Оригінальні дослідження

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Evaluation of the effectiveness of hepatoprotective therapy in patients with hypothyroidism and metabolically associated steatotic liver disease

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Abstract. The article presents the results of a prospective controlled clinical study aimed at evaluating the effectiveness of hepatoprotective therapy using ademethionine as part of a comprehensive treatment approach for patients with hypothyroidism and metabolic dysfunction-associated steatotic liver disease (MASLD). The study included 90 participants divided into three groups: the main group received combined therapy with levothyroxine and sublingual ademethionine; the comparison group received only thyroid hormone replacement therapy; and the control group consisted of apparently healthy individuals. All patients underwent a comprehensive clinical and laboratory examination before and after a 12-week course of treatment, including assessment of liver function parameters, thyroid status, insulin resistance index, and liver morphometric and structural characteristics based on ultrasound data. As a result of the combined treatment with levothyroxine and ademethionine in the main group, a statistically significant reduction was observed in the activity of liver enzymes – alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyltransferase (GGT), alkaline phosphatase (ALP), sorbitol dehydrogenase (SDH), and lactate dehydrogenase (LDH); improvement in thyroid profile (reduction in thyroid-stimulating hormone (TSH), increase in free thyroxine (FT4)); reduction in insulin resistance (decreased homeostatic model assessment of insulin resistance (HOMA-IR) index); improvement in ultrasound liver parameters (reduction in steatosis grade); as well as decreased body mass index and waist circumference (WC). In the comparison group, similar changes were less pronounced and mostly statistically insignificant. In the control group, all parameters remained stable. The obtained results confirm the clinical rationale for including ademethionine in treatment regimens for patients with hypothyroidism and MASLD to achieve more effective correction of hepatic and metabolic disorders, prevention of steatohepatitis progression, and reduction of the risk of fibrotic changes in hepatic parenchyma. The use of a comprehensive approach makes it possible to influence both the etiopathogenetic mechanisms and clinical manifestations of comorbid pathology, thereby increasing treatment efficacy and opening perspectives for further research in this field.

Keywords: hypothyroidism, metabolic dysfunction-associated steatotic liver disease, non-alcoholic fatty liver disease, hepatoprotective therapy, ademethionine, insulin resistance, thyroid hormones, steatosis, liver function.

Hypothyroidism is one of the most common endocrine disorders, accompanied by significant alterations in carbohydrate, lipid, and energy metabolism. A deficiency of thyroid hormones promotes the development of metabolic syndrome, dyslipidemia, and insulin resistance, which in turn creates prerequisites for the progression of MASLD a recently adopted term replacing non-alcoholic fatty liver disease [1].

MASLD is the most prevalent chronic liver disease worldwide, associated with triglyceride accumulation in hepatocytes, inflammatory infiltration, and, in some cases, the development of fibrosis or cirrhosis. Patients with hypothyroidism are at increased risk of steatohepatitis progression due to impaired fatty acid metabolism, decreased mitochondrial β-oxidation activity, and reduced insulin receptor expression [2].

Thyroid dysfunction affects not only hormone synthesis but also the transport of trace elements, the antioxidant system, and the activation of hepatocellular enzymes. Selenium deficiency, for instance, may exacerbate hepatocyte dysfunction through impaired glutathione peroxidase and deiodinase activity [3].

In addition to trace element imbalances, hypothyroidism is associated with decreased activity of the endogenous antioxidant system, contributing to the accumulation of free radicals and increased oxidative stress. This mechanism plays a key role in liver damage in metabolic syndrome. Melatonin use as an antioxidant reduces oxidative burden and improves liver function in patients with comorbid metabolic disorders [4].

Hepatoprotective therapy is regarded as a promising approach in the management of MASLD. Agents based on ademetionine (S-adenosyl-L-methionine) improve phospholipid metabolism, reduce pro-inflammatory cytokine activity, and promote hepatic cell regeneration. A systematic review demonstrated that hepatoprotectors effectively reduce ALT, AST levels, improve insulin resistance, and liver histology in non-alcoholic fatty liver disease [5].

The combination of L-thyroxine with hepatoprotective agents improves liver function markers, reduces steatosis and fibrosis in patients with hypothyroidism, and normalizes lipid profile and glycemic control [6]. However, these studies are limited and not conclusive due to variations in population selection, follow-up duration, and lack of stratification by the degree of thyroid dysfunction.

An analysis of clinical and laboratory indicators in patients with metabolic syndrome and type 2 diabetes revealed a significant increase in ALT, AST, ALP, LDH levels, and decreased cholinesterase activity in patients with NASH [7]. This indicates cytolytic syndrome, impaired detoxification, and decreased synthetic liver function.

Thus, despite existing evidence supporting the beneficial effects of hepatoprotectors in non-alcoholic fatty liver disease/MASLD, the role of hepatoprotective therapy in patients with combined pathology – hypothyroidism and MASLD – requires further investigation. The relevance of the issue lies in the need to develop an individualized treatment approach considering thyroid status, metabolic characteristics, and the extent of liver damage.

Aim. To evaluate the effectiveness of hepatoprotective therapy in patients with hypothyroidism and MASLD by analyzing changes in biochemical liver function parameters, degree of steatosis, and insulin resistance index before and after treatment with ademetionine.

Material and methods

This was a prospective, controlled, cohort study conducted to evaluate the effectiveness of hepatoprotective therapy using the sublingual form of ademetionine at a dose of 800 mg/day in patients with hypothyroidism combined with MASLD. The study involved a comparison of clinical-laboratory, biochemical, hormonal, metabolic, anthropometric, and ultrasound parameters before and after a 12-week course of treatment.

The research was conducted at the Ivano-Frankivsk Regional Clinical Hospital and the Bioelementology Center of the Ivano-Frankivsk National Medical University.

A total of 90 individuals were enrolled in the study and divided into three groups: the main group (n=40) included patients with hypothyroidism and MASLD who received levothyroxine in combination with hepatoprotective therapy (ademetionine); the comparison group (n=30) consisted of patients with the same pathology receiving only thyroid hormone replacement therapy; the control group (n=20) comprised apparently healthy individuals without endocrine or liver pathology.

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The study employed stratified randomization as part of the controlled clinical trial design. Patient allocation to treatment groups was performed using the «sequential numbering» method with the aid of a random number table.

All participants provided written informed consent to take part in the study.

Inclusion criteria were: age between 20 and 65 years, laboratory-confirmed hypothyroidism (elevated TSH with normal or reduced FT4), and a verified diagnosis of MASLD according to the European Association for the Study of the Liver (2020) criteria, confirmed by ultrasound evidence of hepatic steatosis along with at least one metabolic risk factor (abdominal obesity, insulin resistance, or dyslipidemia), and elevated levels of at least one liver enzyme (ALT, AST, GGT, ALP, or SDH).

Exclusion criteria included: hepatitis B or C, diabetes mellitus, active systemic or oncological diseases, glucocorticoid or hepatotoxic therapy in the previous 6 months, pregnancy, or lactation.

Hormonal parameters, including TSH and FT4, were measured using the enzyme-linked immunosorbent assay with DRG kits (USA). Biochemical indicators of liver function included the activity of ALT, AST, GGT, ALP, SDH, cholinesterase, and LDH, determined spectrophotometrically using reagents from Lachema (Czech Republic) and Philips Diagnostics (Germany). The metabolic profile was assessed by measuring fasting glucose levels (glucose oxidase method) and insulin (enzyme-linked immunosorbent assay), followed by the calculation of the HOMA-IR index using the formula: HOMA-IR = (fasting glucose × insulin) / 22.5. The index was interpreted as normal at HOMA-IR <2.77, moderate insulin resistance at 2.77–5.0, and severe resistance at >5.0.

Anthropometric data included body mass index and WC, measured according to the standards of the World Health Organization (1997) and the International Diabetes Federation (2005). Abdominal obesity was diagnosed when WC exceeded 80 cm in women and 94 cm in men. Liver ultrasound was performed to assess parenchymal echogenicity, structural integrity, and the degree of steatosis according to the following grading scale: grade 0 – no steatosis, grade 1 – mild (up to 33% involvement), grade 2 – moderate (34-66%), and grade 3 – severe (>66%).

The hepatoprotective therapy in the main group involved sublingual administration of ademetionine at a dose of 400 mg twice daily for 12 weeks. After completion of the treatment course, all patients underwent repeat evaluation of the specified parameters to assess therapy effectiveness.

Statistical analysis was performed using STATISTICA 10.0 software (StatSoft Inc., USA). Normality of distribution was tested using the Shapiro-Wilk test. For intergroup comparisons, the Student's t-test or the Mann–Whitney U test was used depending on the distribution, and the χ^2-test was used for categorical variables. Differences were considered statistically significant at $p<0.05$.

Results and discussion

Following the completion of the 12-week treatment course, a detailed comparative analysis of clinical and laboratory parameters was conducted among the three clinical groups: the main group (patients with hypothyroidism and MASLD receiving combined therapy with levothyroxine and ademetionine), the comparison group (patients with the same diagnosis receiving only thyroid hormone replacement therapy), and the control group of practically healthy individuals (**Table**).

Given the gastrointestinal changes observed in patients with hypothyroidism, our study prioritized the sublingual form of the hepatoprotective agent ademetionine. The sublingual formulation bypasses cytochrome P450 metabolism and avoids loss of active substance by being absorbed directly into the bloodstream. Since the drug is absorbed through the oral mucosa, it bypasses first-pass hepatic metabolism, which increases its bioavailability and enables a relatively rapid onset of action compared to oral administration [8].

According to the results of the liver function analysis, patients in the main group demonstrated a significant decrease in the activity of cytolytic enzymes, including ALT, AST, GGT, and SDH (Table). The reduction in the activity of these enzymes indicates a decrease in the degree of hepatocellular injury.

In addition, a significant reduction in LDH activity was recorded in patients of the main group (Table). This enzyme is a sensitive marker of cytolysis – its elevation reflects hepatocyte damage and the release of intracellular enzymes into the bloodstream; therefore, its reduction suggests a decrease in liver tissue destruction.

ALP activity, which also decreased in the main group, is a marker of cholestasis – impaired bile

Table. Comparative dynamics of biochemical parameters in patients with hypothyroidism and MASLD before and after treatment (M±SD)

Parameters	Group	n	Before treatment	After treatment	Δ, %	p	Cohen's d	95% CI
AST, U/L	Group I	40	65.38±10.10	41.31±2.89	-36.8%	<0.01	3.24	40.4-42.2
	Group II	30	39.23±2.25	38.62±1.80	-1.6%	<0.01	0.3	38.0-39.3
	HC	20	24.75±5.06	-	-	-	-	22.5-27.0
ALT, U/L	Group I	40	55.32±13.88	41.49±10.41	-25.0%	<0.01	1.13	38.3-44.7
	Group II	30	19.71±6.63	18.36±4.98	-6.8%	<0.01	0.23	16.6-20.1
	HC	20	19.14±4.80	-	-	-	-	17.0-21.2
GGT, U/L	Group I	40	103.81±28.59	70.89±30.20	-31.7%	<0.01	1.12	61.5-80.2
	Group II	30	63.59±11.80	43.73±11.63	-31.2%	<0.01	1.7	39.6-47.9
	HC	20	23.60±9.51	-	-	-	-	19.4-27.8
LDH, U/L	Group I	40	418.59±88.28	353.23±88.90	-15.6%	<0.01	0.74	325.7-380.8
	Group II	30	253.12±9.00	238.95±55.10	-5.6%	<0.01	0.36	219.2-258.7
	HC	20	184.74±33.77	-	-	-	-	169.9-199.5
SDH, U/L	Group I	40	3.29±0.88	1.99±1.01	-39.5%	<0.01	1.37	1.7-2.3
	Group II	30	2.14±0.57	1.33±0.61	-37.9%	<0.01	1.37	1.1-1.5
	HC	20	0.91±0.27	-	-	-	-	0.8-1.0
ALP, U/L	Group I	40	89.65±20.38	89.29±19.70	-0.4%	<0.01	0.02	83.2-95.4
	Group II	30	89.04±0.27	89.06±0.18	0.0%	<0.01	-0.09	89.0-89.1
	HC	20	88.51±28.56	-	-	-	-	76.0-101.0
HOMA-IR	Group I	40	4.58±1.05	3.47±0.63	-24.2%	<0.01	1.28	3.3-3.7
	Group II	30	3.10±0.71	2.68±0.56	-13.5%	<0.01	0.66	2.5-2.9
	HC	20	1.19±0.97	-	-	-	-	0.8-1.6

Note. Group I — patients with hypothyroidism + MASLD who received levothyroxine + ademetionine. Group II — patients with hypothyroidism + MASLD who received levothyroxine only. HC — healthy controls. Δ, % — percentage change in the parameter after treatment. p — statistical significance (p-value), <0.05 considered significant. Cohen's d — effect size (0.2 – small, 0.5 – medium, ≥0.8 – large effect). 95% CI — confidence interval for the post-treatment mean value.

outflow (Table). Its reduction during treatment suggests improvement in the functional state of the hepatobiliary system and a decrease in biliary stasis.

Cholinesterase activity, an indicator of the liver's protein-synthesizing function, showed a positive trend, although the changes were not statistically significant (p>0.05) (Table). Given that reduced cholinesterase activity is a characteristic sign of hepatodepressive syndrome, the observed upward trend may indicate the recovery of synthetic processes in the liver parenchyma.

In the comparison group, where patients received only levothyroxine, the enzyme activity dynamics were less pronounced and did not reach statistical significance (Table). These findings suggest the limited effectiveness of monotherapy in patients with hypothyroidism and MASLD.

Assessment of the metabolic profile under the influence of thyroid hormone replacement therapy and hepatoprotective treatment revealed a significant reduction in insulin resistance in the main group (Table), which may be attributed both to improved liver function and the potential effect of ademetionine on insulin sensitivity. In the comparison group, a downward trend in HOMA-IR was also observed, while in the control group, the index remained within the normal range.

Regarding thyroid status, improvement in hormone levels was observed in both hypothyroid patient groups following replacement therapy: in the main group, TSH levels decreased from 7.84±2.35 to 3.24±1.17 μIU/mL (p<0.01), and FT4 increased from 10.1±2.8 to 14.7±3.2 pmol/L (p<0.01); in the comparison group, TSH decreased from 7.63±2.42 to 4.85±1.69 μIU/mL (p<0.05), and FT4 increased

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from 10.4±2.6 to 13.2±2.9 pmol/L ($p<0.05$). In the control group, thyroid hormone levels remained stable throughout the observation period.

Analysis of the dynamics of morphometric and structural parameters of the liver based on ultrasound examination revealed a significant decrease in the degree of steatosis in the main group: the proportion of patients with grade 3 steatosis decreased from 32.5% to 10%, with grade 2 from 47.5% to 22.5%, while the proportion with grade 1 increased from 20% to 60% ($p<0.01$), indicating a positive trend in hepatic fat infiltration. In the comparison group, a similar trend toward improvement was observed, but it did not reach statistical significance ($p>0.05$). These findings may highlight the additional role of the hepatoprotective agent in improving the morphological condition of liver tissue (Table).

Additionally, changes in anthropometric parameters were recorded: body mass index in the main group decreased from 35.4±0.59 to 30.1± ±3.5 kg/m^2 ($p<0.05$), and WC decreased by 4.8± ±1.6 cm ($p<0.01$), indicating a beneficial effect of the therapy not only on liver and hormonal parameters but also on general metabolic disturbances. In the comparison group, these changes were less pronounced and not statistically significant (body mass index reduction by 0.8 kg/m^2; WC decrease of 2.1±1.3 cm; $p>0.05$). Anthropometric parameters remained stable in the control group.

The results of the conducted study demonstrated that the use of combined therapy with levothyroxine and sublingual ademetionine in patients with hypothyroidism and MASLD leads to a statistically significant improvement in biochemical, hormonal, morphological, and metabolic parameters compared to monotherapy with thyroid hormones. The obtained data are consistent with the findings of previous studies, which indicate the potential synergy between thyroid and hepatotropic therapies in combined pathology of the thyroid gland and liver [6]. This confirms the feasibility of including hepatoprotective therapy in the comprehensive treatment strategy for patients with hypothyroidism and MASLD.

Conclusions

Patients with hypothyroidism and MASLD exhibit significant impairment of liver function, characterized by elevated hepatic enzyme activity, insulin resistance, and thyroid dysfunction. The conducted study demonstrated that the administration of combined therapy with L-thyroxine and ademetionine leads to a significant improvement in biochemical markers of liver function (reduction in ALT, AST, GGT, LDH, ALP, and SDH activity, along with a trend toward increased cholinesterase), a decrease in the HOMA-IR index, normalization of TSH and FT4 levels, and a reduction in the degree of steatosis as evidenced by ultrasound findings. The use of hepatoprotective therapy as part of a comprehensive treatment regimen for these patients shows high clinical efficacy and is advisable for preventing the progression of steatohepatitis and fibrosis. The results are consistent with current concepts of MASLD pathogenesis and confirm the necessity of a multidisciplinary approach in managing patients with combined endocrine and hepatic pathology.

Future research perspectives

Further studies should focus on evaluating the long-term efficacy and safety of hepatoprotective therapy in combination with thyroid hormone replacement therapy in patients with hypothyroidism and MASLD. This includes involving larger cohorts, stratification by type of hypothyroidism, degree of steatosis, and disease duration. Additional imaging techniques (magnetic resonance imaging, fibroelastography), as well as assessment of systemic inflammation markers, fibrogenesis, and oxidative stress, should be employed to gain deeper insights into the pathophysiological mechanisms underlying the interplay between thyroid dysfunction and metabolically induced liver damage.

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Abbreviations

ALP – alkaline phosphatase
ALT – alanine aminotransferase
AST – aspartate aminotransferase
GGT – gamma-glutamyltransferase
HOMA-IR – homeostatic model assessment of insulin resistance
LDH – lactate dehydrogenase
MASLD – metabolic dysfunction-associated steatotic liver disease
SDH – sorbitol dehydrogenase
TSH – thyroid-stimulating hormone
FT4 – free thyroxine
WC – waist circumference

Оцінка ефективності гепатопротекторної терапії в пацієнтів із гіпотиреозом та метаболічно-асоційованою стеатотичною хворобою печінки

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Резюме. У статті представлено результати проспективного контрольованого клінічного дослідження, спрямованого на оцінку ефективності гепатопротекторної терапії із застосуванням адеметіоніну в складі комплексного лікування пацієнтів із гіпотиреозом та метаболічно-асоційованою стеатотичною хворобою печінки (МАСХП). До дослідження було залучено 90 осіб, які були розподілені на три групи: основна група отримувала комбінацію левотироксину та сублінгвального адеметіоніну; група порівняння — лише замісну терапію тиреоїдними гормонами; контрольна група складалася з практично здорових осіб. Усі учасники пройшли комплексне клініко-лабораторне обстеження до та після 12-тижневого курсу терапії, яке включало визначення біохімічних показників функції печінки, оцінку тиреоїдного статусу та індексу інсулінорезистентності. У пацієнтів основної групи відзначено статистично достовірне зниження активності ферментів печінки (аланінамінотрансферази, аспартатамінотрансферази, гамма-глютамілтрансферази, лужної фосфатази, сорбітолдегідрогенази та лактатдегідрогенази), покращення тиреоїдного профілю (зниження рівня тиреотропного гормону, підвищення рівня вільного тироксину), зменшення інсулінорезистентності (зниження індексу HOMA-IR), а також зменшення індексу маси тіла та окружності талії. У групі порівняння подібні зміни були менш вираженими і здебільшого статистично незначущими. У контрольній групі показники залишалися стабільними. Отримані результати обґрунтовують клінічну доцільність включення адеметіоніну до схем лікування пацієнтів із гіпотиреозом і МАСХП з метою покращення корекції гепатичних і метаболічних порушень та зниження ризику прогресування стеатогепатиту.

Ключові слова: гіпотиреоз, метаболічно-асоційована стеатотична хвороба печінки, неалкогольна жирова хвороба печінки, гепатопротекторна терапія, адеметіонін, інсулінорезистентність, тиреоїдні гормони, стеатоз, функція печінки.

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