

отяжким перебігом НЦД за ГіперТТ у 66,7% випадків, у 23,3% випадків пацієнтів з НЦД за ГіпоТТ НЦД та у 46,2% випадків у пацієнтів з КТ НЦД.

У хворих на ХНХ молодого віку встановлено загострення проявів НЦД у 87,5% випадків, серед яких переважає (у 60,0%) ГіпоТТ, у 11,4% спостерігається ГіперТТ і у 28,6% - КТ. У хворих на ХНХ зрілого віку встановлено загострення НЦД у 94,7% випадків, серед яких ГіпоТТ спостерігається у 25,0%, а переважають ГіперТТ (30,6%) та КТ (44,4%).

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ЗМІНИ ПОКАЗНИКІВ НІТРОЗИТИВНОГО ТА ОКСИДАТИВНОГО СТРЕСУ У ХВОРИХ НА ХРОНІЧНИЙ НЕКАМЕНЕВИЙ ХОЛЕЦИСТИТ ЗАЛЕЖНО ВІД ТИПУ СУПРОВІДНОЇ НЕЙРОЦИРКУЛЯТОРНОЇ ДИСТОНІЇ

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CHANGES OF NITROSATIVE AND OXIDATIVE STRESS MARKERS IN PATIENTS WITH CHRONIC ACALCULOUS CHOLECYSTITIS DEPENDING ON THE TYPE OF ACCOMPANYING NEUROCIRCULATORY DYSTONIA

Summary.

In the research paper there are data about intensity of oxidative and nitrosative stress in patients with chronic acalculous cholecystitis depending on the type of accompanying neurocirculatory dystonia. In patients with chronic acalculous cholecystitis and comorbid neurocirculatory dystonia of hypertensive and cardiac type, the intensity of oxidative stress is dominating and there are significant imbalance of antioxidant defense factors. In majority of the patients with chronic acalculous cholecystitis and neurocirculatory dystonia of hypertensive type, on the background of mild amplification of oxidative stress intensity, the nitrosative stress intensity is higher because of induced nitric oxide (NO) synthase activation and hyperproduction of nitrogen monoxide. The latter contributes to the development of peripheral venous dilatation.

Анотація.

В роботі наведені дані щодо інтенсивності оксидативного та нітрозитивного стресу в хворих на хронічний некаменевий холецистит залежно від типу супровідної нейроциркуляторної дистонії. У пацієнтів з хронічним некаменевим холециститом із супровідною нейроциркуляторною дистонією за гіпертонічним та кардіальним типом переважають інтенсивність оксидативного стресу та істотний дисбаланс активності факторів протиоксидантного захисту. У більшості пацієнтів з хронічним некаменевим холециститом із супровідною нейроциркуляторною дистонією за гіпотонічним типом, на тлі помірного підсилення інтенсивності оксидативного стресу, переважає інтенсивність нітрозитивного стресу внаслідок активації прозапальної індукційної NO-синтази, гіперпродукції монооксиду нітрогену, який сприяє розвитку периферичної венозної вазодилатації.

Key words: neurocirculatory dystonia, chronic acalculous cholecystitis, oxidative stress, nitrosative stress

Ключові слова: Нейроциркуляторна дистонія, хронічний некалькульозний холецистит, оксидативний стрес, нітрозитивний стрес

In recent years, there has been a significant increase in the frequency and prevalence of functional diseases of the cardiovascular system (CVS), including vegetative-vascular dystonia (VVD) (neurocirculatory dystonia (NCD), somatogenous autonomic dysfunction) [1, 6]. According to the literature, the incidence of this disease in the population is on average 24-25% [4], which is due to many factors, among which the most important is to increase the level of emotional stress, dysgormonosis, environmental degradation in the region prozhyvannya. NCD therapist - an independent functional disease with a benign course and various disorders of the CVS due to disorders of neurohumoral regulation of vascular tone [2]. Among the contributing factors are hereditary and constitutional features, being in the period of hormonal adjustment (puberty, menopause), lifestyle [4]. However, the widespread neurological doctrine of IRR and NCD has been a factor that causes objective difficulties in diagnosis, determination of pathogenetic mechanisms, treatment and rehabilitation of a significant number of patients [8]. The aim of our study was to elucidate the pathogenetic role of the intensity of lipid peroxidation, nitrosive stress and the state of certain factors of the antioxidant defense system (ANS) in the development and progression of neurocirculatory dystonia (NCD) in patients with chronic non-stone cholecystitis.

Material and methods. A survey of 78 patients at HNH in the phase of exacerbation of the accompanying NCD. The mean age of patients was 31 ± 5.8 years. Depending on the form of NCD patients at HNH were divided into three groups: the first (15 persons) - patients on NCD by HNH and hypertensive type (HiperTT); second - patient and to HNH of NCD on hypotensive type (HipoTT) (30 persons) and third - patients at HNH of NCD for cardiac type (CT) (26 persons). The control group consisted of 30 healthy individuals (30) of the appropriate age. The role of socio-economic factors that may influence the occurrence and course of NCDs was determined in all examined patients. Assessment of complaints and the objective condition of patients was performed daily. Laboratory-instrumental examination was performed on admission to the hospital (before treatment) and on discharge from the hospital. The intensity of OMB in blood serum was determined by the method of Dubinina OE and et al. in the modification of IF Meschishena [4]. The principle of the method is based

on the reaction of interaction of oxidized amino acid residues of proteins with 2,4-dinitrophenylhydrazine with the formation of aldehyde and ketondinitrophenylhydrazones of basic and neutral nature (AKDFG OX and NH). The content in the blood of molecular products of LPO - isolated double bonds (IPZ) in compounds, diene conjugates (DC), ketodienes and conjugated trienes (KST) was determined by I.A. Volchehorskym and et al. The content of malonic aldehyde (MA) in plasma and erythrocytes - with YA Vladimirov, AI Archakov [3]. BMI century in the blood of reduced glutathione was determined tytratsynym method for AV Travina in the modification of IF Meshchishena, I.V. Petrova [4]. The activity of enzymes of the POS system: glutathione peroxidase was determined by IF Meschisheni m, catalases - for M.A. Koroliuk and al. [4] The activity of enzymes was calculated per 1 g of Hb [4]. And Mr. tensyvniist nitrozytyvnoho stress by studying blood levels of stable metabolites monoxide nitrogen (NO) (nitrites, nitrates) method L. C. Green with et al. The activity of inducible NO-syntazy (and NOS) (DRG) by ELISA analysis. Statistical processing of research results was carried out using parametric methods of variation statistics

Results and their discussion. As data, presented in Table. 1 in all groups surveyed patients at HNH determined activation process POL, more significant - in the group of patients on NCD for HiperTT, because content in plasma of blood and red blood cells most toxic of the end product of lipid peroxidation, MA exceed control indices respectively in 2.0 times and at 30.0% ($p < 0.05$). In the group of patients to CT for NCD also registered likely ($p < 0.05$) increase in content in plasma and erythrocytes MA (Table. 1), and in 1.7% to 24.0 ($p < 0.05$) respectively in comparable to PZO. A similar orientation changes MA registered in the group of patients in HipoTT, with this figure exceeded the control respectively 1.5 times and at 18.6% ($p < 0.05$) (Table 1). Uncontrolled significant increase in content in blood and red blood cells end product of lipid peroxidation shows of decompensation processes in ilnoradykalnoho damage cell membranes, and first of all, endothelial, and the growth of the intensity of metabolic intoxication, which has some pathogenetic importance in the development and progression of NCD in patients with acute exacerbation of HNH.

Table 1

Indicators of intensity of lipid peroxidation, oxidative modification of proteins, nitrosive stress and antioxidant protection factors in patients with chronic non-stone cholecystitis depending on the type of concomitant neuro-circulatory dystonia

Indexes	PZO, n=30	HNH with NCD for Hyper TT, n= 15	HNH with NCD for Hypo TT, n= 30	CKD with NCD on CT, n= 26
MA in plasma, $\mu\text{mol} / \text{L}$	2.53 \pm 0.072	4.99 \pm 0.033 *	3.72 \pm 0.025 * / **	4.35 \pm 0.037 * / ** / ***
MA in Er., Mmol / l	8.09 \pm 0.138	10.52 \pm 0.841 *	9.61 \pm 0.321 *	10.03 \pm 0.244 *
IPZ, E220 / ml. to the moat	2.64 \pm 0.031	5.93 \pm 0.071 *	4.48 \pm 0.092 * / **	5.27 \pm 0.078 * / ** / ***
DC, E232 / ml. to the moat	1.46 \pm 0.015	2.95 \pm 0.019 *	2.17 \pm 0.063 * / **	3.43 \pm 0.041 * / ** / ***
KST, E220 / ml. to the moat	0.81 \pm 0.015	1.52 \pm 0.021 *	1.43 \pm 0.017 * / **	1.57 \pm 0.042 * / ** / ***
AKDNFHOH, o.od. h / L protein	1.37 \pm 0.023	3.07 \pm 0.044 *	2.41 \pm 0.050 * / **	3.28 \pm 0.041 * / ** / ***
AKDNFGNH, mmol / g b protein	12.14 \pm 0.137	19.26 \pm 0.826 *	17.73 \pm 0.471 *	22.04 \pm 0.587 * / ** / ***
GV, $\mu\text{mol} / \text{l}$	0.93 \pm 0.013	0.71 \pm 0.008 *	0.53 \pm 0.004 * / **	0.67 \pm 0.008 * / ** / ***
GP, nmol GV / min. \times 1g. Hb	156.79 \pm 1.821	125.14 \pm 5.315 *	180.42 \pm 5.742 * / **	147.56 \pm 5.287 ** / ***
Catalase, mmol / 1 min. \times 1g Hb.	15.52 \pm 0.094	18.17 \pm 0.073 *	10.45 \pm 0.812 * / **	24.34 \pm 1.022 * / ** / ***
NO, $\mu\text{mol} / \text{l}$	14.57 \pm 0.475	10.62 \pm 0.632 *	18.99 \pm 0.908 * / **	7.53 \pm 0.543 * / ** / ***
iNOS, nmol / min	0.50 \pm 0.014	0.74 \pm 0.020 *	1.35 \pm 0.027 * / **	0.91 \pm 0.012 * / ** / ***

Note: * - the difference is probable in comparison with the indicator in PZO ($p < 0,05$);

** - the difference is significant in comparison with the rate in patients with NCDs for HyperTT ($p < 0,05$);

*** - the difference is significant in comparison with the rate in patients with NCDs for HypoTT ($p < 0,05$).

However, we have found a likely increase in content in the blood of the intermediate products of lipid peroxidation - IPZ in BC ix groups observation that exceeded the control performance in the 1st group - 2.2 times ($p < 0,05$), in the 2nd group - in 1.8 times ($p < 0,05$), in the 3rd group - 1.9 times ($p < 0,05$) with the presence of a probable intergroup difference ($p < 0,05$). With similar intensity the content in blood of other intermediate products of FLOOR increased: DK, KST (tab. 1): in the 1st group - accordingly in 2,0 and 1,9 times ($p < 0,05$), patients of the 2nd group - 1.5 and 1.8 times ($p < 0,05$), in patients of the 3rd group - 2.3 and 1.9 times ($p < 0,05$). It is obvious that the maximum value indicators, which point to the intensity of oxidative stress, established in patients with HNH of HiperTT and CT NCD and the minimum - in patients at HNH of HipoTT NCD, but in patients Su ix groups observation found significant the likely increase in intensity of lipid peroxidation in comparable to healthy individuals.

It should be noted, that the free radical effects, in terms of aggravation HNH, touching not only structural li n idiv membranes, but also protein substrates. In particular, in all observation groups there was a probable increase in the content of final products (OMB) in the blood (Table 1): in patients of the 1st group - 2.2 times and 58.6%, respectively ($p < 0,05$), patients of the 2nd group - 1.8 times and 46.0% ($p < 0,05$), patients of the 3rd group - 2.4 times and 81.5% ($p < 0,05$) in compared with the control group. Thus, the maximum intensity of OMB processes was

registered in patients with CT NCD, which is an important prerequisite for endothelial damage, including coronary arteries and the subsequent development of coronary heart disease (CHD) in these patients.

When assessing the state of the POZ system revealed the following changes (Table 1). First of all, it should be noted significantly reduced BMI century in blood GW in all clinical groups observation that in patients of the 1st group was 23.7% ($p < 0,05$), 2nd group - 43.0% ($p < 0,05$), group 3 - 28.0% ($p < 0,05$) compared with 30. That is, a probable insufficiency of the regenerated glutathione system - a universal redox system of erythrocytes, which resists free radical influences, has been established, which has led to a significant intensification of LPO and OMB in these patients. Paradoxically, there is the fact that the maximum degree of suppression systems GV detected in patients at HNH of HipoTT NCD, in which there was the slightest degree of intensity of lipid peroxidation and OMB. However, this fact probably will explain the results of the next stage of our lit. idzhennya. Significant changes were also detected in the functioning of the glutathione- dependent enzyme glutathione peroxidase depending on the clinical variant of the concomitant disease (Table 1). Thus, in the 2nd group observed maximum claim idvyschennya activity GP - 14.9% ($p < 0,05$) compared with the USO. In patients of the 1st group of observation the activity of the enzyme is probably less than the activity of GP in PZO - by 20.0% ($p < 0,05$), and in patients of the 3rd

group the changes were incredible ($p > 0.05$) Table 1). Activation of glutathione system enzymes can be considered as a compensatory mechanism in response to the intensification of LPS processes. But this is not enough to claim idtrymannya content GW at normal levels. One explanation for this may be the lack of necessary for the restoration of glutathione number of recovered equivalents, but also increased the need for it on the conditions of growing endotoxemia in conditions of inflammation in the HNH. On tension system EOP under conditions of aggravation HNH also St. idchyt increase in the activity of catalase red blood cells in patients with HiperTT and CT NCD, respectively 17.2% and 56.8% ($p < 0.05$) compared with the USO, both in patients with HipoTT NCD activity of catalase, was suppressed in% 32.6 ($p < 0.05$) in comparison with control. Compensatory activation of GP and inhibition of catalase activity in patients with CKD and HipoTT explain the maximum degree of HF depletion in this category of patients. The results of the conducted intensity nitrozytyvnoho stress showed that in 100 % of surveyed patients at HNH with accompanying NCD was found significant changes, but changes direction differs depending on the form NCD (tab. 2). In particular, in patients of the 1st and 3rd groups there was a probable decrease in the content of NO metabolites in the blood - respectively not 27.1% and 48.3% ($p < 0.05$), which indicates the development of endothelial dysfunction in these patients. (ED) in comparison with the indicator in ZO with the presence of a probable intergroup difference ($p < 0.05$). Minimal values of the indicator (decrease by almost 50%) were registered in patients with CT NCD, which indicates a significant risk factor for further progression of this form of NCD in coronary heart disease. At the same time, as indicated by the data set out in table. 2, in patients with a combined course of CKD and NCD there was a significant increase in NO in the blood (by 30.3%, $p < 0.05$) compared with group 30, which was significantly different from the values in the comparison groups ($p < 0.05$). The correlation analysis revealed a high degree of inverse correlation ($r = -0.781$, $p < 0.05$) between blood pressure and NO in the blood. It was also found that the intensity nitrozytyvnoho stress in this group of patients increased with increasing intensity of inflammation in the gallbladder ($r = 0.745$, $p < 0.05$ between the content of NO in blood and sialic acids in bile) and degree of dyskinesia biliary tract by biliary type.

Pathological hyperproduction of NO by leukocytes due to inflammation of the gallbladder wall (GV) and biliary tract (GVH) contributed to the activation of nitrosive stress and the development of generalized venous dilatation, which led to a decrease in peripheral vascular resistance and progression of NCDs to hypoTT. At the same time, NO overproduction can be regarded as a compensatory response of the body to the development of hypertensive, hyperkinetic dyskinesia of the gastrointestinal tract and LVH in order to eliminate it, ie to restore the normal contractile capacity of the LMW and sphincter tone of the LVH.

Changes in the content of NO metabolites in the blood serum of patients with CKD with concomitant NCDs by HipoTT occurred due to induction of inflammation and NOS. According to the obtained data (Table 1), the activity and NOS in patients with CKD of all groups was significantly increased: in patients of the 1st group exceeded the rate in PZO 1.5 times ($p < 0.05$), the 2nd group - 2.7 times ($p < 0.05$), the 3rd group - 1.8 times ($p < 0.05$) with the presence of a probable intergroup difference ($p < 0.05$). As can be seen from the data in table 1, the maximum increase in activity and NOS is observed in patients with hypotensive NCDs. Thus, it is due to the pathological induction of activity and NOS in most patients with CKD, it is likely that NCD by HipoTT occurs and progresses. At the same time, patients of other observation groups were found to have a significant probability of activation of this enzyme - an active modulator of the inflammatory response, which was much lower in intensity and did not lead to hyperproduction of NO in patients of groups 1 and 3. It is also possible that patients in these groups have a probable inhibition of endothelial NOS activity, ie true manifestations of ED, which is also a significant risk factor for the formation of NCDs, coronary heart disease and hypertension. At the same time, the results of the study of the activity of enzymes of the glutathione redox detoxification system and POZ, described above, indicate a significant activation of GP and catalase, which probably slightly reduced the intensity of nitrosive stress in patients of groups 1 and 3, but did not affect it intensity in patients of the 2nd group, because they were inhibited. Studies indicate a significant imbalance in endothelial function with ED formation in patients with CKD with HyperTT and CT NCD, as well as an increase in the intensity of nitrosive stress in patients with combined CKD and HipoTT NCD, which make up the vast majority of patients.

Conclusions. In patients with chronic nekamenevym cholecystitis with accompanying neurocirculatory dystonia by hypertensive and cardiac type dominated the intensity of oxidative stress and significant imbalance activity factors protyoksydantnoho protection (brake hlutationovoyi system, compensatory activation of catalase), which promotes the development of endothelial dysfunction and the progression of neuro-tyrkulyatrnoyi dystonia. In most patients with chronic nekamenevym cholecystitis with accompanying neurocirculatory dystonia on hypotonic type on a background of moderate to claim idsylennya intensity of oxidative stress and maximum depletion of the pool of reduced glutathione, prevailing intensity nitrozytyvnoho stress due to the activation of proinflammatory inducible NO-synthase, overproduction monoxide nitrogen, which promotes the development of peripheral venous vasodilation.

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ОБҐРУНТУВАННЯ МЕДИЧНОЇ, СОЦІАЛЬНОЇ НЕОБХІДНОСТІ ТА ЕКОНОМІЧНОЇ ДОЦІЛЬНОСТІ РЕФОРМУВАННЯ ТА ПЛАНУВАННЯ СТОМАТОЛОГІЧНОЇ ОРТОПЕДИЧНОЇ ДОПОМОГИ МОЛОДІ УКРАЇНИ

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SUBSTANTIATION OF MEDICAL, SOCIAL NECESSITY, AND ECONOMIC FEASIBILITY OF REFORMING THE SYSTEM OF ORGANIZATION AND PLANNING OF DENTAL ORTHOPEDIC CARE FOR YOUNG PEOPLE IN UKRAINE

Анотація.

Проведені стоматологічні обстеження 282 осіб молодого віку м. Одеси і 211 в Тернополі та Івано-Франківську і порівняні з даними аналогічних оглядів, проведених в 1995 році (в Одесі – 273 і в Тернополі та Івано-Франківську – 635 осіб). Встановлена значна поширеність і інтенсивність розвитку ортопедичної захворюваності та чітка тенденція до її збільшення, як з віком так і в часі. Доведена регіональна залежність даних показників зі збільшенням її у Західному регіоні країни. Визначена значна частота виникнення зубощелепних аномалій та деформацій, пов'язаних з видаленням зубів, яка становить в даних