CHAPTER 12

METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE AS A FACTOR OF CARDIO-METABOLIC RISKS INTENSIFICATION IN PATIENTS WITH ARTERIAL HYPERTENSION

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INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is an important factor contributing to the severity of hypertension (HTN). The presence of MASLD is associated with increased insulin resistance, chronic systemic inflammation, endothelial dysfunction, and activation of neurohumoral mechanisms, which contribute to elevated blood pressure and impaired blood pressure control[1], [2].

Patients with a combination of hypertension and MASLD are more likely to experience adverse cardiovascular events, myocardial remodeling, and an increased risk of atherosclerosis and cardiovascular mortality. Thus, MASLD should be viewed not merely as a liver disease but as a component of the systemic cardiometabolic continuum, which significantly influences the prognosis in patients with hypertension[2], [3].

1. Emergence of prerequisites of the problem and formulation of the problem

Current trends toward an increasing prevalence of metabolic disorders are leading to a rise in the number of patients with MASLD and hypertension. The combination of these conditions is of particular clinical significance due to their shared pathogenic mechanisms, among which insulin resistance, systemic inflammation, oxidative stress, and impaired vascular regulation play a leading

[1] Khan M.S., Javaid S.S., Dinani A., Wegermann K., Pandey A., Bhatt A.S. et al. The Basics of Metabolic Dysfunction–Associated Steatotic Liver Disease for Cardiologists: Pathophysiology, Diagnosis, and Treatment. Journal of the American College of Cardiology. 2025;86(20):1861–1884. doi:10.1016/j.jacc.2025.08.100

[2] Hakeem A., van Berlo J., Revelo X.S. The Cardiohepatic Axis in Metabolic Disease: Liver to Heart. JACC: Basic to Translational Science. 2025;10(7). doi:10.1016/j.jacbts.2025.05.004.

[3] Møller S., Kimer N., Hove J.D., Barløse M., Gluud L.L. Cardiovascular Disease and Metabolic Dysfunction-Associated Steatotic Liver Disease: Pathophysiology and Diagnostic Aspects. European Journal of Preventive Cardiology. 2025. doi:10.1093/eurjpc/zwae306.

role[1,3]. A growing body of scientific evidence confirms that MASLD is not only a manifestation of metabolic changes but also a factor that can negatively affect the functional state of the cardiovascular system[4], [5].

Particular attention is drawn to the issue of how MASLD affects the course of hypertension and contributes to an unfavorable cardiovascular prognosis. In patients with comorbidities, signs of subclinical atherosclerosis, myocardial remodeling, and an increased risk of cardiovascular complications are more frequently observed[6,7]. Despite active research into this issue, the mechanisms underlying the relationship between MASLD and hypertension, as well as the role of hepatic metabolic changes in the progression of cardiovascular risk, require further clarification.

2. The analysis of existing methods for solving the problem and formulating a task for the optimal development

An analysis of current scientific data indicates that the issue of the co-occurrence of MASLD and hypertension is being actively studied in the context of cardiometabolic risk. Existing studies are primarily focused on assessing the role of insulin resistance, systemic inflammation, endothelial dysfunction, and lipid metabolism disorders in the progression of cardiovascular complications in patients with MASLD[4], [6]. Clinical, laboratory, and instrumental methods are used for risk assessment, including lipid profile analysis, measurement of inflammatory markers, ultrasound of the liver, elastography, and cardiovascular risk scales[7], [8]. However, research findings remain inconclusive regarding the extent of MASLD's impact on the course of hypertension and the development of cardiovascular complications.

Despite a significant number of scientific studies, the issues of early cardiovascular risk stratification in patients with a combination of MASLD and hypertension, as well as the identification of the most informative predictors of

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[4] Zhou X.-D., Zheng M.-H. Heart–liver co-management in MASLD: from concept to clinical practice. Nature Reviews Gastroenterology & Hepatology. 2026;23:117–118. doi:10.1038/s41575-025-01144-x.

[5] Ratti C., Malaguti M., D'Aniello E., Bellasi A., Sanna G. Understanding MASLD – from molecular pathogenesis to cardiovascular risk: A concise review for the clinical cardiologist. Atherosclerosis. 2025;409:120495. doi:10.1016/j.atherosclerosis.2025.120495.

[6] Ismail B., Nabilou P., Gluud L.L., Møller S. Cardiovascular implications of MASLD: overview of the evidence. Scandinavian Journal of Gastroenterology. 2025;60(9):910–918. doi: 10.1080/00365521.2025.2536585.

[7] Lebovics N., Heering G., Frishman W.H., Lebovics E. Lean MASLD and Cardiovascular Disease: A Review. Cardiology in Review. 2025. doi:10.1097/CRD.0000000000000893.

[8] Younossi Z.M., Golabi P., Paik J.M., Henry A., Van Dongen C., Henry L. The Global Epidemiology of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) and Cardiovascular Risk. Hepatology. 2024;79(4):1362–1374. doi:10.1097/HEP.0000000000000654.

an unfavorable prognosis, remain under-researched[9],[10]. This necessitates further study of the specific relationship between metabolic changes in the liver and indicators of cardiovascular status. In this regard, a promising direction is the optimization of approaches to comprehensive cardiovascular risk assessment and the refinement of algorithms for the early identification of patients with a high probability of developing cardiovascular complications.

Aim: To evaluate the pathophysiological role of MASLD in the development of cardiometabolic risks in patients with hypertension.

Materials and methods. The study was conducted at the Department of Internal Medicine № 1 and was approved by the Ethics and Bioethics Committee of the Kharkiv National Medical University. During the study, ethical standards were adhered to in accordance with the "Rules of Ethical Principles for Conducting Medical Research Involving Human Subjects," as approved by the Declaration of Helsinki (1964–2013), the principles of good clinical practice (ICH GCP, 1996), and current national legislation (Order of the Ministry of Health of Ukraine № 281 dated 01.11.2008, "On the Approval of Instructions for the Conduct of Clinical Trials of Medicinal Products and the Review of Clinical Trial Materials, and Model Regulations on Ethics Committees").

A total of 120 patients were examined: the study group consisted of 63 patients with MASLD against a background of HTN, including 32 men and 31 women aged 38 to 59 years (Me = 48.4; 95% CI = 47.2-49.6) and a comparison group – 57 patients with isolated HTN, 28 men and 29 women aged 39 to 59 years (Me = 48.3; 95% CI = 46.8-49.8).

The duration of MASLD in the group of patients with comorbid diseases ranged from 2 to 16 years (Me = 6.57; 95% CI = 5.81-7.32), and hypertension had been diagnosed in the study participants between 2 and 19 years prior (Me = 8.41; 95% CI = 7.34-9.48).

The control group consisted of 20 healthy volunteers, randomized by sex and age – 12 women and 8 men aged 38 to 56 years (Me = 47.13; 95% CI = 45.1-49.1).

The inclusion criteria: the presence of MASLD at the steatohepatitis stage, as determined by laboratory and imaging tests, with concomitant hypertension; the presence of isolated stage I–II, grade 1–2 hypertension; age 18–60 years; and the provision of written informed consent to participate in the study.

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[9] Simon T.G., Roelstraete B., Khalili H., Hagström H., Ludvigsson J.F. Mortality and Cardiovascular Outcomes in Patients With MASLD. Clinical Gastroenterology and Hepatology. 2025;23(2):415–424. doi:10.1016/j.cgh.2024.06.018.

[10] Li Y., Chen Z., Wang X., Zhao Q. Association Between MASLD and Arterial Hypertension: Current Evidence and Clinical Perspectives. Frontiers in Cardiovascular Medicine. 2025;12:1458721. doi:10.3389/fcvm.2025.1458721.

The exclusion criteria: other diffuse and focal liver diseases (including alcohol-related liver disease, viral hepatitis B, C, and D, toxic and drug-induced hepatitis, liver cirrhosis, etc.); stage III, grade 3 hypertension; ischemic heart disease; type I and II diabetes mellitus and other endocrine disorders; oncological diseases; systemic connective tissue diseases; secondary (symptomatic) hypertension; acute and chronic inflammatory diseases of internal organs in the acute phase of any localization, obesity of any stage (BMI ≥ 30 kg/m²), age > 60 years, patient refusal to participate at any stage of the study.

The diagnosis of MASLD at the stage of steatohepatitis was established in accordance with the recommendations of the European Association for the Study of the Liver (EASL, 2024), European Association for the Study of Diabetes (EASD), and European Association for the Study of Obesity (EASO)[11].

The diagnosis of arterial hypertension was determined according to the Order of the Ministry of Health of Ukraine dated 12.09.2024 №1581 "On Approval of the Unified Clinical Protocol of Primary and Specialized Medical Care 'Hypertensive Disease (Arterial Hypertension)'"[12], as well as the recommendations of the 2024 European Society of Hypertension/European Society of Cardiology (ESH/ESC) Clinical Practice Guidelines for the Management of Arterial Hypertension[13].

Office systolic (SBP) and diastolic (DBP) blood pressure were measured auscultatorily using a sphygmomanometer BP AG1-40 (Microlife AG, Switzerland) at rest, three times at 1-2-minute intervals, and the arithmetic means were calculated.

Body mass index (BMI, kg/m²) was calculated using the Quetelet formula – the ratio of body weight (in kilograms) to the square of height (in meters). Results were evaluated according to the WHO classification: BMI values in the range of 18.5 – 24.9 were considered normal, while BMI values between 25.0 and 29.9 were considered indicative of overweight.

Waist circumference (WC) and hip circumference (HC) were measured using a measuring tape (measurement range: 1–200 cm; measurement error: ±1%). According to WHO recommendations, a waist circumference of ≥94 cm

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[11] European Association for the Study of the Liver (EASL), European Association for the Study of Diabetes (EASD), European Association for the Study of Obesity (EASO). EASL–EASD–EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). Journal of Hepatology. 2024;81(3):492–542. doi:10.1016/j.jhep.2024.04.031.

[12] Наказ Міністерства охорони здоров'я України №1581 від 12.09.2024 «Про затвердження Уніфікованого клінічного протоколу первинної та спеціалізованої медичної допомоги "Гіпертонічна хвороба (артеріальна гіпертензія)"».

[13] McEvoy J.W., McCarthy C.P., Bruno R.M. et al. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. European Heart Journal. 2024.

for men and ≥80 cm for women was considered a marker of increased risk, while WC values of ≥102 cm for men and ≥88 cm for women were considered signs of abdominal obesity[14]. According to WHO recommendations, a waist-to-height ratio (WHR) of ≥0.90 for men and ≥0.85 for women, and a waist-to-hip ratio (WHR) of ≥0.50 for all patients regardless of sex, were considered indicators of abdominal obesity[15].

To analyze the lipid profile using an enzymatic colorimetric method, total cholesterol (TC), triglycerides (TG), very low-density lipoprotein cholesterol (VLDL-C), and high-density lipoprotein cholesterol (HDL-C) were measured by precipitation with phosphotungstic acid in the presence of Mg^{2+} ions. Low-density lipoproteins (LDL-C) levels were calculated using the Friedewald W.T. formula (1972).

The atherogenic index (AI) is determined as the base-10 logarithm of the ratio of fasting serum triglyceride levels to high-density lipoprotein cholesterol levels.

Fasting glucose levels were measured using the COBAS s311 biochemical analyzer from Roche Diagnostics (Switzerland) via a standard enzymatic method employing hexokinase; glycated hemoglobin (HbA1c) levels were determined using an immunoturbidimetric assay. To assess insulin levels, the ECLIA (Electrochemiluminescent Immunoassay) test was used with the Elecsys Insulin kit for the COBAS e411 analyzer from Roche Diagnostics (Switzerland). To determine the presence of insulin resistance, the HOMA-IR (Homeostasis Model Assessment of Insulin Resistance) index was calculated using the formula:

$$\text{HOMA-IR} = \frac{\text{Insulin level (mU/mL)} \times \text{Glucose level (mMol/L)}}{22,5}$$

Ultrasound examination of the abdominal organs was performed using the Soneus P7 scanning system (Ultrasign, Ukraine). Using the “AC” mode, a quantitative assessment of fatty structures in the liver parenchyma was performed – the value of the linear attenuation coefficient of ultrasound in the liver parenchyma (AC, dB/cm) was measured. Using the “SE” mode of the ultrasound machine, two-dimensional shear wave elastography (2D-SWE) was performed – the stiffness of the liver parenchyma (Elasticity (E), kPa; m/s) was determined to assess the degree of liver fibrosis. The measurement results were

[14] Baioumi A. Comparing Measures of Obesity: Waist Circumference, Waist-Hip, and Waist-Height Ratios. Nutrition in the Prevention and Treatment of Abdominal Obesity. 2019:29-40. doi:10.1016/b978-0-12-816093-0.00003-3.

[15] Corrêa MM, Facchini LA, Thumé E, et al. The ability of waist-to-height ratio to identify health risk. Rev Saude Publica. 2019;53:66. doi:10.11606/s1518-8787.2019053000895.

analyzed by correlating liver stiffness with the stages of fibrosis and cirrhosis according to the METAVIR scale[16].

The results were statistically analyzed using the software packages "Excel 2019" (Microsoft), "STATISTICA 8.0" (StatSoft Inc.), and "SPSS 16.0.1 for Windows" (SPSS Inc., Chicago, IL, USA).

RESULTS

The mean systolic and diastolic blood pressure in patients with MASLD and HTN were significantly higher than in the other groups (Table 1).

Table 1

Blood pressure parameters of the examined patients

Parameter	MASLD and HTN (n = 63)	HTN (n = 57)	Control group (n = 20)	Significance between groups
SBP, mm Hg	140,00 (95% CI 137,86; 140,55)	134,00 (95% CI 132,12; 135,28)	122,50 (95% CI 121,94; 126,56)	p_{1-2}<0,001* p_{1-3}<0,001* p_{1-3}<0,001*
DBP, mm Hg	85,00 (95% CI 82,72; 86,17)	80,00 (95% CI 78,34; 81,69)	75,00 (95% CI 73,75; 79,25)	p_{1-2}<0,001* p_{1-3}<0,001* p_{2-3}=0,004*

*Notes: * – the difference is statistically significant between groups (p<0.05);*
p_{1-2} – difference between the MASLD + HTN group and the group with isolated HTN;
p_{1-3} – difference between the MASLD + HTN group and the control group;
p_{2-3} – difference between the group with isolated HTN and the control group.

A significant difference in BMI was observed between the study groups and the control group, whereas the corresponding values for patients with comorbidity and isolated hypertension did not differ. The medians of BMI, waist circumference, hip circumference, and waist-to-hip ratio in patients from both groups were higher than those in the control group (Table 2).

An analysis of the distribution of pathological anthropometric characteristics among the examined patients revealed that the group with MASLD and HTN included a significantly higher number of patients with a BMI above 25 kg/m² (65.1% vs. 45.6% in patients with isolated hypertension, p=0.032) and individuals at risk for abdominal obesity based on waist circumference (41.3% vs. 21.1%, p=0,017) (Table 3).

[16] Bedossa P., Poynard T. An algorithm for the grading of activity in chronic hepatitis C. The METAVIR Cooperative Study Group // Hepatology. – 1996. – Vol. 24, № 2. – P. 289–293. DOI: 10.1002/hep.510240201.

Table 2

Anthropometric parameters of the examined patients

Parameter	MASLD and HTN (n = 63)	HTN (n = 57)	Control group (n = 20)	Significance between groups
BMI, kg/m^2	26,9 (95% ДІ 24,45; 29,34)	26,1 (95% ДІ 25,41; 26,72)	22,70 (95% ДІ 22,41 23,46)	p_{1-2}=0,498 p_{1-3}<0,001* p_{2-3}<0,001*
WC, cm	86,0 (95% ДІ 82,9; 87,4)	83,1 (95% ДІ 81,22; 85,51)	71,40 (95% ДІ 67,57; 75,13)	p_{1-2}=0,343 p_{1-3}<0,001* p_{2-3}<0,001*
HC, cm	98,0 (95% ДІ 95,95; 98,40)	97,2 (95% ДІ 95,93; 97,89)	96,50 (95% ДІ 90,79; 97,61)	p_{1-2}=0,301 p_{1-3}=0,177 p_{2-3}=0,436
WHR (WC/HC)	0,89 (95% ДІ 0,86; 0,90)	0,87 (95% ДІ 0,85; 0,89)	0,76 (95% ДІ 0,74; 0,77)	p_{1-2}=0,413 p_{1-3}<0,001* p_{2-3}<0,001*

*Notes: * – the difference is statistically significant between groups (p<0.05);*
p_{1-2} – difference between the MASLD + HTN group and the group with isolated HTN;
p_{1-3} – difference between the MASLD + HTN group and the control group;
p_{2-3} – difference between the group with isolated HTN and the control group.

Table 3

Distribution of pathological anthropometric characteristics among the examined patients

Parameter	MASLD and HTN (n = 63)		HTN (n = 57)		Significance of the difference (based on the χ^2 criteria)
	n	%	n	%	
Signs of overweight based on BMI (25.0–29.9 for men and women)	41	65,1	26	45,6	df=1 c
Signs of risk for abdominal obesity based on WC (≥94 cm for men, ≥80 cm for women)	26	41,3	12	21,1	df=1 c
Signs of abdominal obesity based on WC (≥102 cm for men, ≥88 cm for women)	13	20,6	7	12,3	df=1 c
Signs of abdominal obesity based on WHR (≥0.90 for men, ≥0.85 for women)	30	47,6	21	36,8	df=1 c

*Note: * – the difference is statistically significant between the group with MASLD + HTN group and the group with isolated HTN; (p < 0.05).*

Table 4

Lipid profile parameters of the examined patients

Parameter	MASLD and HTN (n = 63)	HTN (n = 57)	Control group (n = 20)	Significance between groups
TC, mmol/L	6,78 (95% ДІ 6,60; 6,97)	5,61 (95% ДІ 5,34; 5,92)	4,00 (95% ДІ 3,76; 4,24)	$p_{1-2} < 0,001$* $p_{1-3} < 0,001$* $p_{2-3} < 0,001$*
LDL-C, mmol/L	4,11 (95% ДІ 3,99; 4,44)	3,31 (95% ДІ 3,17; 3,58)	2,28 (95% ДІ 2,25; 2,65)	$p_{1-2} < 0,001$* $p_{1-3} < 0,001$* $p_{2-3} < 0,001$*
VLDL-C, mmol/L	0,78 (95% ДІ 0,74; 0,82)	0,63 (95% ДІ 0,59; 0,67)	0,50 (95% ДІ 0,45; 0,56)	$p_{1-2} < 0,001$* $p_{1-3} < 0,001$* $p_{2-3} = 0,017$*
HDL-C, mmol/L	1,01 (95% ДІ 1,01; 1,09)	1,14 (95% ДІ 1,13; 1,21)	1,17 (95% ДІ 1,13; 1,28)	$p_{1-2} < 0,001$* $p_{1-3} < 0,001$* $p_{2-3} = 0,798$
Triglycerides, mmol/L	1,90 (95% ДІ 1,84; 2,02)	2,04 (95% ДІ 1,91; 2,09)	1,27 (95% ДІ 1,16; 1,38)	$p_{1-2} = 0,131$ $p_{1-3} < 0,001$* $p_{2-3} < 0,001$*
Atherogenic index	5,57 (95% ДІ 5,27; 5, 87)	4,09 (95% ДІ 3,82; 4,31)	2,37 (95% ДІ 2,11; 2,64)	$p_{1-2} < 0,001$* $p_{1-3} < 0,001$* $p_{2-3} < 0,001$*

*Notes: * – the difference is statistically significant between groups (p<0.05);*
p_{1-2} – difference between the MASLD + HTN group and the group with isolated HTN;
p_{1-3} – difference between the MASLD + HTN group and the control group;
p_{2-3} – difference between the group with isolated HTN and the control group.

Almost all lipid profile parameters in both study groups showed statistically significant differences compared to control values, except HDL-C, whose levels were significantly reduced only in the group with MASLD and HTN (1.01 mmol/L (95% CI 1.01–1.09), p<0.001). Statistically significant differences were found in the levels of total cholesterol, LDL-C, HDL-C, and the atherogenic index (Table 4).

The mean fasting glucose level in patients with MASLD and HTN was 5.87 mmol/L (95% CI 5.76–5.98) and was significantly higher than the corresponding value in the group with isolated HTN (5.31 mmol/L (95% CI 5.03; 5.57), p<0.001), but in both groups, blood glucose levels were higher relative to control values (4.52 mmol/L (95% CI 4.48; 4.61). In the group with MASLD and HTN, 32 cases of hyperglycemia were identified (50.8%), while in the group with isolated HTN, there were only 4 patients (7.0%) with elevated glucose levels (df=1, $\chi^2 = 27.308$, p<0.001).

Glycated hemoglobin levels showed similar correlations and were significantly higher in the group with MASLD and HTN (5.57% (95% CI 5.41; 5.55)) compared to patients with isolated HTN (5.21% (95% CI 5.08; 5.29), p<0.001) and relatively healthy volunteers (4.47% (95% CI 4.40; 4.53), p<0.001).

Insulin levels in patients with MASLD and HTN averaged 17.03 μU/L (95% CI 16.03; 17.25), which was significantly higher than in the comparison group (13.22 μU/L (95% CI 12.89; 13.51), p<0.001) and in the control group (11.40 μU/L (95% CI 11.27; 12.44), p<0.001) (Figure 1).

The HOMA-IR index was highest in the group of patients with MASLD and HTN (4.67 mmol/L × mU/mL (95% CI 4.17; 4.50)) and was 1.4 times higher than in the group with isolated HTN (3.29 (95% CI 3.14; 3.43), p<0.001), and in the control group by 2.1 times (2.28 (95% CI 2.27; 2.52), p<0.001) (Figure 2).

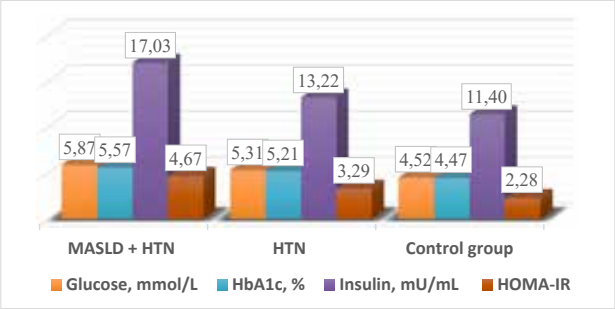


Fig. 1. Carbohydrate metabolism parameters in the examined patients

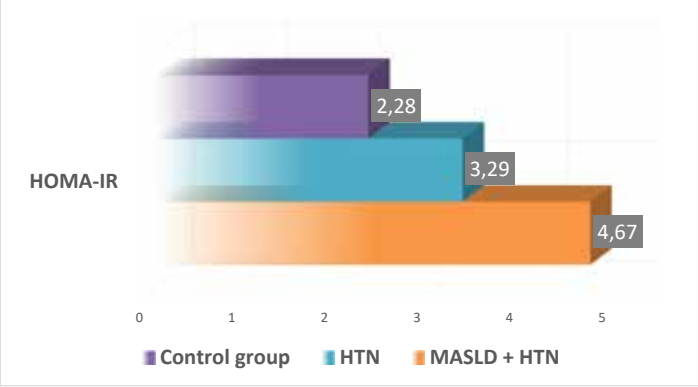


Fig. 2. HOMA-IR index values in the examined patients

According to the ultrasound steatometry results, among patients examined in the MASLD and HTN group, 20 patients had S1 steatosis, 27 had S2 steatosis, and 16 had S3 steatosis. Analysis of shear-wave elastography results revealed that among patients with MASLD and HTN, 4 had no signs of fibrosis, 20 had F1 fibrosis, 33 had F2 fibrosis, and 6 had F3 fibrosis. No cases of stage F4 liver fibrosis according to the METAVIR classification were detected in the patients enrolled in the study (Figure 3).

Patients with MASLD grade S1 of liver steatosis mostly had stage I HTN, while those with MASLD grade S3 had only stage II HTN (Table 5).

Among patients with MASLD grade S1, grade 1 HTN was predominant, whereas among patients with MASLD grade S3, only grade 2 HTN was observed. (Table 6).

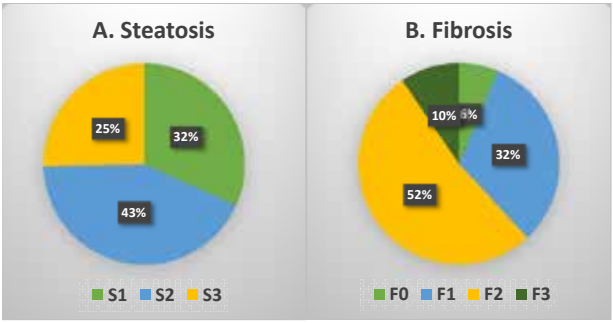


Fig. 3. Distribution of patients with MASLD and HTN by stage of hepatic steatosis (A) and fibrosis (B)

Table 5

Distribution of patients with MASLD and HTN by stages of hypertension and grade of liver steatosis

Grade of steatosis	Stage I HTN (n=21)		Stage II HTN (n=42)		Significance of the difference (based on the χ^2 criteria)	
	n	%	n	%		
S1	15	71,42	5	11,90	df=1 c	df=2 c
S2	6	28,57	21	50,00	df=1 c	
S3	0	0,00	16	38,10	df=1 c	

*Note: * – the difference is statistically significant between the stage I HTN group and the stage II HTN group ($p<0.05$).*

Table 6

Distribution of patients with MASLD and HTN by degree of hypertension and grade of liver steatosis

Grade of steatosis	1st degree HTN (n=19)		2nd degree HTN (n=44)		Significance of the difference (based on the χ^2 criteria)	
	n	%	n	%		
S1	16	84,21	4	9,09	df=1 c	df=2 c
S2	3	15,79	24	54,55	df=1 c	
S3	0	0,00	16	36,36	df=1 c	

*Note: * – the difference is statistically significant between the 1st degree HTN group and the 2nd degree HTN group (p<0.05).*

Stage 1 hypertension was more frequently detected in patients with stage F1 liver fibrosis, while stage 2 hypertension was more often recorded in patients with stage F2 fibrosis (Table 7).

Patients with MASLD and stage F1 liver fibrosis were more likely to have 1st degree hypertension, while patients with MASLD and stage F2 liver fibrosis were more likely to have 2hd degree hypertension (Table 8).

Thus, the progression of steatosis severity and fibrosis stage in MASLD was associated with an increase in both the grade and stage of arterial hypertension, which allows for considering MASLD as an unfavorable

Table 7

Distribution of patients with MASLD and HTN by stages of hypertension and liver fibrosis

Stage of fibrosis	Stage I HTN (n=21)		Stage II HTN (n=42)		Significance of the difference (based on the χ^2 criteria)	
	n	%	n	%		
F0	2	9,52	2	4,76	df=1 χ	df=3 χ
F1	14	66,66	6	14,29	df=1 χ	
F2	5	23,81	28	66,66	df=1 χ	
F3	0	0,00	6	14,29	df=1 χ	

*Note: * – the difference is statistically significant between the stage I HTN group and the stage II HTN group (p<0.05).*

Table 8

Distribution of patients with MASLD and HTN by degree of hypertension and grade of liver fibrosis

Stage of fibrosis	1st degree HTN (n=19)		2nd degree HTN (n=44)		Significance of the difference (based on the χ^2 criteria)	
	n	%	n	%		
F0	1	5,26	3	6,82	df=1 c	df=3 c
F1	13	68,42	7	15,91	df=1 c	
F2	5	26,32	28	63,64	df=1 c	
F3	0	0,00	6	13,64	df=1 c	

*Note: * – the difference is statistically significant between the 1st degree HTN group and the 2nd degree HTN group ($p<0.05$).*

prognostic factor for the progression of cardiometabolic risks in patients with arterial hypertension.

CONCLUSIONS

Based on the results obtained, it was found that the presence of MASLD in patients with arterial hypertension was associated with changes in the cardiometabolic phenotype of the disease.

The progression of the degree of steatosis and the stage of fibrosis in MASLD was accompanied by an increase in the grade and progression of the stage of arterial hypertension. In addition, the coexistence of MASLD and hypertension was associated with a more pronounced increase in body weight, an elevated risk of abdominal obesity, as well as worsening of carbohydrate and lipid metabolism disorders.

The data obtained confirm the pathophysiological role of MASLD in exacerbating cardiometabolic risks in patients with arterial hypertension.

SUMMARY

Metabolic dysfunction-associated steatotic liver disease (MASLD) is an important factor contributing to the severity of hypertension (HTN). The presence of MASLD is associated with increased insulin resistance, chronic systemic inflammation, endothelial dysfunction, and activation of neurohumoral mechanisms, which contribute to elevated blood pressure and impaired blood pressure control.

Patients with a combination of hypertension and MASLD are more likely to experience adverse cardiovascular events, myocardial remodeling, and an increased risk of atherosclerosis and cardiovascular mortality. Thus, MASLD should be viewed not merely as a liver disease but as a component of the systemic cardiometabolic continuum, which significantly influences the prognosis in patients with hypertension.

Aim: To evaluate the pathophysiological role of MASLD in the development of cardiometabolic risks in patients with hypertension.

Materials and methods. A total of 120 patients were examined: the study group consisted of 63 patients with MASLD against a background of HTN, and a comparison group of 57 patients with isolated HTN. The control group consisted of 20 healthy volunteers, randomized by sex and age.

Office systolic and diastolic blood pressure, BMI, and anthropometric parameters were measured; lipid and carbohydrate profiles were analyzed. Abdominal ultrasound, quantitative assessment of hepatic steatosis, and two-dimensional shear wave elastography (2D-SWE) for liver fibrosis assessment were performed. The results were statistically analyzed using the software packages.

Results. The mean systolic and diastolic blood pressure in patients with MASLD and HTN were significantly higher than in the other groups. The medians of BMI, waist circumference, hip circumference, and waist-to-hip ratio in patients from both groups were higher than those in the control group. The group with MASLD and HTN included a significantly higher number of patients with a BMI above 25 kg/m² (65.1% vs. 45.6% in patients with isolated hypertension, p=0.032) and individuals at risk for abdominal obesity based on waist circumference (41.3% vs. 21.1%, p=0,017). Almost all lipid profile parameters in both study groups showed statistically significant differences compared with the control values. When comparing the MASLD and HTN group with the isolated HTN group, statistically significant differences were found in the levels of total cholesterol, LDL-C, VLDL-C, HDL-C, and the atherogenic index.

The mean fasting glucose level in patients with MASLD and HTN was 5.87 mmol/L (95% CI 5.76–5.98) and was significantly higher than the corresponding value in the group with isolated HTN (5.31 mmol/L (95% CI 5.03; 5.57), p<0.001), but in both groups, blood glucose levels were higher relative to control values (4.52 mmol/L (95% CI 4.48; 4.61). Glycated hemoglobin and insulin levels showed similar correlations and were significantly higher in the group with MASLD and HTN. The HOMA-IR index was highest in the group of patients with MASLD and HTN and was 1.4 times higher than in the group with isolated HTN and in the control group by 2.1 times. Patients with MASLD

grade S1 of liver steatosis mostly had stage I and 1st degree of HTN, while those with MASLD grade S3 only grade 2 and stage II of HTN was observed. Similar patterns were observed when comparing the severity and stage of hypertension with the stage of liver fibrosis.

Conclusions. Based on the results obtained, it was found that the presence of MASLD in patients with arterial hypertension was associated with changes in the cardiometabolic phenotype of the disease.

The progression of the degree of steatosis and the stage of fibrosis in MASLD was accompanied by an increase in the grade and progression of the stage of arterial hypertension. In addition, the coexistence of MASLD and hypertension was associated with a more pronounced increase in body weight, an elevated risk of abdominal obesity, as well as worsening of carbohydrate and lipid metabolism disorders.

The data obtained confirm the pathophysiological role of MASLD in exacerbating cardiometabolic risks in patients with arterial hypertension.

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