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Реферати

ОСОБЕННОСТИ ВЕГЕТАТИВНОЙ И СОСУДИСТОЙ РЕГУЛЯЦИИ МОЗГОВОГО КРОВОТОКА У ПАЦІЕНТОВ С ГОЛОВНОЙ БОЛЬЮ НАПРЯЖЕНИЯ

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Целью работы было исследование клинической значимости и патогенетической важности сосудистых факторов, связанных с вегетативной дисфункцией, при головной боли напряжения (ГБН). Авторами показано патогенетическое значение вегетативного и сосудистого механизмов регуляции боли, их взаимодействия, а также их нарушения, что способствует дезадаптации различных уровней регуляции сосудистого тонуса. Проведенное исследование показало изменения вегетативного реагирования и обеспечения деятельности при ГБН, указывающие на неполноценность церебральных регуляторных механизмов у обследованного контингента пациентов, а также на проявления дезадаптации вегетативной системы при прогрессирования заболевания. Выявлено напряжение гуморального-метаболического звена регуляции, истощение резервов вазоконстрикции, в т.ч. церебрального венозного кровотока у 162 пациентов молодого возраста в зависимости от частоты возникновения ГБН, а также при ее переходе от эпизодической частой или хронической. Проведенные исследования показывают изменения вегетативной реактивности и обеспечения деятельности при ГБН, что указывает на неполноценность церебральных регуляторных механизмов, а также на возникающие проявления дезадаптации в течение прогрессирования заболевания. Показано, что патогенетические механизмы и источники возникновения ГБН и ее вариантов имеют мультифакторный характер с вовлечением элементов центральных и периферических вегетативных систем.

Ключевые слова: головная боль напряжения, вегетативная система, регуляция сосудистого тонуса, патогенетические механизмы, ультразвуковое сканирование сосудов головы

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THE PECULIARITIES OF BRAIN BLOODFLOW AUTONOMIC AND VASCULAR REGULATION IN PATIENTS WITH HEADACHE TENSION

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The purpose of the work was to investigate the clinical significance and pathogenetic importance of vascular factors associated with autonomic dysfunction in case of tension headache (THA). The authors showed the pathogenetic significance of the autonomic and vascular mechanisms of pain regulation, their interaction, as well as their disturbances which contributes to the disadaptation of various levels of vascular tone regulation. The study showed changes in the autonomic response and activity managing in case of THA indicating the inferiority of cerebral regulatory mechanisms in examined patients as well as the manifestations of maladaptation of the autonomic system with disease progression. The tension of the humoral-metabolic link of regulation, depletion of vasoconstriction reserves, including cerebral venous blood flow in 162 patients of a young age, depending on the incidence of THA, as well as during its transition from episodic frequent or chronic. Studies have shown changes in autonomic reactivity and maintenance of activity in THA, which indicates the inferiority of cerebral regulatory mechanisms, as well as the occurrence of maladaptation during the progression of the disease. It is shown that the pathogenetic mechanisms and sources of THA and its variants are multifactorial in nature with the involvement of elements of central and peripheral vegetative systems.

Key words: tension headache, vegetative system, vascular tone regulation, pathogenetic mechanisms, head vessels ultrasound investigation

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LIPID METABOLISM FEATURES IN PATIENTS WITH THE COMBINATION OF NON-ALCOHOLIC STEATOHEPATITIS AND ITS CORRECTION

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The purpose of the study was to estimate the effects of different treatment regimens on lipid metabolism in patients with non-alcoholic steatohepatitis (NASH) in combination with obesity (OB) and pathology of the biliary tract (BT) according to the 6-month dynamic observation. A dynamic study of 100 patients with NASH in combination with OB and BT pathology was performed, including the lipid metabolism indices study. It was found that lipid metabolism disorders occurred in all groups of patients, being characterized by an increase in triglycerides, cholesterol of low density lipoproteins, and disorders of metabolism and transport of the total cholesterol and reduced cholesterol levels of high density lipoproteins in comparison with apparently healthy individuals (from $p < 0.05$ to $p < 0.001$). Combined therapy including ursodeoxycholic acid and arginine glutamate in addition to the standard treatment at the comorbid NASH course can be considered as a promising treatment direction in this category of patients, which permits to achieve the restoration of the main lipid metabolism indices (from $p < 0.05$ to $p < 0.001$).

Key words: nonalcoholic steatohepatitis, obesity, lipid metabolism, ursodeoxycholic acid, arginine glutamate.

The work is a fragment of the research project "Cardiovascular risk, vascular pattern, markers of fibrosis and the adipose tissue metabolism in patients with cardiovascular diseases in the comorbidity conditions ", state registration No. 01117U004202.

Non-alcoholic fatty liver disease (NAFLD) is the most common disease in the internal medicine clinic, its prevalence being steadily increasing, particularly under the comorbid course conditions on the background of obesity (OB) and pathology of the biliary tract (BT) [5]. One of the main positions in the pathogenesis of the studied comorbid pathology is the lipid metabolism disorder [2, 3, 6]. Pathological changes in the liver tissue with lipid metabolism disorders and the dyslipidemia (DL) formation develop due to various mechanisms: the cytotoxic fatty acids accumulation; reduction of the S-adenosyl-methionine content in hepatocytes; activation of lipid peroxidation under the influence of free fatty acids; reduction in the rate of b-oxidation of these acids in the liver [4,7].

It is known that in patients with NAFLD the proatherrogenic serum lipid profile is observed, including the low level of high density lipoprotein cholesterol (HDLP cholesterol), high levels of triglycerides (TG) and that of low density lipoprotein cholesterol (LDLP cholesterol) [8,10]. It was established that accumulation of fat in the liver may be an independent factor in DL and suggests a direct pathogenetic chain: liver steatosis - DL - steatohepatitis [9, 14]. Taking into account the significant contribution of the lipid metabolism changes in the formation of NAFLD and OB, we have analyzed possible metabolic biomarkers that can significantly change the NAFLD comorbidity course.

The treatment of patients with NAFLD lies in the liver diseases therapy, as well as in the correction of concomitant metabolic conditions, such as DL, OB, insulin resistance, and type 2 diabetes [11-13, 15]. Today, there are no common standards for NAFLD treatment, particularly under the comorbidity conditions, at the level of the global medical practice.

Given the significant topicality of the NAFLD problem for modern medicine, a detailed study of lipid metabolism is of scientific interest and specific practical use in terms of developing new therapies aimed at inhibiting the progression of comorbid pathological conditions and improving the prognosis for these patients.

The purpose of the study was to estimate the effects of various treatment regimens on lipid metabolism in patients with non-alcoholic steatohepatitis (NASH) in combination with obesity (OB) and pathology of the biliary tract (BT) according to the 6-month dynamic observation.

Materials and methods. The total of 100 patients with NAFLD at the stage of non-alcoholic steatohepatitis (NASH) in combination with OB and BT pathology were examined: chronic non-calculous cholecystitis, chronic calculous cholecystitis, and patients after cholecystectomy, with liver steatosis signs detected during sonographic or morphological examination of liver biopsy. Among the patients, there were 40 (40 %) men and 60 (60 %) women. The mean age of the patients was (51.5±1.11) years. The control group consisted of 30 apparently healthy persons (AHP), comparable in age (mean age of 49.4±2.52 years) and sex (8 men, 22 women) with patients in the treatment group ($p>0.05$). Diagnosis of NASH and BT pathology (chronic non-calculous cholecystitis, chronic calculous cholecystitis, postcholecystectomy syndrome) was established on the basis of detecting the liver steatosis and its degree, moderate hepatomegaly, diffuse thickening of the gallbladder walls exceeding 3 mm and its deformation, the presence or absence of concretions, biochemical signs of cytolysis (hypertransaminasemia with a predominant increase of alanine aminotransferase content, elevated ratio alaninaminotransferase / aspartataminotransferase ratio value over 1.0) and / or cholestasis with negative serological markers of viral hepatitis B, C, D, and autoimmune hepatitis, absence of alcohol overuse and taking hepatotoxic drugs in patients by means of the ultrasound examination of abdominal cavity. The diagnosis of obesity was established in accordance with the global practical recommendations of the World Gastroenterology Organization 2013, WGO Global Guideline Obesity. Body mass index (BMI) was calculated using the Kettle formula. To characterize the lipid metabolism, the total cholesterol (TCH), TG, LDLP cholesterol, HDLP cholesterol were determined. In the blood, the TG content was studied using "Lachema" biotest kits, KFK-2 photoelectrocolorimeter, No. 8815175, 1988. (Zagorsk, Russia), the TCH content was determined by the Ilca method using the Stat Fax biochemical analyzer 1904, No. 19221090, 2008. (Awareness Technology Inc., USA), LDLP cholesterol and HDLP cholesterol levels were determined according to Burstein and Samaj, using KFK-2 photoelectrocolorimeter, No. 8815175, 1988 (Zagorsk, Russia).

To assess the efficacy of different NAFLD treatment regimens, all patients were divided into 3 groups by the adaptive randomization method (distribution of patients into groups initially by the equiprobable allocation, then - into a less numerous group or with equal probability). All the selected groups were statistically comparable in terms of age and sex of patients, the NASH activity degree, BMI obesity degree, and the concomitant BT pathology ($p>0.05$ for all comparisons between the groups). Patients of all groups, regardless of the treatment regimen, were prescribed lifestyle correction for the 6 months period, namely: diet, physical activity, work and rest.

Patients in group 1 (n = 34) received non-medicated treatment with the calorie intake reduction by 200 kcal every 2 weeks due to restriction of simple carbohydrates and animal origin fats and the addition of seasonal fresh vegetables and fruit to the diet. Aerobic physical activity was prescribed in the amount of 150 minutes or more per week, taking into account the physical condition and wishes of patients. Drug treatment included: standard treatment in accordance with the protocols for the provision of medical care at NASH and chronic cholecystitis (metabolic drugs with proven efficacy of L-carnitine, B group vitamins, myotropic spasmolytics: mebeverin hydrochloride or prokinetics - domperidone) for 30 days. Patients in group 2 (n = 33) received non-medicated treatment with reducing the caloric intake by 400 kcal every 2 weeks due to reducing the consumption of simple carbohydrates and animal origin fats with addition of seasonal vegetables and fruits to at least 3 servings per day. Aerobic physical activity was prescribed in the amount of 200 minutes or more per week. Drug treatment: standard treatment in combination with UDCA for 30 days in a dose of 15 mg/kg-1 d-1. Patients in group 3 (n = 33) received non-medicated treatment with a reduction in calorie intake by 600 kcal every 2 weeks, which is achieved by reducing the amount of carbohydrates and fats consumed with mandatory addition of the seasonal vegetable and fruit to the diet, at least 5 servings per day. Aerobic physical activity was prescribed in the amount of 250 and more minutes per week. Drug treatment: standard treatment and UDCA were combined with the use of arginine glutamate: the Glutargin drug (for 5 days: intravenous drip-feed by 5 ml of 40 % solution with 200 ml of physiological solution twice a day, the next 20 days glutargin tablets were assigned by 0.75 g three times a day). The lipid metabolism study was performed at the beginning and at the end of treatment in all patients, as well as 6 months after the treatment in 20 patients in each of the observation groups.

The primary data processing was carried out using the Microsoft Office Excel 2003® suite (No. 74017-641-9475201-57075) (Microsoft Corporation, USA) and the STATISTICA v.6.1 licensing software (AGAR909E415822FA). Taking into account the distribution law (Kolmogorov-Smirnov criterion corrected by Lilliefors), the quantitative indices are given in the form of mean and standard error of the mean (M±m). To compare the mean values between the groups and in the dynamics, the Student (t) and Mann-Whitney (U) criteria were used for independent samplings, the Student's (T) and Wilcoxon criteria (W) were applied for the dependent ones with the Bonferon correction for multiple comparison.

Results of the study and their discussion. Specific feature NAFLD in combination with OB and pathology of the BT is the abnormal lipid metabolism and progression of lipid and disorders. Analyzing indices of blood lipid spectrum in patients with comorbid NAFLD course before starting the treatment, it was established (table 1) that lipid metabolism disorders occurred in all groups of patients, being characterized by an increase in TG, LDLP cholesterol, disorders of metabolism and TCH transport and the low level of HDLP in comparison with apparently healthy persons (AHP) (from $p<0.05$ to $p<0.001$). Moreover, in most patients, high content of TCH coincided with high levels of LDLP cholesterol. The level of TCH increased in 1.3 times in patients of 2 group and 1.5 times in patients of 1 group and 3 group in comparison with AHP ($p <0.001$ for all comparisons). The highest level of LDLP cholesterol with an increase in 2.3 times was recorded in 1 group, among patients of 2 and 3 groups in 2.0 and 1.9 times relative to the control values ($p <0.001$ for all comparisons). TG scores also increased in all observation groups from 1.9 times in 1 and 3 groups to 2.1 times in patients of 2 group ($p <0.001$ for all comparisons). An increase in the level of TCH, TG, and LDLP cholesterol occurred against the background of HDLP cholesterol decrease in all observation groups relative to AHP (from $p <0.05$ to $p<0.001$). The lowest levels of HDLP cholesterol were in patients of 3 group with a decrease in 1.4 times ($p <0.001$).

Since all common lipoproteins are transport forms of cholesterol, the relative proportion of which in them all is different, the richest in cholesterol class of lipoproteins is LDLP cholesterol. Increase of their concentration in the patient’s blood indicates a disorder of the metabolism and cholesterol transport [7, 9]. Reducing the intensity of the reverse TCH transport from tissues to liver due to insufficient synthesis of HDLP cholesterol and a sufficient level of hypercholesterolemia in LDLP cholesterol and hypertriglyceridemia creates conditions not only for development and progression of NAFLD, but also for other disorders. Metabolic preconditions of the NAFLD’s progression in the context of comorbidity are dyslipidemia type II due to hypertriglyceridemia and reduced level of high density lipoprotein. During the follow-up study of lipid metabolism after the completion of the main treatment course in each group, it was found that the blood lipid profile dynamics in the examined patients depended on the particular treatment pattern used at taking prophylactic and therapeutic measures (table 1). To improve the efficiency of therapeutic and preventive measures to reduce the progression NAFLD in patients with OB and pathology of the BT to the standard treatment was proposed the addition of UDCA drugs of arginine glutamate. Thus, the TCH level after 30 days of treatment in group 3 patients reduced by 1.5 times compared to the values before treatment, by 1.6 times after treatment and in 6 months it reached normal values ($p<0.001$ for all

comparisons). In group 2 patients, the THC level reached normal values within 30 days after treatment, reducing by 1.3 times, and in 6 months after treatment - by 1.4 times compared to the baseline ($p<0.001$). In group 1, there only was a tendency to the TCH reduction by 1.2 times in 30 days after treatment ($p<0.05$). The values of TG in group 3 patients were the lowest (fig. 1) and statistically differed in 30 days and 6 months after treatment from the values of group 1 ($p<0.001$) and 6 months after treatment from the values of group 2 ($p<0.01$).

Table1

Lipid metabolism indices in patients with NASH with concomitant OB and BT pathology in the treatment dynamics (M ± m)

Index	AHP, n=30	Group	Before treatment	30 days after	6 months after (n=20)
TCH, mmol/L	5.23 ±0.17	1 (n=34)	7.62±0.39 ###	6.61±0.32 *###	6.70±0.35 ###
		2 (n=33)	6.83±0.43 ###	5.20±0.18 ***″″″	5.05±0.19 ***″″″
		3 (n=33)	7.86±0.53 ###	5.42±0.22 ***″″	4.85±0.17 ***″″″
TG, mmol/L	1.14 ±0.09	1 (n=34)	2.22±0.19 ###	1.88±0.13 ###	1.89±0.15 ###
		2 (n=33)	2.35±0.16 ###	1.49±0.09 ***#″	1.36±0.08 ***″″
		3 (n=33)	2.21±0.16 ###	1.27±0.08 ***″″″	1.03±0.08 ***″″″¶¶
LDLP cholesterol, un.	38.5 ±2.2	1 (n=34)	87.1±8.7 ###	78.8±6.7 ###	78.6±8.7 ###
		2 (n=33)	76.3±7.8 ###	54.8±3.7 **###″″	53.5±4.6 **##″″
		3 (n=33)	71.8±5.7 ###	48.4±2.2 ***##″″″	43.9±2.1 ***″″″¶
HDLP cholesrterol, mmol/L	1.62 ±0.05	1 (n=34)	1.44±0.06 #	1.37±0.06 #	1.34±0.08 #
		2 (n=33)	1.39±0.08 #	1.50±0.05	1.45±0.08
		3 (n=33)	1.15±0.07 ###″″	1.48±0.06 ***	1.57±0.07 ***″

Note: 1. * – p<0.05; ** – p<0.01; *** – p<0.001compared to the level before treatment; 2. # – p<0.05; ## – p<0.01; ### – p<0.001 compared to AHP; 3. ″ – p<0.05; ″″ – p<0.01; ″″″ – p<0.001 compared to group 1 patients; 4. ¶ – p<0.05; ¶¶ – p<0.01 compared to group 2 patients.

TG parameters after treatment in group 1 did not reach normal values and differed only from AHP ($p <0.001$). We observed the lowest level of LDLP cholesterol in group 3 (fig. 2) with the reduction by 1.6 times relative to the indices before treatment, which statistically distinguished these patients' indices from the similar values of patients in groups 2 and 1 within 6 months after treatment (from $p<0.05$ to $p<0.001$). The highest level of HDLP cholesterol 6 months after the treatment, with a 1.4-fold increase, was observed in group 3 patients that statistically distinguished their indices from the lowest similar level in group 1 ($p<0.05$).

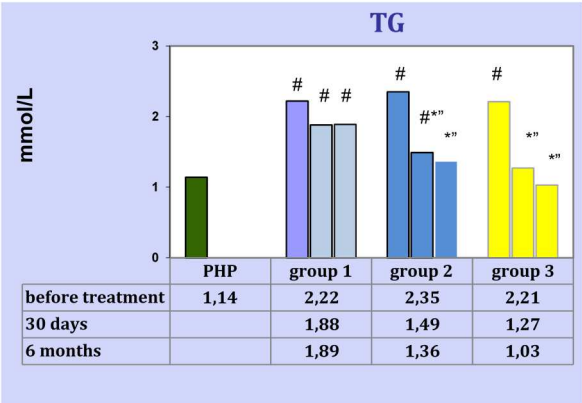

Fig. 1. - Dynamics of TG indices changes in patients with NASH in combination with OB and BT pathology.

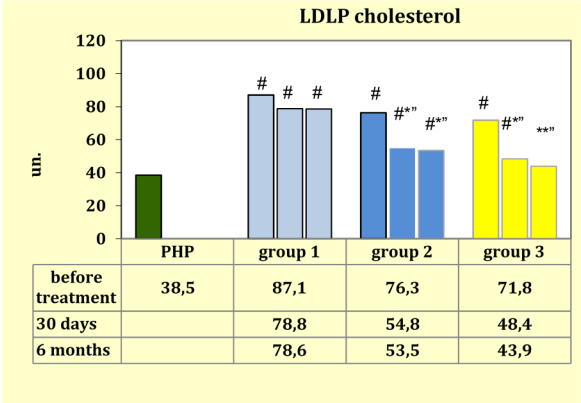

Fig. 2. - Dynamics of LDLP cholesterol changes in patients with NASH in combination with OB and BT pathology.

Thus, arginine glutamate, which was included into the treatment complex, can be attributed to the class of lipotropic substances with the lipid-correcting, hypolipidemic action, which was confirmed in the works of other authors [1]. The comparative intergroup analysis indicates a lack of the standard treatment efficacy and confirms the need to include more effective drugs into the complex of treatment measures that can have a greater impact on lipid metabolism. Thus, inclusion of the UDCA and arginine glutamate combination into the standard treatment of NASH-associated with OB and BT pathology is not only clinically positive but also contributes to the positive dynamics of lipid metabolism indices, i.e. the use of this combination is pathogenetically substantiated.

Combination medical therapy in patients with the inclusion to the standard treatment UDCA and arginine glutamate, can significantly improve the lipid profile, that can reduce the risk of progression of inflammatory and fibrosing reactions in patients with NAFLD in combination with OB.

The implementation of research results into practice will expand the possibilities of early diagnostics of NASH in patients with OB and pathology of the BT and enable the prediction of the disease course in terms of identifying possible risk factors.

The results of our study on changes in lipid metabolism indices and their correction possibility in comorbid patients with NASH do not contradict the results of other researchers [3-5, 8]. NASH has been associated with several cardiovascular risk factors including obesity, dyslipidemia. NASH is also characterized by atherogenic dyslipidemia and high-density lipoprotein dysfunction, which is convincingly proved in the works of other researchers [4, 8, 10].

In our opinion, it can be explained by the UDCA substance's capability to initiate the divergence of various biochemical processes in the body, the consequence of which is modulating a number of important physiological processes, particularly, the lipid metabolism correction. UDCA promotes the DL correction, especially in the context of administering other hepatoprotectors, namely, arginine glutamate. An important aspect, in our opinion, of the pharmacological properties of arginine glutamate is its probable effect on the TG and LDLP cholesterol levels correction in serum, which are important components of the DL formation, being a pathogenetic basis of steatohepatitis, particularly under the comorbidity conditions on the background of OB and BT pathology.

Conclusions

1. In patients with NASH, the combined course of the disease contributes to changes in lipid metabolism with an increase in TG, LDLP cholesterol, with the metabolism and TCH transport disorders against the background of a HDLP cholesterol reduction (from $p<0.05$ to $p<0.001$), which contributes to maintaining structural changes in the hepatobiliary system.

2. Combined therapy of patients with a comorbid NASH course can significantly improve the lipid metabolism, which can contribute to reducing risks of the disease progression. The results of the study indicate that the combined therapy with inclusion of standard UDCA and arginine glutamate in the comorbid NASH course can be considered a promising trend in this category of patients' treatment, permitting recovery of the main lipid metabolism indices (from $p<0.05$ to $p<0.001$).

Prospects for further research *lie in studying the indices of fibrosis in the dynamics of treating the patients with a comorbid NASH course.*

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Реферати

ОСОБЛИВОСТІ ЛІПІДНОГО ОБМІНУ У ХВОРИХ З КОМОРБІДНИМ ПЕРЕБІГОМ НЕАЛКОГОЛЬНОГО СТЕАТОГЕПАТИТУ ТА ЙОГО КОРЕКЦІЯ

Філіппова А.Ю.

Метою роботи було оцінити вплив різних схем комплексного лікування на показники ліпідного обміну у пацієнтів з неалкогольним стеатогепатитом (НАСГ) в поєднанні з ожирінням (ОЖ) та патологією біліарного тракту (БТ) за даними 6-місячного динамічного спостереження. Проведено динамічне обстеження 100 пацієнтів з НАСГ в поєднанні з ОЖ і патологією БТ, яке включало дослідження показників ліпідного обміну. Встановлено, що порушення ліпідного обміну відбувалися у всіх групах хворих, характеризувалися збільшенням тригліцеридів, холестерину ліпопротеїнів низької щільності і порушенням обміну і транспорту загального холестерину і зниженням рівня холестерину ліпопротеїнів високої щільності в порівнянні з практично здоровими особами (від $p<0,05$ до $p< 0,001$). Комбінована терапія з додаванням до стандартного лікування препаратів урсодезоксихолевої кислоти і аргініну глутамату за коморбідного перебігу НАСГ може розглядатися як перспективний напрямок в лікуванні цієї категорії пацієнтів, який дозволяє добитися нормалізації основних показників ліпідного обміну (від $p<0,05$ до $p<0,001$).

Ключові слова: неалкогольний стеатогепатит, ожиріння, ліпідний обмін, урсодезоксіхоліева кислота, аргініну глутамат.

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ОСОБЕННОСТИ ЛИПИДНОГО ОБМЕНА У ПАЦИЕНТОВ С КОМОРБИДНЫМ ТЕЧЕНИЕМ НЕАЛКОГОЛЬНОГО СТЕАТОГЕПАТИТА И ЕГО КОРРЕКЦИЯ

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Целью работы было оценить влияние разных схем комплексного лечения на показатели липидного обмена у пациентов с неалкогольным стеатогепатитом (НАСГ) в сочетании с ожирением (ОЖ) и патологией билиарного тракта (БТ) по данным 6-месячного динамического наблюдения. Проведено динамическое обследование 100 пациентов с НАСГ в сочетании с ОЖ и патологией БТ, которое включало исследование показателей липидного обмена. Установлено, что нарушения липидного обмена происходили во всех группах больных, характеризовались увеличением триглицеридов, холестерина липопротеинов низкой плотности и нарушением обмена и транспорта общего холестерина и снижением уровня холестерина липопротеинов высокой плотности в сравнении с практически здоровыми лицами (от $p<0,05$ до $p<0,001$). Комбинированная терапия с добавлением к стандартному лечению препаратов урсодезоксихолевой кислоты и аргинина глутамата при коморбидном течении НАСГ может рассматриваться как перспективное направление в лечении этой категории пациентов, которое позволяет добиться нормализации основных показателей липидного обмена (от $p<0,05$ до $p<0,001$).

Ключевые слова: неалкогольный стеатогепатит, ожирение, липидный обмен, урсодезоксихолиевая кислота, аргинина глутамат.

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НАУКОВЕ ОБҐРУНТУВАННЯ КОНВЕРСІЇ МЕТОДУ ОСТЕОСИНТЕЗУ ВОГНЕПАЛЬНИХ ПЕРЕЛОМІВ ДОВГИХ КІСТОК

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Метою дослідження було покращення результатів лікування вогнепальних переломів довгих кісток шляхом впровадження конверсії зовнішнього остеосинтезу на внутрішній. Масив дослідження становили 290 поранених з вогнепальними переломами довгих кісток кінцівок, які отримали ушкодження в 2014-2018 рр при проведенні антитерористичної операції. При поступленні на другий рівень у 118 (40,7%) поранених було проведено конверсію стержневого апарату зовнішньої фіксації на внутрішній остеосинтез, у 172 (59,3%) - апарат був остаточним методом лікування. Проведення конверсії остеосинтезу призводило до збільшення добрих функціональних результатів на 20,8% та зменшення відносної кількості незадовільних – на 11,9% ($p_\alpha<0,05$). Конверсія методу остеосинтезу є важливим методом профілактики контрактур крупних суглобів, який дозволяє знизити їх рівень у поранених з вогнепальними переломами з 33,0% до 6,5%.

Ключові слова: відновлення функції, вогнепальні переломи, конверсія методу, остеосинтез кісток, система медичного забезпечення.

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