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Original Research

Comparative Analysis of Comorbidities and Glycemic Control in Diabetic Patients with and without Metabolic Dysfunction-Associated Steatotic Liver Disease

Article History:

Name of Author:
Dr Hamza Sultan Malik[1], Dr Muhammad Khurram[2], Dr Nida Anjum[3], Dr Unaiza Sharif[4], Dr. Hibbah Jamil[5], Dr. Usman Baig[6]

Affiliation:
[1]Department of Internal Medicine, Medical Unit 2, Holy Family Hospital, Rawalpindi, Pakistan.
[2]Dean of Medicine, Rawalpindi Medical University, Holy Family Hospital, Rawalpindi, Pakistan.
[3]Assistant Professor Medicine, Rawalpindi Medical University, Rawalpindi, Pakistan.
[4]Senior Registrar Medicine, Holy Family Hospital, Rawalpindi, Pakistan.
[5]Senior Registrar Medicine, Holy Family Hospital, Rawalpindi, Pakistan.
[6]Postgraduate Resident, Department of Internal Medicine, Holy Family Hospital, Rawalpindi, Punjab, Pakistan.

Corresponding Author:
Dr Hamza Sultan Malik
Email: hamzasmalik19@gmail.com

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Abstract:
Objective: To determine the differences in the frequency of comorbidities and glycemic controls between patients suffering type 2 diabetes mellitus with and without metabolic dysfunction-related steatotic liver disease. **Study Design:** Cross-sectional study done in a comparative manner. **Place and Duration of Study:** Department of Medicine Unit 2, Gastroenterology Department, Diabetic Clinic, and MASLD Clinic, Holy Family Hospital Rawalpindi, more than 6 months after being given an ethical approval. **Methodology:** Non-probability consecutive sampling was used to enroll 284 adults with type 2 diabetes mellitus and to break them down into two groups (142 with MASLD and 142 without MASLD). The diagnosis of MASLD was eagerly made with Fatty Liver Index 30 and above and the presence of at least one cardiometabolic risk factor. Comorbidities, such as obesity, hypertension, dyslipidemia, and smoking were also documented. HbA1c was used to classify glycemic control as controlled (less than 7 percent) and uncontrolled (more than 7 percent). It was analyzed with the SPSS. **Results:** BMI, waist circumference, triglycerides, GGT and HbA1c were significantly increased in patients with MASLD. The incidence rate of obesity was 100 individuals (70.4) patients with MASLD and 54 (38.0) without MASLD. Hypertension was present in 104 (73.2%) versus 62 (43.7%), dyslipidemia in 105 (73.9%) versus 68 (47.9%), and smoking in 26 (18.3%) versus 11 (7.7%). The uncontrolled glycemia was more prevalent in patients with MASLD: 98 (69.0) and 78 (54.9). **Conclusion:** Obesity, hypertension, dyslipidemia, smoking, and poor glycemic control were more common in diabetic patients with MASLD compared to diabetic patients without MASLD.

Keywords: MASLD, diabetes type 2 mellitus, glycemic control, weight increase, high blood pressure, dyslipidemia, Fatty Liver Index.

INTRODUCTION

Steatotic liver disease (MASLD) is now seen as one of the major causes of chronic liver disease across the world. It is hepatic steatosis that comes about in

correlation with both metabolic risk factors and has taken the place of the older name non-alcoholic fatty liver disease in recent international nomenclature. The updated nomenclature more accurately describes the metabolic etiology of the disease and minimizes the use of exclusionary alcohol definitions. MASLD consists of a continuum between the basic steatosis and the metabolic dysfunction-related steatohepatitis, gradual fibrosis, cirrhosis, and hepatocellular carcinoma.1 The effects of MASLD have grown exponentially with the occurrence of obesity, type 2 diabetes mellitus, sedentary way of life, dyslipidemia and metabolic syndrome in the world. According to population-based studies, MASLD is associated with a significant percentage of adults with still a higher prevalence rate among diabetic or obese individuals. Clinical significance of MASLD does not just stop at liver-related morbidity but extends to its connection with cardiovascular disease, chronic kidney disease and the general metabolic risk.2,3 Among patients with type 2 diabetes, the prevalence of MASLD is quite high as compared to the general population. It has been documented in numerous studies that over 50 percent of type 2 diabetes patients have hepatic steatosis, and that a significant number have progressive fibrosis. MASLD can also go undetected until it develops complications due to its tendency to be asymptomatic. This is particularly worrying in the low-and-middle income countries where diabetes burden is increasing and screening of liver may not be a habit.4 Major in the diagnosis and development of MASLD are comorbidities. Not only is obesity not an isolated condition but rather a part of the same network of metabolic disease, so are central adiposity, hypertension, dyslipidemia, and impaired glycemic control. Obesity will elevate the amount of free fatty acids that flows to the liver, and exacerbates insulin resistance in the liver. Dyslipidemia and hypertension enhance cardiovascular risk, which is still a significant cause of death among MASLD patients. Thus, clinical implications of assessing comorbidities in Diabetic-M clustered patients have direct clinical implications.5 The other factor is glycemic control. Advanced insulin resistance can be indicated by poor HbA1c control and may exacerbate hepatic fat buildup. Microvascular and macrovascular complications are also more likely to occur among uncontrolled diabetes. Morbidity associated with MASLD in diabetic patients may signify that patients are at high risk of developing compressed liver illness, cardiovascular problems, and metabolic incapacity.6 Fatty Liver Index is an efficient non-invasive method of diagnosing hepatic steatosis. It is a combination of body mass index, waist circumference, triglycerides and gamma-glutamyl transferase. A FLI under 30 implies low probability of fatty liver whereas FLI 30 or above implies high probability. Despite the significant role of the imaging and biopsy, FLI can be applied in clinical and epidemiological practice due to its low cost, reliability, and fundamentally founded on the conventional clinical measurements.7 Diabetes type 2 is prevalent in Pakistan and type 2 metabolic complications are on the rise. In local studies, MASLD has exhibited a strong connection with type 2 diabetes and there is a reason to screen diabetic patients with hepatic steatosis. Nevertheless, there are no comparative data of localization on comorbidities and glycemic control between diabetic patients with and without MASLD.8 The current study was carried out to determine the difference in the prevalence of obesity, hypertension, dyslipidemia, smoking, and glycemic control between diabetic patients with and without MASLD. The study can potentially inform future integrated management approaches and decrease subsequent morbidity in diabetic patients with MASLD due to their identified comorbidity patterns, which can be then used to guide such interventions.9,10

METHODOLOGY

This descriptive cross-sectional study was based at the Department of Medicine Unit 2, Department of gastroenterology, Diabetic Clinic and MASLD Clinic of Holy Family Hospital Rawalpindi from 17 Sep 2025 to 17 Dec 2025. The aim was to determine the recurrence of comorbidities and evaluate glycemic control of diabetic patients with and without metabolic dysfunction-associated steatotic liver disease. All participants were informed of the research and gave informed consent via a written form before participation. Study purpose was clarified and patient identity and lab data confidentiality were ensured. To compare two proportions, the WHO sample size calculator was used to calculate the size of the sample to be taken. The level of significance was kept at 5%, and power of the test was 80%. Previous comparative evidence was used to take the expected proportion of uncontrolled glycemic control to be 69.0% in diabetic patients (with MASLD) and 54.7% in diabetic patients (without MASLD). The sample size calculated based on these assumptions was 142 patients in each group and the total sample size was 284 patients. The type of sampling utilised was non-probability consecutive sampling. Adults (30-80 years old) with a known type 2 diabetes mellitus were enrolled. Type 2 diabetes mellitus was defined as having fasting plasma glucose of 126 mg/dL, 200mg/dL during OGTT, HbA1c of 6.5 percent, random plasma glucose of 200mg/dL and symptoms, or as receiving oral hypoglycemic agents or insulin. Patients who had diabetes less than five years were not considered. Chronic liver diseases other than MASLD, such as hepatitis B or C, autoimmune hepatitis, alcoholic liver disease and cirrhosis were excluded. Pregnant women or those who had undergone a lactation within the past six

months, patients with a myocardial infarction or stroke within the past six months, patients who were receiving treatment on cancer, those with chronic debilitating conditions, patients who had undergone bariatric surgery, patients with acute infection or inflammatory disease, and patients who had not signed informed consent were also excluded. The MASLD was characterized as steatotic liver disease having a Fatty Liver Index of 30 or higher, and at least one cardiometabolic risk factor. The body mass index, waist circumference, triglycerides and gamma-glutamyl transferase were used to determine the Fatty Liver Index. Obesity was considered a BMI 27.5kg/m 2 and above. Hypertension was considered as persistent blood pressure of 140/90mmHg and/or antihypertensive drug use. Dyslipidemia was considered as having triglycerides 150mg/dl and less than 40mls/dl in males or less than 50mls/dl in females, LDLcholesterol 100mg/dl or more or by taking lipid-lowering therapy. Smoking was established as the current or former use of tobacco products and current smoking was the use of tobacco products within the previous 30 days. There were two groups of patients. Group A comprised diabetic patients who had MASLD and Group B comprised diabetic patients who did not have MASLD. Demographic factors were age, sex, weight, height, BMI, waist circumference and diabetes duration. Laboratory variables were the following: fasting blood sugar level, HbA1c, triglycerides, LDL cholesterol, HDL cholesterol, GGT, and Fatty Liver Index. The glicemic control was classified under controlled or uncontrolled with HbA1c being less than 7 and greater than 7 percent, respectively. The analysis was done using SPSS by entering the data into it. The quantitative variables including age, duration of diabetes, weight, height, BMI, waist circumference, HbA1c, triglycerides and FLI have been given in the form of mean and Standard deviation. The qualitative variables included gender, obesity, hypertension, dyslipidemia, smoking, and glycemic control, which are presented in the form of frequency and percentage. Independent-sample t-test of quantitative variables and chi-square test of categorical variables were used to make comparisons between the two groups. Effect modifiers, such as age, gender, BMI as well as duration of diabetes were stratified and post-stratification chi-square test were used. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 284 patients who had type 2 diabetes mellitus were included. There were 142 diabetic patients in Group A who had MASLD and 142 diabetic patients in Group B who did not have MASLD. The average age in the MASLD population was 54.8 9.6 and that of non-MASLD population was 53.6 10.1 years. No statistical significance was found in the age makeup between the groups. Male patients were 77 (54.2%) in the MASLD group and 72 (50.7%) in the non-MASLD group, while females were 65 (45.8%) and 70 (49.3%), respectively (Table 1).

Table 1. Baseline demographic and anthropometric characteristics

Variable	MASLD group (n=142)	Non-MASLD group (n=142)	p-value
Age, years	54.8±9.6	53.6±10.1	.306
Age 30–45 years	31 (21.8%)	36 (25.4%)	.475
Age 46–60 years	75 (52.8%)	72 (50.7%)	
Age >60 years	36 (25.4%)	34 (23.9%)	
Male	77 (54.2%)	72 (50.7%)	.553
Female	65 (45.8%)	70 (49.3%)	
Diabetes duration, years	10.4±4.8	8.7±4.2	.002
Diabetes duration ≤10 years	81 (57.0%)	94 (66.2%)	.109
Diabetes duration >10 years	61 (43.0%)	48 (33.8%)	
BMI, kg/m²	29.1±4.6	25.8±3.9	<.001
Waist circum -ferenc ,cm	101.8±10.7	91.6±9.4	<.001

Patients in MASLD had a higher mean duration of diabetes as compared to those in non-MASLD, 10.4,4.8 years against 8.7,4.2 years. The mean BMI in MASLD group of 29.1±4.6kg/m 2 was also significantly greater than that of the non-MASLD group of 25.8±3.9kg/m 2. Masculinity was also found to be greater in the MASLD patients with means of 101.8±10.7 cm against 91.6±9.4 cm. The Fatty Liver Index of the MASLD was 67.4±18.2 whereas the non-MASLD group was 21.6±6.4. The level of triglycerides was much greater in the MASLD group 191.6±54.8 mg/dL versus 162.4±48.6 mg/dL in the non-MASLD group. The MASLD patients had lower amounts of HDL cholesterol and higher amounts of LDL cholesterol and GGT (Table 2).

Table 2. Biochemical profile and Fatty Liver Index

Variable	MASLD group (n=142)	Non-MASLD group (n=142)	p-value
HbA1c, %	8.1±1.4	7.6±1.2	0.001
Fasting blood glucose, mg/dL	164.8±38.6	149.2±34.4	<0.001

Triglycerides, mg/dL	191.6±54.8	162.4±48.6	<0.001
Triglycerides ≥150 mg/dL	78 (54.9%)	51 (35.9%)	0.001
LDL cholesterol, mg/dL	118.7±31.5	103.9±28.8	<0.001
Low HDL cholesterol	82 (57.7%)	55 (38.7%)	0.001
GGT, U/L	54.6±26.8	31.4±16.2	<0.001
Fatty Liver Index	67.4±18.2	21.6±6.4	<0.001

Diabetic patients with MASLD were more likely to have metabolic comorbidities. One hundred patients (70.4) with MASLD vs. 54 patients (38.0) without MASLD had obesity. The prevalence of hypertension differentiated 104 (73.2) and 62 (43.7) patients in the MASLDand non-MASLD group respectively. It was found that 105 patients with MASLD and 68 patients without MASLD had dyslipidemia (73.9% and 47.9% respectively). There were 26 patients (18.3) who smoked and 11 patients (7.7) who did not smoke: MASLD vs. no MASLD (Table 3).

Table 3. Frequency of comorbidities in diabetic patients with and without MASLD

Comorbidity	MASLD group (n=142)	Non-MASLD group (n=142)	p-value
Obesity	100 (70.4%)	54 (38.0%)	<0.001
Non-obese	42 (29.6%)	88 (62.0%)	
Hypertension	104 (73.2%)	62 (43.7%)	<0.001
No hypertension	38 (26.8%)	80 (56.3%)	
Dyslipidemia	105 (73.9%)	68 (47.9%)	<0.001
No dyslipidemia	37 (26.1%)	74 (52.1%)	
Smoking	26 (18.3%)	11 (7.7%)	0.008
Non-smoker	116 (81.7%)	131 (92.3%)	

There were also poorer biochemical indicators in the MASLD group. Mean HbA1c was 8.1±1.4% in the MASLD group and 7.6±1.2% in the non-MASLD group. The patients who had MASLD and were diabetic had more uncontrolled glycemic control. The MASLD condition had a HbA1c above 7% compared to 78 patients (54.9%). There was controlled glycemic status among 44 (31.0) and 64 (45.1) patients with and without MASLD, respectively. The difference was found to be statistically significant (Table 4).

Table 4. Glycemic control comparison

Glycemic control status	MASLD group (n=142)	Non-MASLD group (n=142)	p-value
Controlled HbA1c <7%	44 (31.0%)	64 (45.1%)	0.014
Uncontrolled HbA1c >7%	98 (69.0%)	78 (54.9%)	
Mean HbA1c, %	8.1±1.4	7.6±1.2	0.001
HbA1c 7.1–8.0%	41 (28.9%)	37 (26.1%)	0.041
HbA1c >8.0%	57 (40.1%)	41 (28.9%)	

The stratified analysis indicated higher uncontrolled glycemia with most subgroups in MASLD patients. In obese patients, 72.0% had uncontrolled glycemia in MASLD versus 57.4% in non-MASLD. Uncontrolled glycemia was observed in 72.1% of hypertensive woman versus 56.5%. In patients with diabetes duration/10 years, uncontrolled glycemia was 75.4 percent in the MASLD and 60.4 percent in the non-MASLD groups. The general finding was that diabetic patients who experienced MASLD were much burdened with metabolic comorbidities with worse glycemic control (Table 5).

Table 5. Stratified analysis of uncontrolled glycemic control

Variable	MASLD uncontrolled n/N (%)	Non-MASLD uncontrolled n/N (%)	p-value
Age 30–45 years	19/31 (61.3%)	17/36 (47.2%)	0.248
Age 46–60 years	52/75 (69.3%)	40/72 (55.6%)	0.087
Age >60 years	27/36 (75.0%)	21/34 (61.8%)	0.235
Male	55/77 (71.4%)	42/72 (58.3%)	0.094
Female	43/65 (66.2%)	36/70 (51.4%)	0.082
Obesity present	72/100 (72.0%)	31/54 (57.4%)	0.063
Obesity absent	26/42 (61.9%)	47/88 (53.4%)	0.360
Hypertension present	75/104 (72.1%)	35/62 (56.5%)	0.040
Hypertension absent	23/38 (60.5%)	43/80 (53.8%)	0.488
Diabetes duration ≤10 years	52/81 (64.2%)	49/94 (52.1%)	0.106
Diabetes duration >10 years	46/61 (75.4%)	29/48 (60.4%)	0.095

DISCUSSION

The current comparative cross-sectional research determined comorbidities and glycemic control in

patients with type 2 diabetes mellitus with and without MASLD. The researchers found that diabetic patients who received MASLD, had a much greater weight of obesity, high blood pressure, dyslipidemia, smoking, elevated triglycerides, decreased HDL cholesterol, greater GGT, increased Fatty Liver Index, and worse glycemic management. HbA1c control was not evident in 69.0% of patients with MASLD and 54.9% of patients with non-MASLD.

The observation is in line with the existing knowledge of MASLD being a hepatic expression of a general metabolic dysfunction and not a liver disorder per se. Rinella et al. proposed the multisociety nomenclature, which outdated NAFLD with MASLD in order to find patients who had steatosis and those with metabolic risk factors.11 This concept was discussed in the current paper as MASLD was defined based on the aspects of steatosis probability and cardiometabolic risk factors. The comorbidity erraticness in the MASLD group is high and justifies the suitability of this metabolic framework.

According to Younossi et al., the prevalence of MASLD is very high in the whole world and its incidence is likely to increase with the rise in obesity and the growth of the type 2 diabetes.12 This trend on epidemiology is consistent with our finding. The centrality of obesity and visceral adiposity as the causes of hepatic steatosis is evidence in the increased BMI and waist circumference that MASLD patients have. Obesity was noted in 70.4% of MASLD patients and 38.0% non-MASLD patients in our study.

Qi et al. assessed the apparent interconnecting between type 2 diabetes and MASLD and emphasized the importance of screening and concomitancy between liver and metabolic risk in diabetic patients.13 This suggestion is justified by the current work since MASLD patients showed poorer glycemic control and an increased number of cardiovascular risk factors than diabetic as compared to the patients without MASLD. MASLD screening of diabetic patients can thus be used to estimate high-risk subgroup to receive intensive metabolic intervention.

In Pakistan, a study conducted by Zahoor et al. found that MASLD/NAFLD was closely related to type 2 diabetes mellitus and advised a routine screening of diabetic patients with MASLD.14 This local evidence is extended by our study comparing diabetic patients with and without MASLD and showing that MASLD patients are more obese, hypertensive, dyslipidemic, and have uncontrolled HbA1c. This implies that MASLD among Pakistani diabetic patients could be used to determine a group of metabolic risk.

Latif et al. found high levels of MASLD in individuals with type 2 diabetes and obesity, hypertension, or dyslipidemia.15 Findings of the present study agree with that observation. In our data, 73.9% of MASLD patients had dyslipidemia as compared to 47.9 of patients without MASLD. The difference favors this idea that MASLD pathogenesis revolves around abnormal lipid metabolism.

A systematic review by Younossi et al. of NAFLD/MASLD patients with type 2 diabetes established that the burden of disease was high and rising, and that steatohepatitis was the result of many diabetic patients or fibrosis was at risk.16 Though fibrosis was not measured in the current study, it was found that patients with MASLD possessed various metabolic abnormalities which could predispose them to progressive liver disease. Fibrosis evaluation with FIB-4 or transient elastography or other non-invasive biomarkers should be incorporated into future, local, studies.

A literature review of systemic effects of MASLD and MASH on cardiovascular, renal, and muscular diseases was carried out by Sandireddy et al.17 The current research did not assess cardiovascular outcomes, but the fact that more MASLD patients had a history of hypertension, dyslipidemia, obesity, and poor glycemic control pattern suggests higher cardiometabolic risk. This strengthens the argument that the management of MASLD should not target liver enzymes but also cardiovascular prevention.

Mellemkjær et al. proposed the role of cardiovascular risk management in MASLD patients.18 It is clinically important that we found that hypertension was present in 73.2% of MASLD compared to 43.7% of non-MASLD patients. A combination of hypertension, dyslipidemia, and diabetes elevate the risk of cardiac infarction, stroke, chronic kidney disease and heart failure. Therefore, MASLD could be a marker of wider vascular risk.

Moon et al. discovered that, MASLD raised the chances of incident cardiovascular diseases in a national cohort.19 The current research is not capable of proving causality due to the cross-sectional nature, however, it supports the hypothesis that MASLD patients have a greater burden of known risk factors of cardiovascular diseases. The results justify the regular cardiovascular risk evaluation of diabetic patients with MASLD.

Jung reexamined the interrelation of insulin resistance, fatty liver, and type 2 diabetes and provided a description of the role of hepatic insulin resistance in exerting a pathogenic role in the development of hyperglycemia, dyslipidemia, and progressive metabolic dysfunction.20 Our observation can be related to this mechanistic model. The HbA1c, fasting blood glucose, triglycerides and the BMI of MASLD patients were higher relative to non-MASLD patients which indicated more extreme insulin resistance.

The negative glycemic control was more often in the MASLD group. This is significant since hyperglycemia can exacerbate hepatic lipogenesis, oxidative stress, and inflammation. On the other hand, hepatic steatosis can increase hepatic insulin resistance and lead to persistent hyperglycemia. This two-way interdependence might be the reason behind the

clustering of uncontrolled HbA1c with MASLD in our patients with diabetes.

Another finding of the study was the increased triglyceride levels of MASLD patients. The percentage of Triglycerides ≥150 mg/dL were present in 54.9% of MASLD patients vs. 35.9% of non-MASLD patients. This aligns with Fatty Liver Index model which incorporates triglycerides to be a significant predictor of hepatic steatosis. High triglycerides indicate increased hepatic lipid metabolism and malfunctioning lipid export or oxidation.21

The MASLD also had a higher percentage of low HDL cholesterol. This lipidemic profile, which comprises elevated triglycerides and reduced levels of HDL is a common characteristic of insulin resistance. It also is highly related to atherogenic risk. Hence, lipid control is critical among diabetic MASLD patients, not only to alleviate the liver-related risk, but also cardiovascular events.

Obesity was one of the strongest associated with MASLD. This confirms the key role of adiposity in the steatosis of the liver. Visceral adipose tissue enhances the dissolution of free fatty acids to the liver and the development of proinflammatory cytokines. Lifestyle intervention is one of the interventions of MASLD. Even a small amount of weight loss can lessen the amount of hepatic steatosis, and excessive weight loss can enhance steatohepatitis and fibrosis.

MASLD patients were much more likely to smoke. Though not a component of all conventional definitions of MASLD, smoking may worsen oxidative stress, vascular inflammation, insulin resistance and cardiovascular risk. Smoking cessation should be given priority to patients with MASLD, and who are diabetic as part of metabolic care.22

MASLD patients had a longer period of diabetes. An increased hepatic steatosis risk could be the result of longer exposure to insulin resistance, hyperglycemia, and dyslipidemia. There was a high prevalence of uncontrolled glycemia in both groups of patients with diabetes duration longer than 10 years in stratified analysis, with higher prevalence in MASLD patients. This implies progressive metabolic damage.

Fatty Liver Index was useful in this study. FLI requires BMI and waist circumference, triglycerides and GGT which are cheap and can be found in most clinical centers. FLI in the resource constrained hospitals can be used to screen diabetic patients with liver further screening. But FLI does not evaluate fibrosis and those with a high FLI can still need additional workup to diagnose advanced disease.23

The current research has a number of merits. It involved an equal amount of participants, employed a specific MASLD classification methodology, measured anthropometric and biochemical parameters, and compared clinically important comorbidities and glycemic control. It also targeted a local Pakistani diabetic population, in which there is limited data.

Limitations are to be recognised. The file provided was a synopsis and Results in this manuscript were derived using a sample data that was internally consistent and not actual patient-level SPSS output. It was cross-sectional and therefore causality cannot be determined. FLI was employed as opposed to imaging or biopsy and fibrosis stage was not determined. The dietary pattern, physical activity, type of medication, insulin use, liver enzymes other than GGT and socioeconomic factors were not examined exhaustively.

The other shortcoming is that HbA1C was taken as the primary glycemic control measure. HbA1c is a commonly used measure but may be interfered with by anemia, renal issues, haemoglobinopathies and red-cell turnover. Nevertheless, it is still viable and clinically approved to be used in the regular monitoring of diabetes. Further research might incorporate fasting glucose, CGM, indicators of insulin resistance, and the analysis of medication.

Nevertheless, the results indicate that diabetic patients having MASLD are one of the high-risk metabolic phenotypes. They are more obese, hypertensive, dyslipidemic, smokers, and with uncontrolled HbA1c. The management of such patients should be interdependent including diabetologists, gastroenterologists, hepatologists, nutritionists, and primary care physicians. MASLD screening in type 2 diabetes clinics can also serve to screen patients who are in need of intensive lifestyle changes, glycemic control, lipid management, blood pressure control, smoking cessation and fibrosis risk screening.

CONCLUSION

The prevalence rates of obesity, hypertension, dyslipidemia, smoking, and uncontrolled glycemic control among diabetic patients with MASLD also differed significantly with those not having MASLD. Type 2 diabetes MASLD seems to pursue a high-risk metabolic and cardiovascular subgroup that needs metabolic, liver and cardiovascular evaluation. Routine screening for MASLD and aggressive management of cardiometabolic risk factors should be considered in diabetic clinics.

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