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Content

MEDICAL SCIENCES | МЕДИЦИНСКИЕ НАУКИ

TECHNICAL SCIENCE | ТЕХНИЧЕСКИЕ НАУКИ

ющие дезинтоксивационные препараты (реосорбилакт) 400 мл, парацетамол 500 мг внутривенно. Следует подчеркнуть, что применеие инфузионной терапии не снижало терапевтического эффекта парентерального интерферона.

Отсутствие побочных реакций отмечено у 17 (32,6%) пациенток, и, следует отметить, что чаще отсутствие реакции отмечалось у женщин с высокими титрами вирусной инфекции (герпес, папилломавирус), что, возможно, является показателем иммуносупрессивного состояния либо снижения собственной продукции эндогенного интерферона у этих пациенток. Данное предположение может послужить основанием для дальнейших исследований.

Таким образом, проведенное клиническое исследование показало высокую эффективность применения α-интерферона-2b в комплексе терапии хламидиоза, мико- и уреаплазменной инфекции. В большинстве случаев, с применеием интерферонотерапии, мы наблюдали элиминацию папилломавируса, вируса простого герпеса и цитомегаловируса, значительное снижения бактериальной контаминации.

Выводы

1. Интерферонотерапию следует включать в комплекс терапии микст-инфекций гениталий, применение α-интерферона-2b при лечении хронических инфекций является эффективным и патогенетически обоснованным.
2. Курс терапии бактериальной инфекции включает применение α-интерферона-2b не менее 1млн в сутки 10 дней, хламидиоза 1млн.в сутки 10-20 дней, герпетической инфекции 1-2млн. в сутки 10-20 дней, папилломавирусной инфекции 3 млн в сутки 10 дней, при повторном положительном результате проводится курс в объеме 30 млн (3 млн в сутки 10 дней). После терапии интерфероном курс лечения может быть продолжен индукторами интерферона.
3. Лечение внутриклеточных форм инфекции, вирусов и вирусных ассоциаций требует назначения интерферонотерапии в качестве «заместительной» терапии недостатка естественных факторов иммунитета, способствующих элиминации данных возбудителей.
4. Назначение α-интерферона-2b (альфарекина) позволяет достичь полной ликвидации хламидийной инфекции, элиминации вируса папилломы человека.
5. В последующем, в периоде реабилитации рекомендуется проведение курсов иммуномодуляторов, вакцинации специфическими противовирусными вакцинами.

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THE CORRELATION BETWEEN METABOLIC, HEMODYNAMIC, STRUCTURAL AND FUNCTIONAL DISORDERS IN PATIENTS WITH NONALCOHOLIC FATTY LIVER DISEASE AND ARTERIAL HYPERTENSION

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ABSTRACT

It is analyzed the factors of progression of metabolic disorders in the liver and the changes in intracardiac hemodynamics in patients with nonalcoholic fatty liver disease in combination with hypertension. The predictors of progression of metabolic disorders in the liver in case of NAFLD associated with AH are the following: ALT activity; the level of total cholesterol; VLDL; HOMA-IR index; BMI and the presence of diastolic dysfunction due to disorders of diastolic relaxation; left ventricular systolic dysfunction with a decrease of contractile ability; left ventricular remodeling that contributes to the development and progression of liver fibrosis and increases the risk of cardiovascular complications.

Keywords: arterial hypertension, nonalcoholic fatty liver disease, metabolic disorders, the structural and functional changes in the myocardium.

The topicality of the problem of nonalcoholic fatty liver disease (NAFLD) and arterial hypertension (AH) comorbidity is caused by the high rates of their prevalence and progression of complications [1]. At present time, NAFLD is the most common liver disease and one of the leading factors in the development of chronic diseases. According to modern concepts, NAFLD is positioned as an independent risk factor for the development and progression of cardiovascular (CV) disease (CVD) and CVD, in turn, is one of the most important causes of morbidity and mortality in patients with NAFLD [7].

The fact that CV risk factors are more common in patients with NAFLD is not accidental, as «fatty liver» is responsible for the implementation of the metabolic components of cardiovascular risk, such as very low density lipoproteins (VLDL), C-reactive protein (CP) and the components of blood clotting. On the other hand, association of CVD with obesity indirectly affects the

progression of metabolic disorders in the liver by the secretory activity of adipose tissue [7].

The combination of NAFLD with hypertension, of course, has a mutually potentiating effect on the course of both diseases: NAFLD increases the rate of development of fibrotic changes in the liver parenchyma and the formation of portal hypertension, and at the same time an organ damage and the development of structural and functional disorders of the myocardium associated with the progression of hypertension are going on [3].

The joint pathogenetic mechanisms of AH and NAFLD include insulin resistance (IR) and compensatory hyperinsulinemia, which in turn stimulates the production of growth factors (platelet factor, insulin-like factor, fibroblast growth factor), which leads to proliferation of smooth muscle cells and fibroblasts, and in the end causes vasoconstriction, increases blood pressure (BP) and induces fibrosis [2,10].

The presence of AH in patients with NAFLD is an additional risk factor for the progression of dyslipidemia (DL), IR, deterioration of carbohydrate metabolism and liver function that are associated with excess body weight, which also worsens the course of the disease and contributes to the development of complications [5].

It was found that hypertension is an independent predictor of formation of portal fibrosis in patients with NAFLD, where the leading role belongs to angiotensin II and activation of transforming growth factor generation (TGF- β1) [8].

In recent years, certain data were obtained about the high risk of development of cardiometabolic disorders in the myocardium of patients with NAFLD and hypertension. The presence of pro-inflammatory status and atherogenic DL in these patients suggests a possible joint pathogenetic link between hepatic steatosis, DL and atherosclerosis. [6]

Therefore, a great scientific and practical interest is riveted to the study of structural and functional changes of the myocardium in patients with combined course of NAFLD and AH [4].

Currently, studies are being conducted that will allow to find out whether the accumulation of fat in the liver causes IR of myocardium, or maybe it causes myocardial steatosis and metabolic disorders of the heart with the assistance of humoral mechanisms which closely correlate with fat content in the liver.

Thus, fatty liver is closely related to IR, atherosclerosis and metabolic disorders. And hepatic steatosis is a predictor of CV events [3].

These mechanisms provide more evidence about the relationship between NAFLD and IR syndrome. IR provides the network, in which the clinical significance of progression of atherosclerotic vascular lesions is implemented. However, it is still not fully understood what metabolic events exactly contribute to the emergence of CV events against the background of NAFLD [4].

Despite considerable interest in this issue, the publications devoted to the study of factors affecting the progression of NAFLD in patients with AH, are contradictory. The uncertainty of the prognosis of NAFLD combined with AH regarding the impact of various metabolic disorders on the development and progression of hepatic steatosis and cardiac hemodynamics disorders dictates the need to find early markers and predictors, that are responsible for the initiation of an inflammatory process in the liver, as well as for the development of structural and functional changes in the myocardium.

The purpose of the research was to study the causative factors for progression of metabolic disorders in the liver and changes of intracardiac hemodynamics in patients with NAFLD combined with AH.

Materials and methods. The study involved 65 patients with NAFLD (37 men and 28 women). The mean age of patients was 56,4 ± 4,6 years. Depending on the nosology, patients were divided into 2 groups, representative by gender and age. The first group included patients with isolated NAFLD (n = 31), second (n = 34) - with combined course of NAFLD and AH of 2nd degree.

The patients were examined by a single program, which consisted of evaluating of physical findings (analysis of complaints, history taking, examination and evaluation of anthropometric indices, the calculation of body mass index (BMI) according to Quetelet formula). Laboratory studies included the study of the functional state of the liver (the activity of alanine aminotransferase (ALT), aspartate aminotransferase (AST), γ-glutamyl-transpeptidase (GGT) in the blood serum by conventional means); assessment of lipid spectrum of blood serum.

Serum insulin level was determined by immune-enzyme assay ELISA (DRG kits, USA). Assessment of IR level was performed using HOMA (homeostasis model assesment) with the calculation of IR index (HOMA-IR) using the formula: HOMA-IR = insulin, mcU / mL * glucose, mmol / L / 22.5.

The contents of total cholesterol (TC), triglycerides (TG), high density lipoproteins (HDL) in the blood serum were determined by enzymatic colorimetric method using «Human» kits (Germany); very low density lipoproteins (VLDL), low density lipoproteins (LDL) and atherogenic index (AI) were determined according to the conventional calculation method.

The concentration of fasting plasma glucose (FPG) was determined by glucose-oxidase method. The concentration of CRP in the blood serum was determined by enzyme immunoassay with the «DRG» set of reagents (USA).

Transthoracic echocardiography was performed by using the diagnostic system «Phillips IU» (USA) according to standard procedures in the M and B modes, using the recommendations of the American Society of Echocardiography to estimate the main indicators: end-systolic volume (ESV) of the left ventricle (LV), end-diastolic volume (EDV) of the LV, left ventricular end-systolic dimension (ESD), left ventricular end-diastolic dimension (EDD), left ventricular stroke volume (SV), ejection fraction (EF). LV diastolic function was assessed at the time of registration of transmitral diastolic flow in pulsed wave Doppler regimen.

The data of 20 healthy individuals (control group), comparable in age and gender with the examined patients, were used to evaluate the obtained results. Statistical analysis was performed by MS Excel v 7.0 using the Student's t test, with the minimum accepted level of significance $p < 0.05$. A correlation analysis with the calculation of Pearson's correlation coefficient and Spearman's rank correlation coefficient was used to determine the relationships between variables.

Results and discussion. The indexes of systolic (SBP) and diastolic (DBP) pressure in patients of groups 1 and 2 were the following, respectively: SBP, mm Hg – 126.9 ± 3.4 and 158.6 ± 4.8 (control group 118.4 ± 2.6 ($p < 0.05$); DBP, mm Hg – 80.2 ± 4.3 and 94.2 ± 3.4 (control group 73.4 ± 2.2 ($p < 0.05$).

The analysis of anthropometric parameters found a significant increase of BMI (in groups 1 and 2: 32.4 ± 1.8 kg / m² and 36.2 ± 4.8 kg / m², respectively; control group 22.3 ± 1.8 kg / m² ($p < 0.05$)). At the same time, BMI of patients of group 2 with

combined course of NAFLD and hypertension was significantly higher (p <0,05) than the one in patients with isolated NAFLD.

The analysis of the functional state of liver in patients of groups 1 and 2 was characterized by intensification of cytolytic processes in the liver, which were most expressed in patients with a combination of NAFLD and AH, that testified to the mutual aggravating character of metabolic disorders and high risk of formation of fibrotic changes in the liver (Table 1).

Table 1.

The peculiarities of blood serum biochemical indexes of examined patients (M± m)

Index, units of measurement	Control group (n=20)	NAFDL (n=31)	NAFDL+AH (n=34)
AST, mmol/L	0.36±0.05	0.47±0.06	0.64±0.7*/#
ALT, mmol/L	0.48±0.07	0.56±0.07	0.78±0.5*/#
GGTP, MU/L	43.7±12.7	57.1±15.2*	61.8±18.6*/#
HOMA-IR	1.6±1.3	3.6±1.8*	5.1±2.6*/#
TG, mmol/L	1.2±0.4	3.1±0.2*	4.3±0.6*/#
TC, mmol/L	4.2±0.8	6.2±0.4*	7.1±0.6*/#
HDL, mmol/L	1.3±0.06	1.1±0.05	0.72±0.03*/ #
LDL, mmol/L	3.0±0.21	3.36±0.3	4.1±0.4*/#
Thrombocytes, 109/L	226.0±23	220.0±24	200.0±44*/#
Fibronectin, mcg/mL	337.2±7.8	395.0±8.2*	484.0±9.8*/#
CRP, mg/L	2.85±0.21	6.45±0.28*	8.44±0.34*/#

Notes:
* - P <0.05 - significant differences in comparison with the control group;
# - P <0.05 - significant differences in comparison with patients with NAFLD

The level of CRP in blood serum exceeded the reference values in both groups of examined patients. The greatest increase was observed in patients with combined course of AH and NAFLD. A correlation of CRP with the following indexes was established: BMI (r = 0.47; p <0.001), the level of the FPG (r = 0.44; p <0.001), ALT (r = 0.49; p <0.001), TG (r = 0.37; p <0.04), HOMA-IR index (r = 0.41; p <0.001). The level of CRP should be considered as an additional prognostic criterion for increased risk of cardiovascular events in case of combined course of NAFLD and hypertension against the background of obesity.

The changes of hepatic transaminases also depended on BMI and were more pronounced in patients with combined course of NAFLD and hypertension.

A decrease of insulin sensitivity according to the HOMA-IR criterion was found in both groups of patients. Maximum values were observed in group 2 (p <0.05) and they positively correlated with BMI (r = 0.44; p <0.001), and the level of TG (r = 0.39; p <0.001).

The disorders, identified in our research, confirm the data that the pathological processes that occur in the liver in case of NAFLD lead to disruption of apoptosis and contribute to the development of systemic metabolic changes [9].

Changes of lipid metabolism were significantly more frequent in patients of group 2 with the combined course of NAFLD and AH, in comparison with patients of group 1 (82.3% and 46.20%, respectively; p <0.05). The level of total cholesterol in patients of group 2 was significantly higher than the one in comparison group and the control group as well (p <0.05) (Table 1).

An increase of concentration of total cholesterol and triglycerides in the group 1 directly depended on BMI (r = 0.61, p <0.05; r = 0.64, p <0.05, respectively), which is associated with the progression of metabolic disorders in the liver, in particular, with an excess intake of fats and carbohydrates in the liver, which are transformed into fatty acids that are substrates for the synthesis of TG. This confirms the theory regarding the influence of DL on the progression of NAFLD [10].

It was found that the decline of HDL in patients with NAFLD and hypertension was observed significantly more frequently and was more pronounced than in group 1 (groups 1 and 2: 20.0% and 54.2%; p <0.05, respectively).

Significant disorders of coagulation hemostasis were found in examined patients. Thus, patients with combined course of NAFLD and AH had 1.4-fold elevated serum levels of fibronectin compared with the control group (p <0.05).

Since fibronectin is a protein of extracellular matrix and a marker of severity of mesenchymal inflammation [5], these changes indicate the presence of the hypercoagulable syndrome in patients with NAFDL, and in combination with hypertension it contributes to further progression of apoptosis of the liver cells, strengthening of IR, development and deepening of hypoxia of cardiomyocytes, activation of free radical lipid oxidization [3].

There was a significant (p <0.05) reduction of platelets in the blood serum of patients with combined course of hypertension and NAFLD in comparison with the control group, which may indirectly indicate a high risk of fibrosis formation in these patients.

Thus, overweight, DL, IR, systemic inflammation and the presence of hypertension are the main predictors of progression of metabolic disorders in the liver and the deterioration of its functional state.

The analysis of echocardiographic parameters of patients of both groups showed the presence of structural, functional and hemodynamic disturbances, the severity of which was greater in patients with a combination of NAFLD and hypertension (Table 2).

A decrease of early (E) and late (A) diastolic filling velocities was going on in both groups of patients: (E, groups 1 and 2: 0.96 m / c and 0.74 m / c, respectively; control group 1.21 m / c (p <0.05)); (A, groups 1 and 2: 0.91 m / c and 0.78 m / c, respectively; control group 1.10 m / c (p <0.05)), while the E/A

more pronounced in patients with a combination of NAFLD and hypertension.

Table 2

Hemodynamic indexes of patients with NAFDL and combination of NAFDL and AH (M±m)

Indexes	Control group, (n=20)	NAFDL (n=31)	NAFDL+AH (n=34)
EDV, cm^3	118.1±5.4	131.2±4.7*	162.4±5.6*#
ESV, cm^3	47.4±2.3	58.3±5.2*	76.4±0.8*#
LV EDD, cm	4.32±0.03	4.89±0.06*	5.38±0.05*#
LV ESD, cm	3.26 ±0.02	3.92 ±0.02*	4.29±0.04*#
Stroke volume (SV), cm^3	75.4 ±1.36	78.9±1.38	96.6±0.76*#
Ejection fraction (EF),%	65.2±1.41	61.3±2.16	56.9±1.48*#

* – $p<0.05$ – significance of differences compared with the control group;
# – $p<0.05$ – significance of differences in comparison with NAFDL patients

The state of cardiohemodynamics in both groups of patients with NAFLD was significantly worse than the one of the control group and had certain differences between the patients with isolated NAFLD and the ones with combination of NAFLD and hypertension. The LV ESD index progressively increased in patients of groups 1 and 2 compared with the control group, and the difference between groups was significant ($p <0.05$). The increase of EDV occurred in both groups and was maximal in patients with NAFLD and hypertension, significantly exceeding the indexes of group 1 as well as the ones of the control group. Diastolic function was worse in patients with a combination of NAFLD and hypertension than in patients of group 1. Also, patients with a combination of NAFLD and hypertension had significantly higher indexes of ESV and EDV.

When evaluating the velocity and timing parameters of the systole and diastole we noted a decrease of transmitral flow maximum velocity during atrial systole phase: 1.2-fold in patients of group 1 and 1.3-fold in patients of group 2. Also, a 1.4-fold increase of the left atrium systole phase duration was detected in both groups of patients. Along with these, patients of group 2 had 1.2-fold shortening of mechanical diastole.

Confirmed LV dysfunction in patients with isolated NAFDL and combined course of NAFLD and hypertension was unidirectional and corresponded to diastolic dysfunction of type I.

Thus, the combination of NAFLD and hypertension causes structural, functional and hemodynamic changes, which are characterized by diastolic dysfunction due to the disorders of diastolic myocardial relaxation, as well as compensatory preclinical systolic dysfunction of LV with decreased contractile ability and increased end-diastolic left ventricular compliance.

Comparison of laboratory data, the parameters of intracardiac hemodynamics, structural and functional characteristics of the myocardium in patients with isolated course of NAFLD and NAFLD comorbid with hypertension demonstrates the highest activity of the inflammatory process in the liver in patients with combined course of NAFLD and hypertension, which is associated with the risk of development and progression of liver fibrosis. This effect is intensified under the influence of lipid and carbohydrate metabolism disorders, IR, hypercoagulation and systemic inflammation.

Conclusion.

1. The course of NAFLD associated with AH is accompanied by the atherogenic DL, IR, systemic inflammation, impaired carbohydrate metabolism that are connected with disorders of intracardiac hemodynamics, structural and functional changes in the myocardium.

2. The predictors of progression of metabolic disorders in the liver in case of NAFLD associated with AH are the following: ALT activity; the level of total cholesterol; VLDL; HOMA-IR index; BMI and the presence of diastolic dysfunction due to disorders of diastolic relaxation; left ventricular systolic dysfunction with a decrease of contractile ability; left ventricular remodeling that contributes to the development and progression of liver fibrosis and increases the risk of cardiovascular complications.

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ELENIUM AND FREE RADICAL OXIDATION STATUS OF BLOOD IN PATIENTS WITH ACUTE-ON-CHRONIC LIVER FAILURE OF ALCOHOLIC ETIOLOGY

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ABSTRACT

The selenium concentration and free radical status in blood serum were studied in patients with acute-on-chronic liver failure of alcoholic etiology. This patient group had decreased selenium in blood serum (the average value 75,0±20,1 mcg/l). With the increase of staging of the pathological process, the depth of selenium deficiency in patients increases. Plasma concentration of selenium remained unchanged after the treatment. Decrease of selenium is characterized by activation of free radical processes in blood serum of these patients.

Keywords: acute-on-chronic liver failure, alcoholic liver disease, selenium, free radical oxidation

Acute-on-chronic liver failure (ACLF) is a kind of pathology that combines acute hepatitis on a background of alcoholic cirrhosis [1]. According to a prospective multi-center study, the active alcohol intake in the last 3 months prior to the aggravation is the most common reason that leads to an exacerbation of chronic liver failure (42,9%). In addition, alcoholic cirrhosis (58,4%) observed in these patients more often than cirrhosis of viral etiology (14,8%) and mixed (9,3%) [5].

In the pathogenesis of the acute is the basic process of free-radical oxidation, in contrast to which there antioxidant antiradical system, the main elements of which are selenium-containing enzymes [8, 9].

Prolonged duration of alcoholic liver disease and a high probability of death determine this pathology as a socially significant disease [6]. Investigation of the mechanisms of change of free radical and selenium status would make it possible to introduce additional diagnostic criteria of the disease and ways of their medical correction [7].

The aim of the study is to evaluate the selenium and free radical statuses of blood serum of patients with acute-on-chronic liver failure of alcoholic etiology for the optimization of the diagnostic of this nosology.

Materials and methods

We examined 60 patients aged 23 to 74 years (mean age 48,8±11,2 years) with acute-on-chronic liver failure of alcoholic etiology 1-3rd stages, exhibited according to the criteria of the American Association for the Study of Liver Diseases (AASLD). After clinical and laboratory examination, all patients received standard basic therapy. Re-examination of patients was carried out after 15 days. Exclusion criteria were patients with chronic viral hepatitis and decompensated chronic diseases of other etiologies.

Comparison group consisted of 21 men (mean age - 47,6±12,5 years) without clinical signs of pathology of the hepatobiliary system, living together with patients and the same feed with them, to eliminate differences in exogenous selenium admission. We used the CAGE questionnaire and Alcohol Use Disorders Identification Test for detecting chronic alcohol intoxication.

The selenium content in the blood serum was adjusted by fluorimetric method on N.A. Golubkina (1995) [3]. Chemiluminescence (CML) method was used for integral assessment of free radical oxidation of blood serum. We studied the spontaneous and induced Fe CML, identifying [2]: lightsum for 1 minute spontaneous CML (S_{sp}); lightsum (S_{ind-1}) for 2 minutes after the «fast» flash; a maximum of «fast» flashes (h) induced by CML. We analyzed the kinetics of CML, initiated H_20_2 in the presence of luminol [2], by the following parameters: lightsum for 2 minutes CML (S_{ind-2}). the maximum of luminescence (H). The intensity of the CML was measured in relative units. Registration CML was performed on the fluorescent spectrometer LS 50B («Perkin Elmer»).

The data were processed by Microsoft Office Excel 2010 software, Statistica 6.03. Calculate: mean (M), standard deviation (SD), confidence limits (CL). Statistical significance of differences between mean values was estimated by Mann-Whitney test. Determining the link between non-parametric values was assessed by Spearman correlation analysis. The critical level of significance in hypothesis was $p<0,05$.

Results and discussion. Average serum selenium level in patients with acute-on-chronic liver failure of alcoholic etiology was 75,5±20,1 mcg/l and was lowered as compared with the comparison group 105,8±13,6 mcg/l ($p<0,00001$). The distribution of provision of selenium in the examined patients is displayed in Figure 1.