0)	15.5	1,56	< 0.001
ALP (U/L)	280.5 (110.2)	200.1 (100.5)	8.2	1,56	0.006
HbA1c (%)	8.5 (1.1)	7.8 (1.2)	4.5	1,56	0.038
BMI (kg/m²)	33.8 (4.0)	29.5 (3.8)	10.2	1,56	0.002
Total cholesterol (mg/dL)	205.1 (38.2)	185.0 (35.0)	3.5	1,56	0.067
LDL (mg/dL)	108.9 (30.1)	98.5 (28.0)	2.1	1,56	0.153
HDL (mg/dL)	36.5 (7.8)	37.8 (8.0)	0.4	1,56	0.527
Triglycerides (mg/dL)	240.1 (100.5)	180.2 (70.1)	6.8	1,56	0.012

Comorbidity burden

Among the co-existing conditions recorded, gastric complaints were the most common at 76% (n = 49), followed by retinopathy at 68% (n = 44) and hypertension at 46% (n = 30). Foot complications were reported in 42% (n = 27) of patients, blood pressure-related issues in 45% (n = 29), hyperlipidemia in 31% (n = 20), chronic illness in 21% (n = 14), cardiovascular disease in 18% (n = 12), and chronic kidney disease in 20% (n = 13) as shown in table 06.

Table 6. Frequency of comorbid conditions among the study cohort

Comorbidity	Number of Patients (n)	Percentage (%)
Gastric issues	49	76%
Retinopathy	44	68%
Hypertension	30	46%
BP-related problems	29	45%
Foot complications	27	42%
Hyperlipidemia	20	31%
Chronic illness	14	21%
Cardiovascular disease	12	18%
Chronic kidney disease	13	20%

Discussion

Every biomarker examined in Tables 1 through 6 that reached statistical significance in this cohort mirrors a pattern already described in the regional and international literature on fatty liver disease in T2DM. What follows takes each significant variable in turn: first the clinical problem it represents, then what this cohort's data showed, and finally a published study that corroborates the direction of the finding.

Demographic and baseline profile: Fatty liver disease and T2DM are increasingly recognized as overlapping conditions worldwide, yet local data comparing the two current diagnostic frameworks, MAFLD and NAFLD, within the same diabetic cohort remain scarce in Pakistan, leaving clinicians without a clear sense of how much combined hepatic and metabolic screening burden to expect in routine diabetic care. In this cohort, the demographic and clinical profile was consistent with a population at high risk for fatty liver disease: a mean age of 51.5 ± 11.8 years, a near-equal gender split, hypertension in just over half of patients (50.9%), and 89.5% of patients already receiving diabetic medication. Nearly seven out of every ten patients (68.8%) met criteria for either MAFLD or NAFLD. A comparably designed cross-sectional study of 250 diabetic patients attending a tertiary gastroenterology department in Karachi reported an even higher NAFLD prevalence of 71.2%, together with significant associations between fatty liver disease and BMI, poor glycemic control, dyslipidemia, hypertension, and coronary artery disease[29], closely matching both the magnitude and the clinical pattern observed in this cohort.

Glycemic status: Poor glycemic control is one of the most consistently reported drivers of hepatic and metabolic complications in T2DM, and whether men and women differ meaningfully in how well their diabetes is controlled remains a live clinical question with direct implications for how screening and counselling are targeted. In this study, 88.9% of patients met HbA1c-based criteria for T2DM, and a one-way ANOVA of blood glucose by gender reached statistical significance ($F = 12.5$, $p = 0.001$), indicating a measurable gender-based difference in glycemic status within this cohort. This mirrors a comparative cross-sectional study of 258 T2DM patients in Ethiopia, which found a statistically significant gender-based difference in glycemic control, with women showing poorer HbA1c outcomes than men[30], supporting gender as a genuine, reproducible source of glycemic variability in T2DM populations rather than an artifact specific to this cohort.

Anthropometric findings: Obesity sits at the core of the shared pathophysiology linking T2DM and fatty liver disease, and body mass index has repeatedly emerged as one of the strongest predictors of NAFLD in diabetic cohorts, making the BMI distribution of any T2DM population directly relevant to how much fatty liver disease should be expected within it. The large majority of patients in this study, 78.1% ($n = 50$), fell into the obese category, with an average BMI of 33.9 kg/m² within that group, while only 4.7% had a normal BMI. A large Chinese cohort of T2DM patients similarly identified BMI, alongside waist-to-hip ratio and triglycerides, as one of the strongest independent predictors of NAFLD[20], reinforcing obesity as the dominant anthropometric driver of fatty liver disease risk in this cohort as well.

Significant biochemical and anthropometric differences between MAFLD/NAFLD-positive and -negative patients: Hepatic steatosis rarely occurs in isolation from the wider metabolic disturbance that characterizes T2DM. Each biomarker that reached statistical significance between fatty-liver-positive and fatty-liver-negative patients in this cohort is considered individually below, following the same problem–finding–reference structure used above.

ALT. Alanine aminotransferase is the most widely used surrogate marker of hepatic fat accumulation, and its clinical usefulness rests on evidence that elevated ALT does not merely track existing fatty liver disease but can independently predict incident T2DM in prospective cohorts. In this study, ALT was significantly higher in both the MAFLD-positive group (45.1 vs. 29.5 U/L, $p < 0.001$) and the NAFLD-positive group (50.2 vs. 29.8 U/L, $p < 0.001$) than in their respective negative counterparts. This is consistent with a prospective cohort of over 132,000 adults in which elevated ALT independently predicted incident T2DM[17], and with a South Asian cohort of T2DM patients in which

ultrasonography-confirmed NAFLD was associated with significantly higher ALT and strong discriminatory power on receiver-operating-characteristic analysis[18].

AST. Aspartate aminotransferase is a less liver-specific marker than ALT but remains a clinically useful indicator of hepatocellular injury in the setting of metabolic disease. AST was significantly elevated in both the MAFLD-positive group (30.5 vs. 21.8 U/L, $p = 0.004$) and the NAFLD-positive group (35.1 vs. 22.5 U/L, $p < 0.001$) in this cohort. A comparable pattern was reported in a case-control study from Northwest Ethiopia, where AST was significantly higher in T2DM patients than in matched non-diabetic controls[19], supporting AST as a consistent correlate of metabolic liver injury across diabetic populations.

ALP. Alkaline phosphatase is a less liver-specific marker, but it has nonetheless been linked to incident diabetes and fatty liver disease in large population-based cohorts. In this study, ALP was significantly higher in the NAFLD-positive group (280.5 vs. 200.1 U/L, $p = 0.006$) but did not reach significance in the MAFLD comparison (255.3 vs. 210.1 U/L, $p = 0.099$). This aligns with the same prospective cohort in which raised ALP, alongside ALT, tracked with incident T2DM risk[17], and suggests that ALP may be a more sensitive marker under the exclusion-based NAFLD framework than under the positive-criteria MAFLD framework in this population.

BMI. Obesity is one of the most consistently identified risk factors for fatty liver disease in diabetic populations, with BMI repeatedly emerging among the strongest predictors of NAFLD. In line with this, BMI was significantly higher in both the MAFLD-positive group (34.5 vs. 28.5 kg/m², $p < 0.001$) and the NAFLD-positive group (33.8 vs. 29.5 kg/m², $p = 0.002$) in this cohort. This mirrors both the large Chinese T2DM cohort in which BMI was among the strongest predictors of NAFLD[20] and the Karachi-based cross-sectional study, in which higher BMI was significantly associated with NAFLD in a diabetic population[29].

HbA1c. Glycemic control has been identified as a major, and potentially underappreciated, predictor of NAFLD severity, in some analyses contributing more to disease severity than BMI itself. This cohort showed the same pattern: HbA1c was significantly higher in the MAFLD-positive group (8.8% vs. 7.5%, $p < 0.001$) and the NAFLD-positive group (8.5% vs. 7.8%, $p = 0.038$). This is consistent with a cohort in which glycated haemoglobin was found to be a major predictor of disease severity in patients with NAFLD[21], reinforcing the idea that sustained hyperglycemia, rather than diabetes diagnosis alone, tracks with fatty liver disease activity.

Total cholesterol and LDL. Atherogenic dyslipidemia, characterized by elevated LDL cholesterol and triglyceride-rich lipoproteins, is a well-recognized feature of the metabolic disturbance underlying NAFLD. In this cohort, total cholesterol (215.8 vs. 180.2 mg/dL, $p < 0.001$) and LDL (115.6 vs. 95.2 mg/dL, $p = 0.002$) were both significantly higher in the MAFLD-positive group, though neither reached significance in the NAFLD comparison. This pattern is consistent with reviews describing dyslipidemia management as a central component of NAFLD care[22], and with atherogenic-index findings from a large T2D cohort in which lipid-related indices significantly distinguished MAFLD-positive from MAFLD-negative patients[31], again suggesting that the two diagnostic frameworks may be capturing somewhat different aspects of the underlying metabolic disturbance.

Triglycerides. Hypertriglyceridemia is consistently reported as one of the strongest biochemical correlates of fatty liver disease in diabetic patients, and this cohort was no exception: triglycerides were markedly higher in both the MAFLD-positive group (280.5 vs. 165.7 mg/dL, $p < 0.001$) and the NAFLD-positive group (240.1 vs. 180.2 mg/dL, $p = 0.012$). This is consistent with the large Chinese T2DM cohort[20], dyslipidemia-focused reviews of NAFLD management[22], and the Karachi cohort in which dyslipidemia was significantly associated with NAFLD[29].

MAFLD-only versus NAFLD-only classification. Because MAFLD and NAFLD rest on different diagnostic logic, positive metabolic criteria versus exclusion of alcohol and

other liver disease, patients who are biochemically similar can be classified differently depending on which framework is applied, a distinction with real consequences for who gets flagged for further work-up. In this cohort, roughly a quarter of patients were MAFLD-positive but NAFLD-negative, meaning these patients would have been missed entirely under the older diagnostic framework, most likely because of reported alcohol use or another liver condition that excluded a NAFLD diagnosis. A large case–control study of over 2,500 patients with T2D similarly found that MAFLD-positive patients formed a biochemically and demographically distinct subgroup, with significant differences in ALT and gender distribution compared with MAFLD-negative patients[31], reinforcing that MAFLD-specific criteria capture patients who would not necessarily be flagged under NAFLD alone. This distinction is not unique to type 2 diabetes: similar metabolic-associated steatotic liver disease patterns have been documented in type 1 diabetes as well, underscoring that it is the underlying metabolic dysfunction itself, rather than diabetes subtype, that MAFLD criteria are designed to capture[27].

Comorbidity burden: Fatty liver disease in diabetic patients has been linked to a wide range of extrahepatic complications, and unmanaged disease can progress toward decompensated cirrhosis or hepatocellular carcinoma, conditions in which liver transplantation becomes the only definitive treatment option. In this cohort, gastric complaints (76%) and retinopathy (68%) were the most common comorbidities, alongside a substantial burden of hypertension (46%) and chronic kidney disease (20%). These patterns are broadly consistent with narrative reviews describing nephrological, pulmonary, and dermatological complications in the context of MAFLD/NAFLD[24], and the high rate of hypertension observed here fits with the Karachi-based cohort, in which hypertension and coronary artery disease were both significantly more common among diabetic patients with NAFLD[29]. Left unaddressed, this trajectory can progress toward decompensated disease in which liver transplantation becomes the only definitive option, as described in a multicenter Brazilian series of transplant recipients[28]. Lifestyle-focused intervention remains central to managing both T2DM and fatty liver disease regardless of which diagnostic label is applied: cohort evidence from other diabetic populations has shown that sustained healthy lifestyle behaviors are associated with a lower risk of microvascular complications over time[25], and longitudinal data on diabetes remission has shown that meaningful improvements in glycemic control are achievable in a subset of patients, offering some reassurance that the metabolic derangements documented in this cohort are not necessarily permanent[26].

Conclusion

Both MAFLD and NAFLD are highly prevalent among patients with T2DM attending a diabetes center in Rawalpindi and Islamabad, with more than two-thirds of the cohort meeting criteria for one or both conditions. Fatty liver-positive patients showed significantly worse liver enzyme profiles, higher BMI, poorer glycemic control, and more pronounced dyslipidemia than fatty liver-negative patients, reinforcing the close metabolic relationship between hepatic steatosis and diabetes. Given the high comorbidity burden observed, routine hepatic and metabolic screening should be considered a standard part of diabetic care in similar clinical settings.

Limitations and Future Directions

This study is limited by its cross-sectional design, single-center setting, and a sample size smaller than the originally calculated target, which restricts the ability to draw causal conclusions or generalize findings broadly across the wider Pakistani T2DM population. Future work should aim to follow patients longitudinally to better understand how MAFLD and NAFLD diagnoses relate to long-term outcomes such as cardiovascular events, fibrosis progression, and mortality, ideally across multiple centers and with larger sample sizes that more closely match the statistically calculated requirement.

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