ORIGINAL ARTICLE

CONTENTS

Six-month effects of liraglutide and dapagliflozin on lipid profile, cardiovascular risk, and NT-proBNP levels in patients with metabolic dysfunction-associated steatotic liver disease

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ABSTRACT

Aim: This study assessed and compared changes in lipid profile and cardiovascular risk in patients with metabolic dysfunction-associated steatotic liver disease after six months of liraglutide or dapagliflozin treatment. We also evaluated changes in N-terminal pro-B-type natriuretic peptide levels.

Materials and Methods: This prospective, randomized, parallel-group study included 115 adult patients with metabolic dysfunction-associated steatotic liver disease. Participants were randomized into three groups: control (n = 36, lifestyle intervention only), dapagliflozin (n = 41, 10 milligrams daily), or liraglutide (n = 38, titrated to 1.8 milligrams daily). All patients adhered to a Mediterranean diet and moderate physical activity. Lipid profile and N-terminal pro-B-type natriuretic peptide levels were measured at baseline and six months. Cardiovascular risk was assessed using five validated scales: Globorisk, Framingham Risk Score, American College of Cardiology/American Heart Association atherosclerotic cardiovascular disease Risk Calculator, Prospective Cardiovascular Münster Score, and World Health Organization cardiovascular risk charts.

Results: All groups showed significant within-group improvements in total cholesterol, low-density lipoprotein cholesterol, triglycerides, and high-density lipoprotein cholesterol ($p < 0.001$), with liraglutide showing greater lipid improvements intergroup ($p < 0.05$). Cardiovascular risk scores decreased significantly in all groups, with no differences between them. N-terminal pro-B-type natriuretic peptide levels increased significantly in the control and liraglutide groups, but remained unchanged in the dapagliflozin group.

Conclusions: Liraglutide and dapagliflozin are effective in improving lipid profile and reducing cardiovascular risk. Liraglutide showed superior efficacy in lipid improvement. Changes in N-terminal pro-B-type natriuretic peptide require further investigation.

KEY WORDS: metabolic dysfunction-associated steatotic liver disease, cardiovascular risk, liraglutide, dapagliflozin, NT-proBNP

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INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is one of the most common forms of chronic liver disease, affecting over 30% of the global adult population and continuing to rise [1]. In most cases, MASLD develops in the context of obesity, insulin resistance, and type 2 diabetes mellitus, all of which are closely associated with lipid metabolism disturbances [2].

Dyslipidemia in MASLD is characterized by elevated triglyceride (TG) levels, decreased high-density lipoprotein (HDL-C), increased low-density lipoprotein (LDL-C) concentrations [3]. These changes contribute to a higher cardiovascular risk and simultaneously aggravate the progression of hepatic steatosis [4].

Current evidence indicates that cardiovascular diseases (CVDs), rather than hepatic complications, are the leading cause of mortality in patients with MASLD. This elevated risk is not only driven by associated metabolic conditions such as diabetes, hypertension, and dyslipidemia, but also by direct mechanisms linked to chronic systemic inflammation, oxidative stress, and endothelial dysfunction characteristic of MASLD [5].

Given this, the assessment of cardiovascular risk in MASLD patients becomes clinically significant. Several validated tools are available to estimate the 10-year risk of cardiovascular events, including the ASCVD Risk Calculator (ACC/AHA), Framingham Risk Score, Prospective Cardiovascular Münster (PROCAM) Score, WHO cardiovascular risk charts, and Globorisk [6-10].

Utilizing multiple models enables a more accurate stratification of cardiovascular risk. However, these tools do not consider hepatic fibrosis or steatosis, which may influence outcomes [11].

N-terminal pro-B-type natriuretic peptide (NT-proBNP) is a stable biomarker primarily used to assess cardiac wall stress and is most commonly applied in the context of heart failure. However, it is increasingly being explored in broader cardiovascular risk assessment, especially

in populations with metabolic dysfunction. This is particularly relevant in patients with metabolic disorders such as obesity, insulin resistance, and MASLD [12].

In the context of hepatic steatosis, particularly in early stages (S1–S2), a trend toward lower NT-proBNP levels has been observed. This may be related to hyperinsulinemia-mediated suppression of natriuretic peptide secretion. Such reductions may mask early signs of cardiac stress and lead to an underestimation of cardiovascular risk [13].

This reduction may complicate the detection of early stages of cardiac involvement, as traditional cardiovascular risk stratification tools do not consider hepatic steatosis as a modulating factor of NT-proBNP levels. In the context of treatment, it becomes especially important to monitor NT-proBNP changes in relation to steatosis severity, as a potential indicator of therapeutic response [14].

While lifestyle modification remains the cornerstone of MASLD management, including adherence to a Mediterranean diet and regular physical activity, these measures are often insufficient in patients with advanced metabolic derangements. Pharmacologic agents such as glucagon-like peptide-1 receptor agonists (GLP-1) and sodium-glucose cotransporter-2 inhibitors (SGLT2) have shown promising effects on body weight, glycemic control, and hepatic steatosis [15].

Liraglutide, a representative of GLP-1 receptor agonists, has shown effectiveness in reducing body weight, liver steatosis, improving glycemic profiles, and lowering alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels in patients with MASLD [16].

Dapagliflozin, a representative of SGLT2 inhibitors, like liraglutide, has a positive effect on reducing blood glucose levels and decreasing liver inflammation. It is also suggested that this drug can reduce liver fat infiltration by inhibiting lipid and bile acid synthesis through suppression of LXRα-mediated pathways [17].

AIM

To assess and compare changes in lipid profiles and cardiovascular risk, based on five stratification scales, in patients with MASLD before and after 6 months of treatment with liraglutide or dapagliflozin. Additionally, to evaluate NT-proBNP levels in relation to the degree of hepatic steatosis before and after treatment.

MATERIALS AND METHODS

This prospective study was part of a dissertation project and was carried out at the clinical base of the Department of Internal Medicine №1, Bogomolets National Medical University, Kyiv, Ukraine. Ethical approval was obtained from the Bioethics Committee of the institution (Protocol №187, dated 23.09.2024).

The study followed the principles outlined in the Declaration of Helsinki, the Council of Europe Convention on Human Rights and Biomedicine, and relevant Ukrainian legislation. Written informed consent was obtained from all participants.

PATIENTS

The study population included patients aged 26–67 years with a confirmed diagnosis of MASLD, based on steatometric evidence of liver steatosis and at least one cardiometabolic risk factor in accordance with the 2023 MASLD criteria [18].

Exclusion criteria included: a history of cardiovascular events, liver cirrhosis, alcoholic liver disease, viral hepatitis, oncological and hematological diseases, pregnancy and lactation.

STUDY DESIGN

This was a prospective, randomized, parallel-group study conducted in two phases of stratification. Initially, 115 patients with MASLD were enrolled and randomized into two groups. The control group (n=36) received standardized non-pharmacological treatment, including a Mediterranean diet and at least 150 minutes per week of moderate-intensity physical activity. The remaining 79 patients comprised the intervention group, receiving the same lifestyle interventions plus a pharmacological agent.

In the second step, the intervention group was further randomized into two subgroups. Group IA (n=41) received dapagliflozin at a fixed dose of 10 mg once daily for 6 months. Group IB (n=38) received liraglutide starting at 0.6 mg once daily, titrated weekly to 1.8 mg and maintained for 6 months.

Randomization was computer-generated and stratified by age to ensure balanced distribution across the main study arms and subgroups.

VISITS

During the initial visit, all patients underwent a physical examination, complaints and anamnesis were collected, instrumental assessment included liver steatometry, performed using the Soneus P7 (UltraSign, Ukraine) device located at the clinical base of the Department of Internal Medicine №1, and laboratory tests—lipid profile and NT-proBNP.

Table 1. Baseline characteristics of study participants. X±SD or Me [25%;75%]

Indicators		Control group (n = 36)	Group IA (n = 41)	Group IB (n = 38)	Significance of difference, p
Age, years		43.3 ± 11	41.7 ± 10.7	39.6 ± 11.2	0.368
Age distribution	25-34 years	8 (22.2 %)	12 (29.3 %)	10 (26.3 %)	0.972
	35-44 years	11 (30.6 %)	13 (31.7 %)	13 (34.2 %)	
	45-54 years	12 (33.3 %)	10 (24.4 %)	9 (23.7 %)	
	55-67 years	5 (13.9 %)	6 (14.6 %)	6 (15.8 %)	
Sex	Men	25 (69 %)	24 (59 %)	30 (79 %)	0.147
	Women	11 (31 %)	17 (41 %)	8 (21 %)	
Severity of steatosis distribution	S1	9 (25 %)	14 (34.2 %)	8 (21.1 %)	0.526
	S2	14 (38.9 %)	12 (29.3 %)	18 (47.4 %)	
	S3	13 (36.1 %)	15 (36.5 %)	12 (31.5 %)	
Smoking (yes, %)		12 (33.3 %)	9 (21.9 %)	11 (28.9 %)	0.529
Medication use (yes, %) *		5 (13.6 %)	6 (14.6 %)	4 (10.5 %)	0.849
Diabetes mellitus (yes, %)		23 (63.9 %)	28 (68.3 %)	23 (60.5 %)	0.77
Arterial hypertension (yes, %)		7 (19.4 %)	9 (21.9 %)	7 (18.4 %)	0.921
Other comorbidities (yes, %) **		5 (13.8 %)	8 (19.5 %)	3 (7.9%)	0.329
Systolic blood pressure (mmHg)		132.5 ± 13.6	133.7 ± 16.6	132.2 ± 15.6	0.896
Body mass index (kg/m2)		30.95 ± 3.4	31.61 ± 3.1	32.16 ± 4.4	0.371
Total cholesterol (mmol/L)		5.4 ± 1.0	5.3 ± 1.2	5.2 ± 1.3	0.844
LDL-C (mmol/L)		3.3 ± 0.7	3.1 ± 0.8	3.1 ± 0.9	0.639
HDL-C (mmol/L)		1.14 [1.05; 1.3]	1.18 [1.00; 1.34]	1.13 [1.01; 1.24]	0.699
TG (mmol/L)		2.1 [1.89; 2.47]	2.3 [2.01; 2.75]	2.4 [2.01; 2.92]	0.110
Globorisk (10-year risk, %)		25.1 [16.2; 33.9]	29.7 [19.9; 43.1]	20.2 [11.6; 29.1]	0.167
Framingham (10-year risk, %)		12.4 [6.8; 19.9]	15.2 [6.1; 23.5]	12.8 [8.9; 25.9]	0.793
ACC/AHA ASCVD (10-year risk, %)		8.2 [3.8; 11.7]	10.4 [6.2; 18.9]	5.1 [3.4; 11.2]	0.317
PROCAM (10-year risk, points)		38.1 ± 10.1	41.2 ± 11.2	38.7 ± 11.9	0.440
WHO CVD (10-year risk, %)		16 [11; 17]	17 [12; 27]	15 [9; 27]	0.322
NT-proBNP (pg/ml)		22.7 [18.6; 26.1]	32.1 [23.6; 75.5]	16.9 [13.5; 36.8]	<0.001

Note: * - medication use includes levothyroxine, sertraline or antihypertensive therapy (perindopril, enalapril + hydrochlorothiazide or valsartan); ** - other comorbidities include autoimmune thyroiditis, hypothyroidism, depressive disorder

Source: compiled by the authors of this study

After 6 months of the prescribed treatment, all aforementioned laboratory and instrumental assessments were repeated to assess the results obtained.

CARDIOVASCULAR RISK ASSESSMENT

During the first visit and after 6 months, cardiovascular risk was assessed using five validated risk scales: ASCVD Risk Calculator (ACC/AHA), Framingham Risk Score, PROCAM, WHO cardiovascular risk charts and Globorisk [6-10].

These scales were selected because they incorporate type 2 diabetes mellitus and/or multiple lipid profile indicators, making them particularly relevant for assessing cardiovascular risk in patients with MASLD.

STATISTICAL ANALYSIS

Statistical analysis was performed using IBM SPSS Statistics version 29.0. The Shapiro–Wilk test was used to assess the normality of data distribution. Continuous variables were presented as mean ± standard deviation (SD) for normally distributed data, or as median with interquartile range [Median (Q1–Q3)] for non-normally distributed data. Between-group comparisons of continuous variables were performed using the independent samples t-test (for normally distributed data) or the Wilcoxon rank-sum test (for non-normally distributed data). Comparisons among more than two groups were conducted using one-way analysis of variance (ANOVA) or the Kruskal–Wallis test, as appropriate. Post hoc analysis for multiple pairwise comparisons was performed using the Bonferroni correction. Categorical

Table 2. Intra-group changes in lipid profile, NT-proBNP, and cardiovascular risk (five scales) before and after 6-month therapy in MASLD patients. X±SD or Me [25%;75%]

Indicators	Control group (n = 36)		Group IA (n = 41)		Group IB (n = 38)		Significance of difference, p
	Before	After	Before	After	Before	After	
Total cholesterol (mmol/L)	5.4 ± 1.0	4.8 ± 0.8	5.3 ± 1.2	4.6 ±1.0	5.2 ± 1.3	4.3 ± 1.0	p1<0.001 p2<0.001 p3<0.001
LDL-C (mmol/L)	3.2 [2.8; 3.6]	2.8 [2.4; 3.1]	3.1 ± 0.8	2.7 ±0.7	3.1 ± 0.9	2.5 ± 0.7	p1<0.001 p2<0.001 p3<0.001
HDL-C (mmol/L)	1.2 ± 0.22	1.26 ±0.23	1.2 [1.0; 1.3]	1.3 [1.1; 1.5]	1.1 [1.0; 1.2]	1.4 [1.2; 1.5]	p1<0.001 p2<0.001 p3<0.001
Triglycerides (mmol/L)	2.1 [1.9; 2.5]	1.83 [1.62; 2.24]	2.3 [2.0; 2.8]	1.9 [1.6; 2.2]	2.4 [2.0; 2.9]	1.8 [1.5; 2.1]	p1<0.001 p2<0.001 p3<0.001
Globorisk (10-year risk, %)	25.1 [16.2; 33.9]	19.5 [12.4; 27.6]	29.8 [19.9; 43.1]	19.6 [16.0; 35.1]	20.2 [11.6; 29.1]	13.6 [8.8; 26.5]	p1<0.001 p2<0.001 p3<0.001
Framingham (10-year risk, %)	12.4 [6.8; 19.9]	9.4 [5.2; 15.7]	15.2 [6.1; 23.5]	9.7 [4.5; 21.3]	12.8 [8.9; 25.9]	8.7 [5.9; 19.3]	p1<0.001 p2<0.001 p3<0.001
ACC/AHA ASCVD (10-year risk, %)	8.2 [3.8; 11.7]	6.0 [2.7; 9.1]	10.4 [6.2; 18.9]	6.1 [4.4; 14.0]	5.1 [3.4; 11.2]	3.0 [2.0; 10.5]	p1<0.001 p2<0.001 p3<0.001
PROCAM (10-year risk, points)	38.1 ± 10.1	33.0 ± 10.7	41.2 ± 11.2	34.0 ± 10.4	38.7 ± 11.9	32.0 ± 10.5	p1<0.001 p2<0.001 p3<0.001
WHO CVD (10-year risk, %)	16 [11; 17]	14 [9; 16]	17 [12; 27]	14 [12; 23]	15 [9; 27]	10 [7; 18]	p1<0.001 p2<0.001 p3<0.001
NT-proBNP (pg/ml)	22.7 [18.6; 26.1]	30.7 [24.6; 34.9]	32.1 [23.6; 75.5]	38.3 [27.8; 78.4]	16.9 [13.5; 36.8]	22.8 [18.3; 46.0]	p1=0.002 p2=0.396 p3=0.022

Note: p1 - statistical significance of the difference between the control group and Group IA, p2 - statistical significance of the difference between the control group and Group IB, p3 - statistical significance of the difference between Group IA and Group IB
Source: compiled by the authors of this study

variables were compared using the chi-squared (χ^2) test. A p-value < 0.05 was considered statistically significant.

ETHICS

This work complies with the principles of the Declaration of Helsinki.

RESULTS

Baseline demographic, clinical, and biochemical characteristics of the study participants are presented in Table 1. Patients were categorized into three groups: the control group (n = 36), Group IA (dapagliflozin, n = 41), and Group IB (liraglutide, n = 38). Most baseline characteristics were comparable across groups, allowing for a reliable assessment of treatment effects. However, baseline NT-proBNP levels differed significantly between groups and were therefore considered in the interpretation of outcome data.

Significant improvements in lipid profile indicators were observed in all groups after 6 months of treatment. Total cholesterol, LDL cholesterol, and triglyceride levels significantly decreased, while HDL cholesterol levels increased ($p < 0.001$ for all within-group comparisons). These findings reflect a favorable impact of all three treatment strategies—including lifestyle intervention, dapagliflozin, and liraglutide—on lipid metabolism in patients with MASLD. Detailed results are presented in Table 2.

All five cardiovascular risk scores demonstrated a statistically significant reduction after 6 months of

Table 3. Between-group comparison of lipid profile and cardiovascular risk scores after 6 months of treatment in patients with MASLD (X ± SD or Me [25%; 75%])

Indicators	Control group (n = 36)	Group IA (n = 41)	Group IB (n = 38)	Significance of difference, p
Total cholesterol (mmol/L)	4.86 ± 0.79	4.63 ±1.01	4.29 ± 1.04	p1 = 0.60 p2 = 0.04 p3 = 0.28
LDL-C (mmol/L)	2.85 ± 0.58	2.7 ± 0.71	2.45 ± 0.69	p1 = 0.62 p2 = 0.04 p3 = 0.29
HDL-C (mmol/L)	1.23 ± 0.23	1.27 ± 0.23	1.37 ± 0.21	p1 = 0.68 p2 = 0.02 p3 = 0.15
Triglycerides (mmol/L)	1.95 ± 0.44	1.90 ± 0.45	1.79 ± 0.39	p = 0.109
Globorisk (10-year risk, %)	19.5 [12.4; 27.6]	19.6 [16.0; 35.1]	13.6 [8.8; 26.5]	p = 0.106
Framingham (10-year risk, %)	9.4 [5.2; 15.7]	9.7 [4.5; 21.3]	8.7 [5.9; 19.3]	p = 0.975
ACC/AHA ASCVD (10-year risk, %)	6.0 [2.7; 9.1]	6.1 [4.4; 14.0]	3.0 [2.0; 10.5]	p = 0.24
PROCAM (10-year risk, points)	33.0 ± 10.7	34.0 ± 10.4	32.0 ± 10.5	p = 0.43
WHO CVD (10-year risk, %)	14 [9; 16]	14 [12; 23]	10 [7; 18]	p = 0.198

Note: p – statistical significance of the overall difference between the three groups; p1 – significance between control and Group IA; p2 – between control and Group IB; p3 – between Group IA and Group IB
Source: compiled by the authors of this study

treatment ($p < 0.001$ for all within-group comparisons), further supporting the positive impact of the interventions on cardiometabolic risk in patients with MASLD.

Changes in NT-proBNP levels differed across the groups. A statistically significant increase in median NT-proBNP was observed in the control group and the liraglutide group ($p < 0.05$ for both), while no significant change was detected in the dapagliflozin group.

The intergroup analysis after 6 months of treatment revealed significant improvements in lipid profile parameters in Group IB (liraglutide) compared to both the control group and Group IA (dapagliflozin) ($p < 0.05$). No statistically significant differences were observed between the control and dapagliflozin groups in terms of lipid profile ($p > 0.05$).

Despite overall reductions in cardiovascular risk across all groups, no significant differences in cardiovascular risk scores were found between the treatment groups ($p > 0.05$). Detailed comparisons are presented in Table 3.

DISCUSSION

In this prospective 6-month study, patients with MASLD underwent evaluation of lipid profile, NT-proBNP levels, and cardiovascular risk using five validated risk stratification tools (Globorisk, Framingham Risk Score, ASCVD Risk Calculator, PROCAM, and WHO CVD risk chart) [6-10].

All three treatment strategies—standardized lifestyle intervention, addition of dapagliflozin, and addition of liraglutide—were associated with significant improvements in lipid parameters and reductions in cardiovascular risk within each group [19]. These findings confirm the clinical utility of both pharmacologic agents in addressing dyslipidemia and cardiometabolic risk in MASLD patients [20].

Among the groups, the most pronounced improvement in lipid profile was observed in the liraglutide group, which showed significantly better outcomes compared to both the control and dapagliflozin groups [16]. This observation may be attributed to the known mechanisms of GLP-1 receptor agonists, including weight reduction, improved insulin sensitivity, and enhanced reverse cholesterol transport [21].

In contrast, the intergroup analysis of cardiovascular risk scores did not reveal statistically significant differences between the treatment arms. Although all groups showed significant within-group reductions, the observed similarity between treatment strategies

may reflect several factors, including the relatively short follow-up duration, modest baseline cardiovascular risk in the study population, and limited sensitivity of existing risk estimation tools to capture subtle therapeutic effects [11]. Future studies with longer observation periods and potentially more sensitive or dynamic risk assessment models may help to elucidate differential cardiovascular benefits between treatment approaches [22].

Notably, changes in NT-proBNP differed between groups. A significant increase in median NT-proBNP was observed in the control and liraglutide groups, whereas no significant change occurred in the dapagliflozin group. These divergent patterns warrant further investigation. The absence of significant NT-proBNP changes in the dapagliflozin group might reflect biological heterogeneity at baseline, given the wider distribution of initial NT-proBNP values in this group. This underscores the need for larger, more homogeneous samples to confirm potential treatment-related effects on NT-proBNP dynamics.

The lack of intergroup differences in cardiovascular risk scores and NT-proBNP changes highlights the complexity of interpreting these parameters over a relatively short treatment duration and underscores the importance of longer follow-up periods.

CONCLUSIONS

This 6-month prospective study demonstrated that both liraglutide and dapagliflozin significantly improved lipid profiles and reduced cardiovascular risk in patients with MASLD. Total cholesterol, LDL cholesterol, and triglyceride levels decreased, while HDL cholesterol increased in all groups, confirming the metabolic benefit of the interventions. Notably, intergroup analysis revealed more pronounced improvements in lipid parameters in the liraglutide group compared to both the control and dapagliflozin groups, suggesting a potential advantage of GLP-1 receptor agonists in modulating lipid metabolism.

Cardiovascular risk, assessed using five validated stratification tools, decreased consistently within all groups, although no significant differences were observed between the treatment arms.

NT-proBNP levels increased significantly in the control and liraglutide groups but remained unchanged in the dapagliflozin group; further studies are warranted to clarify the clinical relevance of this finding.

Overall, these results support the use of liraglutide and dapagliflozin as effective components of MASLD management, with liraglutide demonstrating superior efficacy in improving lipid parameters over a 6-month treatment period.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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