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Comparative effects of liraglutide and dapagliflozin on lipid profile and cardiovascular risk in patients with metabolic dysfunction-associated steatotic liver disease and type 2 diabetes: a 6-month randomized study

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Abstract: *Metabolic dysfunction-associated steatotic liver disease frequently coexists with type 2 diabetes mellitus, resulting in increased cardiometabolic risk. Pharmacologic agents such as glucagon-like peptide-1 receptor agonists and sodium-glucose cotransporter-2 inhibitors may improve lipid metabolism and cardiovascular outcomes, but comparative data remain limited. To evaluate and compare the magnitude of change (delta values) in lipid profile parameters and cardiovascular risk scores, assessed using five validated stratification tools, in patients with ьetabolic dysfunction-associated steatotic liver disease and type 2 diabetes mellitus following 6-month treatment with liraglutide or dapagliflozin. Materials and This 6-month prospective, randomized study included 72 patients with metabolic dysfunction-associated steatotic liver disease and type 2 diabetes mellitus allocated to three groups: control (lifestyle intervention; n=23), dapagliflozin (10 mg daily; n=26), or liraglutide (up to 1.8 mg daily; n=23). Lipid profiles and cardiovascular risk were assessed at baseline and after treatment using five validated tools (Globorisk, Framingham Risk Score, ASCVD Risk Calculator, PROCAM, WHO CVD chart). Intergroup comparisons were based on changes from baseline. All groups showed significant within-group improvements in lipid parameters, with reductions in total cholesterol, low-density lipoproteins, and triglycerides and increases in high-density lipoproteins ($p<0.001$). The liraglutide group demonstrated greater improvements in total cholesterol, low-density lipoproteins, and high-density lipoproteins compared to control and dapagliflozin ($p<0.01$). Cardiovascular risk scores declined significantly within each group. Between-group comparisons revealed significant differences for the Framingham score (favoring liraglutide over control) and the PROCAM score (favoring both pharmacologic treatments over control). No consistent differences were observed between liraglutide and dapagliflozin across other risk models. Both liraglutide and dapagliflozin improved lipid profiles and reduced cardiovascular risk in patients with metabolic dysfunction-associated steatotic liver disease and type 2 diabetes mellitus. Although no statistically significant superiority of liraglutide over dapagliflozin was confirmed for cardiovascular risk scores, a consistent trend toward greater lipid improvement was noted. Further studies with larger samples and longer follow-up are needed to clarify these findings.*

Keywords: Liver Diseases, Cardiovascular Risk, Liraglutide, Dapagliflozin, Type 2 Diabetes Mellitus, Metabolic Dysfunction-Associated Steatotic Liver Disease.

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Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is increasingly recognized as a major hepatic manifestation of systemic metabolic dysfunction [1]. Among its strongest associations is with type 2 diabetes mellitus (T2DM), a condition present in more than 55% of patients with MASLD and known to accelerate both hepatic and cardiovascular complications [2-3]. The coexistence of MASLD and T2DM has been associated with greater severity of steatosis, higher fibrosis progression rates, and increased risk of cardiovascular events [4].

Dyslipidemia plays a central role in the pathophysiology of MASLD, especially in patients with T2DM, in whom characteristic alterations include elevated triglycerides, decreased high-density lipoprotein cholesterol (HDL-C), and increased levels of small dense low-density lipoproteins (LDL-C) [5]. These changes not only promote hepatic fat accumulation but also represent key drivers of atherosclerotic cardiovascular disease (ASCVD) [6].

Recent guidelines underscore that cardiovascular disease, not liver-related complications, remains the leading cause of mortality in patients with MASLD, especially when accompanied by T2DM. This dual metabolic burden requires an integrated approach to risk reduction, including aggressive management of lipid abnormalities and careful assessment of individual cardiovascular risk [7].

Multiple validated tools are available to estimate 10-year cardiovascular risk, such as the ASCVD Risk Calculator (ACC/AHA), Framingham Risk Score, Prospective Cardiovascular Münster (PROCAM) Score, WHO cardiovascular risk charts, and Globorisk [8–12]. While these instruments are widely used, they typically do not account for hepatic steatosis or fibrosis, which may influence cardiovascular outcomes [5]. Consequently, dynamic assessment of changes (delta values) in these scores during therapy may offer additional insight into treatment effectiveness.

In recent years, several antidiabetic agents have gained attention for their hepatometabolic effects beyond glucose control. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) and sodium-glucose cotransporter-2 (SGLT2) inhibitors are widely used in the management of T2DM and have demonstrated benefits in reducing liver fat content, improving liver enzymes, lipid parameters and potentially lowering cardiovascular risk [13–14].

Liraglutide, a GLP-1 RA, reduces hepatic steatosis primarily through weight loss, improvement in insulin sensitivity, and anti-inflammatory effects. Additionally, it has been shown to enhance reverse cholesterol transport and increase HDL-C levels, contributing to improved lipid homeostasis [13].

Dapagliflozin, an SGLT2 inhibitor, exerts its effect by promoting glucosuria, improving glycemic control, and inducing mild caloric loss. In MASLD, its mechanisms include reduction of hepatic fat infiltration, improvement of mitochondrial function, and downregulation of lipogenesis via suppression of liver X receptor alpha (LXRα)-mediated pathways [15]. These complementary mechanisms suggest both agents may be effective in ameliorating the hepatic and cardiovascular burden in this high-risk population.

However, head-to-head comparisons of these agents in MASLD patients with T2DM are limited, particularly in terms of direct evaluation of the magnitude of change in lipid profile components and cardiovascular risk scores during treatment [16].

Aim

To evaluate and compare the magnitude of change (delta values) in lipid profile parameters and cardiovascular risk scores, assessed using five validated stratification tools, in patients with MASLD and type 2 diabetes mellitus following 6-month treatment with liraglutide or dapagliflozin.

Materials and Methods

This study was conducted as part of a dissertation project at the clinical base of the Department of Internal Medicine №1, Bogomolets National Medical University (Kyiv, Ukraine).

All procedures adhered to ethical standards set forth in the Declaration of Helsinki, the Council of Europe Convention on Human Rights and Biomedicine, and national legislation

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of Ukraine. All participants provided written informed consent prior to enrollment.

Patients. Eligible participants were adults aged 26 to 67 years with previously confirmed diagnoses of both metabolic dysfunction-associated steatotic liver disease (MASLD) and type 2 diabetes mellitus, as defined by the 2023 MASLD criteria [17].

Key exclusion criteria included any history of cardiovascular events, liver cirrhosis, alcoholic liver disease, viral hepatitis, malignancies, hematologic disorders, pregnancy, or lactation.

Study Design. This was a prospective, randomized, parallel-group study employing a two-stage stratification approach. A total of 72 patients met the inclusion criteria were enrolled and randomly assigned into two main groups. The control group (n = 23) received standard lifestyle modification therapy, which included adherence to a Mediterranean diet and at least 150 minutes of moderate-intensity aerobic activity per week.

The remaining 49 patients were allocated to the pharmacologic intervention group, which combined the same lifestyle recommendations with antidiabetic drug therapy. In the second phase of stratification, this group was subdivided into two treatment arms:

– Group IA (n = 26) received dapagliflozin at a fixed daily dose of 10 mg for 6 months.
– Group IB (n = 23) received liraglutide, initiated at 0.6 mg once daily and titrated weekly up to 1.8 mg, maintained throughout the 6-month period.

Randomization was performed using a computer-generated sequence and stratified by age to ensure balance across study arms and subgroups.

Study Visits. At baseline, each patient underwent a comprehensive clinical evaluation, including history-taking, physical examination, liver steatometry (Soneus P7, UltraSign, Ukraine), and laboratory testing (lipid profile, alanine aminotransferase [ALT], aspartate aminotransferase [AST]). All assessments were repeated after the 6-month intervention period to evaluate treatment effects.

Cardiovascular Risk Assessment. Cardiovascular risk was evaluated at baseline and after 6 months using five validated scoring tools: the ASCVD Risk Calculator (ACC/AHA), Framingham Risk Score, Prospective Cardiovascular Münster (PROCAM) Score, WHO CVD Risk Charts, and Globorisk [8-12]. These models were selected for their relevance to populations with metabolic dysfunction, as they incorporate type 2 diabetes mellitus and/or lipid profile indicators.

Statistical Analysis. Statistical analyses were performed using IBM SPSS Statistics software (version 29.0). Data distribution was assessed using the Shapiro–Wilk test. Normally distributed variables were expressed as mean ± standard deviation (SD), while non-normally distributed data were reported as median and interquartile range [Median (Q1–Q3)].

Comparisons between two groups were made using the independent samples t-test (for normal distribution) or the Wilcoxon rank-sum test (for non-normal distribution). Differences among three groups were analyzed using one-way ANOVA or the Kruskal–Wallis test, as appropriate. Post hoc pairwise comparisons were adjusted using the Bonferroni correction. Categorical variables were compared using the chi-squared (χ^2) test. Statistical significance was set at $p < 0.05$.

Results

Table 1 summarizes the baseline demographic, clinical, and biochemical characteristics of the study population. Participants were divided into three groups: control (n = 23), dapagliflozin group (Group IA, n = 26), and liraglutide group (Group IB, n = 23). Baseline comparability across groups supports the validity of subsequent intergroup comparisons.

Significant improvements in lipid profile parameters were observed in all three groups after 6 months of treatment. Total cholesterol, LDL cholesterol, and triglyceride levels decreased significantly, while HDL cholesterol levels increased ($p < 0.001$ for all within-group comparisons). Summary data are presented in Table 2.

Similarly, all five cardiovascular risk assessment tools demonstrated statistically significant reductions in each group following the intervention ($p < 0.05$ for all within-group comparisons).

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

Indicators		Control group (n = 23)	Group IA (n = 26)	Group IB (n = 23)	Significance of difference, p
Age, years		46.9 ± 9.5	46.9 ± 9.3	46.5 ± 9.5	p = 0.99
Sex	Men	15 (65 %)	19 (73 %)	17 (74 %)	p = 0.31
	Women	8 (35 %)	7 (27 %)	6 (26 %)	
Severity of steatosis distribution	S1	5 (21.7 %)	5 (19.2 %)	3 (13 %)	p = 0.70
	S2	10 (43.5 %)	8 (30.8 %)	11 (47.8 %)	
	S3	8 (34.8 %)	13 (50 %)	9 (39.2 %)	
Smoking (yes, %)		7 (30.4 %)	7 (26.9 %)	6 (26 %)	p = 0.94
Medication use (yes, %) *		3 (13 %)	4 (15.4 %)	2 (8.7 %)	p = 0.78
Arterial hypertension (yes, %)		5 (21.7 %)	7 (26.9 %)	4 (17.4 %)	p = 0.72
Other comorbidities (yes, %) **		2 (8.7 %)	5 (19.2 %)	2 (8.7%)	p = 0.43
Systolic blood pressure (mmHg)		131.8 ± 14.3	133.9 ± 16.5	135.2 ± 15.2	p = 0.78
Body mass index (kg/m2)		31.9 ± 3.0	32.5 ± 2.9	34.1 ± 3.9	p = 0.08
ALT (IU/L)		31 [18; 38]	32.5 [25; 43]	33 [25; 40]	p = 0.52
AST (IU/L)		27 [23; 41]	28 [24; 35]	27 [21; 38]	p = 0.85
Total cholesterol (mmol/L)		5.7 ± 1.0	5.9 ± 0.9	5.7 ± 0.9	p = 0.77
LDL-C (mmol/L)		3.4 ± 0.8	3.3 ± 0.8	3.5 ± 0.7	p = 0.84
HDL-C (mmol/L)		1.1 [1.1; 1.4]	1.3 [1.1; 1.4]	1.1 [1.0; 1.3]	p = 0.42
Triglycerides (mmol/L)		2.1 [1.9; 2.8]	2.3 [1.9; 2.8]	2.2 [1.9; 2.9]	p = 0.93
Globorisk (10-year risk, %)		27.9 [16.7; 33.9]	30.5 [21.4; 44.6]	20.8 [15.7; 40.2]	p = 0.22
Framingham (10-year risk, %)		15.1 [9.2; 21.5]	17.7 [13.5; 32.5]	15.2 [10.1; 30.3]	p = 0.62
ACC/AHA ASCVD (10-year risk, %)		8.9 [4.2; 11.7]	11.2 [7.8; 20.2]	7.9 [3.8; 17.7]	p = 0.30
PROCAM (10-year risk, points)		39.4 ± 9.4	44.3 ± 9.9	41.2 ± 11.2	p = 0.30
WHO CVD (10-year risk, %)		16 [13; 17]	19 [14; 27.5]	16 [10; 26]	p = 0.34

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

Changes in lipid profile parameters and cardiovascular risk scores across the three study groups over the 6-month treatment period are presented in Table 3. All groups demonstrated reductions in total cholesterol, LDL-C, and triglycerides, along with an increase in HDL-C. The liraglutide group showed significantly greater changes in total cholesterol ($p < 0.01$ vs. control), LDL-C ($p < 0.01$ vs. both groups), and HDL-C ($p < 0.01$ vs. both groups). Triglyceride reductions were also more substantial in both intervention groups compared to the control group ($p < 0.01$), with no significant difference between Group IA and Group IB ($p > 0.05$).

Regarding cardiovascular risk scores, all five tools demonstrated numerical reductions in each group. Statistically significant intergroup differences were observed for the Framingham and PROCAM scores. For the Framingham score, a greater reduction was observed in the liraglutide group compared to the control group ($p = 0.04$). In the PROCAM score, both the

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

Indicators	Control group (n = 23)		Group IA (n = 26)		Group IB (n = 23)		Significance of difference, p
	Before	After	Before	After	Before	After	
Total cholesterol (mmol/L)	5.7 ± 1.0	5.1 ± 0.8	5.9 ± 0.9	5.1 ± 0.8	5.7 ± 0.9	4.7 ± 0.8	p1 < 0.001 p2 < 0.001 p3 < 0.001
LDL-C (mmol/L)	3.4 ± 0.8	3.0 ± 0.7	3.3 ± 0.8	2.9 ± 0.7	3.5 ± 0.7	2.7 ± 0.6	p1 < 0.001 p2 < 0.001 p3 < 0.001
HDL-C (mmol/L)	1.1 [1.1; 1.4]	1.2 [1.1; 1.4]	1.3 [1.1; 1.4]	1.4 [1.2; 1.5]	1.1 [1.0; 1.3]	1.4 [1.2; 1.5]	p1 < 0.001 p2 < 0.001 p3 < 0.001
Triglycerides (mmol/L)	2.1 [1.9; 2.8]	1.87 [1.72; 2.38]	2.3 [1.9; 2.8]	1.8 [1.5; 2.2]	2.2 [1.9; 2.9]	1.6 [1.3; 2.1]	p1 < 0.001 p2 < 0.001 p3 < 0.001
Globorisk (10-year risk, %)	27.9 [16.7; 33.9]	22.1 [13.2; 27.6]	30.5 [21.4; 44.6]	21.9 [18.4; 37.2]	20.8 [15.7; 40.2]	14.8 [10.5; 28.6]	p1 < 0.001 p2 < 0.001 p3 < 0.001
Framingham (10-year risk, %)	15.1 [9.2; 21.5]	11.9 [7.3; 16.6]	17.7 [13.5; 32.5]	14.9 [9.7; 25.4]	15.2 [10.1; 30.3]	12.7 [5.9; 20.4]	p1 < 0.001 p2 < 0.001 p3 < 0.001
ACC/AHA ASCVD (10-year risk, %)	8.9 [4.2; 11.7]	6.5 [3.1; 9.1]	11.2 [7.8; 20.2]	6.4 [5.5; 15.2]	7.9 [3.8; 17.7]	4.1 [2.2; 11.1]	p1 < 0.001 p2 < 0.001 p3 < 0.001
PROCAM (10-year risk, points)	39.4 ± 9.4	34.8 ± 9.7	44.3 ± 9.9	36.4 ± 9.0	41.2 ± 11.2	34.9 ± 9.6	p1 < 0.001 p2 < 0.001 p3 < 0.001
WHO CVD (10-year risk, %)	16 [13; 17]	15 [11; 16]	19 [14; 27.5]	16 [13; 24]	16 [10; 26]	13 [8; 18]	p1 < 0.001 p2 = 0.002 p3 < 0.001

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.

dapagliflozin group (p = 0.02) and the liraglutide group (p = 0.04) showed significantly greater reductions versus control. No statistically significant differences were found between the intervention groups for any of the cardiovascular risk tools (p > 0.05).

Discussion

In this 6-month prospective study, patients with MASLD and type 2 diabetes mellitus were evaluated for changes in lipid profile parameters and cardiovascular risk using five validated stratification tools (Globorisk, Framingham Risk Score, ASCVD Risk Calculator, PROCAM, and WHO CVD risk chart) [8-12]. The analysis focused on the magnitude of change (delta values) to assess the comparative effectiveness of liraglutide and dapagliflozin, alongside standardized lifestyle intervention.

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Table 3. Intergroup comparison of changes (Δ) in lipid profile and cardiovascular risk scores after 6 months of treatment (X ± SD or Me [25%; 75%]).

Indicators	Control group (n = 23)	Group IA (n = 26)	Group IB (n = 23)	Significance of difference, p
Total cholesterol (mmol/L)	-0.54 [-0.65; -0.44]	-0.74 [-0.88; -0.65]	-0.97 [-1.21; -0.85]	p1 < 0.05 p2 < 0.01 p3 < 0.01
LDL-C (mmol/L)	-0.42 [-0.48; -0.32]	-0.44 [-0.48; -0.35]	-0.77 [-0.83; -0.66]	p1 > 0.05 p2 < 0.01 p3 < 0.01
HDL-C (mmol/L)	0.05 [0.03; 0.05]	0.13 [0.1; 0.14]	0.23 [0.2; 0.26]	p1 < 0.01 p2 < 0.01 p3 < 0.01
Triglycerides (mmol/L)	-0.28 [-0.31; -0.22]	-0.53 [-0.61; -0.44]	-0.65 [-0.89; -0.59]	p1 < 0.01 p2 < 0.01 p3 > 0.05
Globorisk (10-year risk, %)	-0.52 ± 1.46	-0.64 ± 2.87	-0.67 ± 4.56	p = 0.35
Framingham (10-year risk, %)	-3.59 ± 1.78	-4.76 ± 3.43	-6.28 ± 4.58	p1 = 0.52 p2 = 0.04 p3 = 0.34
ACC/AHA ASCVD (10-year risk, %)	-2.34 [-2.93; -1.17]	-3.76 [-5.42; -1.82]	-2.66 [-5.68; -1.41]	p = 0.27
PROCAM (10-year risk, points)	-4.67 ± 2.83	-7.87 ± 4.4	-7.62 ± 3.58	p1 = 0.02 p2 = 0.04 p3 = 0.97
WHO CVD (10-year risk, %)	-2 [-3; -1]	-3 [-4; -1]	-3 [-7.5; -2]	p = 0.06

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.

Within-group analysis demonstrated significant improvements in all lipid profile components across the three study groups, confirming the metabolic benefit of both pharmacological and non-pharmacological approaches [18]. Notably, the liraglutide group showed the most pronounced improvements in total cholesterol, LDL-C, and HDL-C levels compared to the control and dapagliflozin groups [19]. These findings are consistent with previously reported data on the lipid-modulating effects of GLP-1 receptor agonists, which are thought to enhance reverse cholesterol transport, reduce hepatic lipogenesis, and improve insulin sensitivity [14].

Triglyceride levels also decreased significantly in all groups, with both pharmacologic interventions outperforming lifestyle modification alone. Although liraglutide demonstrated numerically greater triglyceride reduction compared to dapagliflozin, the difference did not reach statistical significance [20].

Cardiovascular risk, as assessed by all five tools, declined significantly within each group. Intergroup comparisons revealed that the liraglutide group achieved a greater reduction in Framingham risk score compared to the control group, while both active treatment groups demonstrated significantly greater reductions in PROCAM scores. No statistically significant differences were observed between the two pharmacologic agents across the other risk models, which may reflect the overall effectiveness of both drugs in addressing cardiometabolic risk,

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as well as the inherent limitations of standard cardiovascular risk calculators in detecting subtle therapeutic differences, particularly in populations with existing metabolic disease [21].

Although liraglutide did not demonstrate statistically significant superiority over dapagliflozin in the change of lipid parameters and cardiovascular risk scores, a numerical trend toward a greater effect was observed. This trend can be explained by the limited duration of treatment and the small sample size. This observation warrants further investigation in larger, longer-term studies.

Conclusions

This 6-month prospective study demonstrated that both liraglutide and dapagliflozin significantly improved lipid profiles and reduced cardiovascular risk in patients with MASLD and type 2 diabetes mellitus. Total cholesterol, LDL cholesterol, and triglyceride levels decreased, while HDL cholesterol increased in all study groups.

Intergroup comparisons based on changes from baseline (delta values) revealed more pronounced improvements in total cholesterol, LDL-C, and HDL-C levels 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 significantly within each group. Statistically significant intergroup differences were observed for the Framingham score, favoring liraglutide over control, and for the PROCAM score, favoring both pharmacological treatments over lifestyle modification alone, with no consistent difference between liraglutide and dapagliflozin.

The observation that only the PROCAM score demonstrated statistically significant intergroup differences for both pharmacological treatments, while the Framingham score detected a significant difference only for liraglutide versus control, suggests that these models may be more sensitive to capturing between-group treatment effects in this specific patient population. However, this hypothesis requires confirmation in larger studies.

Informed Consent of Patients

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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Conflict of Interest

The authors declare that they have no financial, academic, or personal conflicts of interest related to the publication of this article.

Consent to publication

The author has read and approved the final version of the manuscript. All authors consented to the publication of this manuscript.

AI Disclosure

No AI tools were used in the preparation of this manuscript.

Ethical approval

Approved by the Bioethics Committee, protocol №187, dated 23.09.2024.

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Порівняльний вплив ліраглутиду та дапагліфлозину на ліпідний профіль і серцево-судинний ризик у пацієнтів з стеатотичною хворобою печінки, асоційованою з метаболічною дисфункцією, та цукровим діабетом 2 типу: рандомізоване 6-місячне дослідження

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Анотація: *Стеатотична хвороба печінки, асоційована з метаболічною дисфункцією, часто поєднується з цукровим діабетом 2 типу, що призводить до підвищення кардіометаболічного ризику. Фармакологічні засоби, такі як агоністи рецепторів глюкагоноподібного пептиду-1 та інгібітори натрійзалежного котранспортера глюкози-2, можуть покращувати ліпідний обмін і серцево-судинні наслідки, однак порівняльні дані залишаються обмеженими. Оцінити та порівняти величину змін (дельти) показників ліпідного профілю та серцево-судинного ризику, розрахованого за п'ятьма валідованими шкалами, у пацієнтів із стеатотичною хворобою печінки, асоційованою з метаболічною дисфункцією, і цукровим діабетом 2 типу після 6-місячного лікування ліраглутидом або дапагліфлозином. Це 6-місячне проспективне рандомізоване дослідження включало 72 пацієнтів із стеатотичною хворобою печінки, асоційованою з метаболічною дисфункцією, і цукровим діабетом 2 типу, розподілених на три групи: контроль (модифікація способу життя; n=23), дапагліфлозин (10 мг/добу; n=26) або ліраглутид (до 1,8 мг/добу; n=23). Ліпідний профіль та серцево-судинний ризик оцінювали на початку та після лікування за п'ятьма валідованими шкалами (Globorisk, Framingham Risk Score, ASCVD Risk Calculator, PROCAM, WHO CVD chart). Міжгрупові порівняння проводили за змінами від вихідного рівня. У всіх групах відмічено достовірні внутрішньогрупові покращення ліпідних показників: зниження рівнів загального холестерину, ліпопротеїнів низької щільності та тригліцеридів і підвищення ліпопротеїнів високої щільності (p<0.001). Група ліраглутиду продемонструвала більш виражені покращення загального холестерину, ліпопротеїнів низької щільності та ліпопротеїнів високої щільності порівняно з контрольною групою та групою дапагліфлозину (p<0.01). Показники серцево-судинного ризику достовірно знизилися в кожній групі. Міжгрупові порівняння показали значущі відмінності для шкали Framingham (на користь ліраглутиду порівняно з контролем) та шкали PROCAM (на користь обох фармакологічних втручань порівняно з контролем). Узгоджених відмінностей між ліраглутидом і дапагліфлозином за іншими моделями ризику не виявлено. Ліраглутид і дапагліфлозин покращують ліпідний профіль та знижують серцево-судинний ризик у пацієнтів із стеатотичною хворобою печінки, асоційованою з метаболічною дисфункцією, і цукровим діабетом 2 типу. Хоча статистично значущої переваги ліраглутиду над дапагліфлозином*

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щодо показників серцево-судинного ризику не встановлено, спостерігалася стійка тенденція до більш вираженого покращення ліпідного профілю. Необхідні подальші дослідження з більшою вибіркою та тривалішим спостереженням для уточнення цих результатів.

Ключові слова: стеатотична хвороба печінки, асоційована з метаболічною дисфункцією, серцево-судинний ризик, ліраглутид, дапагліфлозин, цукровий діабет 2 типу.