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РОЛЬ РОЗЛАДІВ ГЕМОСТАЗУ У ПАТОГЕНЕЗІ ПРОГРЕСУВАННЯ НЕАЛКОГОЛЬНОЇ ЖИРОВОЇ ХВОРОБИ ПЕЧІНКИ ЗА КОМОРБІДНОСТІ З ХРОНІЧНОЮ ХВОРОБОЮ НИРОК

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ROLE OF HEMOSTASIS DISORDERS IN PATHOGENESIS OF NON-ALCOHOL FATTY LIVER DISEASE PROGRESS ON THE BACKGROUND OF CHRONIC KIDNEY DISEASE

Резюме

Мета роботи *– встановити особливості змін ланок системи згортання крові, протизгортаючої активності крові, фібринолізу у хворих на неалкогольну жирову хворобу печінки (НАЖХП) залежно від стадії хронічної хвороби нирок (ХХН).*

Resume

The aim of the research *– to establish the features of changes in the parts of blood coagulation system, anticoagulant activity of the blood, and fibrinolysis in patients with non-alcoholic fatty liver disease (NAFLD) depending on the stage of chronic kidney disease (CKD).*

Матеріал та методи дослідження: *Обстежено 444 хворих: з яких 84 хворих на НАЖХП із ожирінням I ступеня (1 група), яка містила 2 підгрупи: 32 хворих на неалкогольний стеатоз (НАСП) та 52 хворих на неалкогольний стеатогепатит (НАСГ); 270 хворих на НАЖХП із коморбідним ожирінням I ступеня та ХХН I-III стадії (2 група), у тому числі 110 хворих на НАСП та 160 хворих на НАСГ. Групу контролю склали 90 хворих на ХХН I-III стадії із нормальною масою тіла (3 група). Для визначення залежності перебігу НАЖХП від форми та стадії ХНН групи хворих були рандомізовані за віком, статтю, ступенем ожиріння, активністю НАСГ.*

Material and methods of research: *444 patients were examined: 84 of them were with NAFLD and obesity I degree (group 1), which contained 2 subgroups: 32 patients with non-alcoholic steatosis (NAS) and 52 patients with non-alcoholic steatohepatitis (NASH); 270 patients with NAFLD with comorbid obesity of the I degree and CKD I-III stage (group 2), including 110 patients with NAS and 160 patients with NASH. The control group consisted of 90 patients with CKD of I-III stage with normal body weight (group 3). To determine the dependence of the NAFLD course on the form and stage of the CKD, the group of patients was randomized according to age, sex, degree of obesity, and activity of NASH.*

Результати дослідження. *Аналіз показників гемостазу та фібринолізу у обстежених хворих на НАСГ залежно від стадії ХХН показав, що із зростанням стадії ХХН активність зсідання зростає, за виключенням вмісту фібриногену (найбільш ймовірно внаслідок коагулопатії споживання), активність чинників протизсідаючої системи зменшується, сумарна та ферментативна активність фібринолізу знижуються, а неферментативна компенсаторно зростає. Таким чином, метаболічна інтоксикація, оксидативний стрес, які супроводжують перебіг НАЖХП за умов ожиріння та ХХН, сприяють активації калікреїн-кінінової системи, утворенню плазміну та тромбіну з подальшим порушенням рівноваги між ними, розвитку стазу, сладж-феномену, утворенням тромбоцитарних та еритроцитарних агрегатів у системі кровообігу. Наслідком значної активації гемокоагуляції на тлі пригнічення СФА є місцеве згортання крові в артеріях.*

Research results. *Analysis of hemostasis and fibrinolysis indices in examined patients with NASH, depending on the stage of CKD showed that with the growth of the CKD stage, the activity of the cohort increases, with the exception of the fibrinogen content (most likely due to coagulopathy consumption), the activity of the anti-coagulants decreases, the total and enzymatic activity of fibrinolysis is reduced, and non-enzymatic compensator increases. Thus, metabolic intoxication, oxidative stress, which accompany the flow of NAFLD with obesity and CKD, promote the activation of the calicreatin-kinin system, the formation of plasma and thrombin, with subsequent disturbance of equilibrium between them, the development of stasis, slag phenomenon, the formation of platelet and erythrocytic aggregates in blood circulation system. The consequence of significant activation of hemocoagulation against the suppression of total fibrinolytic activity (TFA) is the local clotting of blood in the arteries.*

Висновок. *Таким чином, встановлена роль хронічного запалення при ХХН у формуванні розладів гемостазу та в патогенезі прогресування НАСГ на тлі ожиріння, які в цілому можна охарактеризувати як гіперкоагуляційний синдром внаслідок істотного гальмування чинників протизсідаючої та фібринолітичної систем та активації плазмових факторів коагуляції (фібриноген) внаслідок хронічного запалення.*

Conclusion. *Thus, the role of chronic inflammation in CKD in the formation of hemostasis disorders and in the pathogenesis of NASH progression on the background of obesity, which in general can be characterized as hypercoagulative syndrome due to significant inhibition of anticoagulation factors and fibrinolytic systems and the activation of plasma coagulation factors (fibrinogen) due to chronic inflammation, has been established.*

Keywords: *nonalcoholic fatty liver disease, chronic kidney disease, fibrinolysis, homeostasis.*

Ключові слова: *неалкогольна жирова хвороба печінки, хронічна хвороба нирок, фібриноліз, гомеостаз.*

Introduction. An important problem in internal medicine is the problem of the comorbidity of non-alcoholic fatty liver disease (NAFLD) with obesity and chronic kidney disease (CKD), which has a significant overall medical and social significance (V.T. Ivashkin, 2016; Y.M. Stepanov, 2014). The spectrum of diseases included in the concept of NAFLD includes non-alcoholic steatosis of the liver (NAS), steatohepatitis (NASH), which may be accompanied by liver fibrosis (LF) and transformed into cirrhosis of the liver (LC) (G.D. Fadeenko, 2018; N.V. Kharchenko, 2016). The prevalence of NAFLD in the population is 10-33% (N.V. Kharchenko, G.A. Anokhina, 2011). In the last 5 years in Ukraine, the incidence rate of NAFLD increased by 76.6%. 12-40% patients with NAS developed NASH in 8-13 years with LF. The average age of patients is 50 years (N.V. Kharchenko, G.A. Anokhina, 2011).

The development of NAFLD in obese patients is due to a number of proven factors: insulin resistance, chronic postprandial hyperglycemia, glucose toxicity, glycosylation of structural and transport proteins, hyperlipidemia, dyslipidemia, hepatotoxicity of hypolipidemic agents, etc.) (O.Y. Babak, E.A. Lapshina, 2016; C.M. Tkach, T.L. Cheverda, 2016) and a number of not yet established factors, the study of which is very relevant.

The purpose of the study: to determine the features of changes in the blood coagulation system, anticoagulation activity of the blood, and fibrinolysis in patients with NAFLD depending on the stage of CKD.

Material and methods. 444 patients were examined: of which 84 patients with obesity grade I (group 1), which contained 2 subgroups: 32 patients with NAS and 52 patients with NASH; 270 patients with NAFLD with comorbid obesity of the I grade and CKD I-III stage (group 2), including 110 patients with NAS and 160 patients with NASH. The control group consisted of 90 patients with CKD of I-III stage with normal body weight (group 3). To determine the dependence of the NAFLD course on the form and stage of the CKD, the group of patients was randomized according to age, sex, degree of obesity, and activity of NASH.

Diagnosis of NAFLD was established in accordance with the unified clinical protocol approved by the order of the Ministry of Health of Ukraine No.826 dated on November 6, 2014 [142], in the presence of criteria for the exclusion of chronic diffuse liver disease of the viral, hereditary, autoimmune or drugs origin as causes of cholestatic or cytolytic syndromes, as well the results of ultrasonographic (USG) examination and morphological examination of liver [94, 142]. Diagnosis and treatment of CKD were performed in accordance with the recommendations of the clinical guidelines of the State Institute "Institute of Nephrology, NAMS of Ukraine" (2012) [101, 102, 141, 149]. The study included patients with CKD I-III stage without a nephrotic syndrome with chronic complicated pyelonephritis in the phase of exacerbation decrease or with a latent course.

The total coagulation potential of blood (PT, TT), plasma fibrinolytic activity, plasminogen potential activity (PPA), fibrinogen level in blood plasma, activity of antithrombin III (AT III), activity of XIII factor were studied using the sets of reagents of the company "Simko Ltd" (m Lviv) according to the methods of N. Titsa [178]. Using the reagents of the same company, we studied the state of enzymatic (EFS) and non-enzymatic fibrinolysis (NEF) in blood plasma. The principle of the method is that when azofibrin is incubated with a standard amount of plasminogen in the presence of fibrinolysis activators that are contained in blood plasma, plasmin is formed, whose activity is estimated by the degree of coloring of the solution in alkaline medium in the presence of E-amino-capronic acid (EF) or without (NEF). The difference between them determines the state of the EFS. By the same method, but without the use of plasminogen and E-aminocaproic acid, the proteolytic activity of blood plasma was determined using azoalbumin, azocasein, azocol (Simko Ltd, Lviv) [178], and the total activity of proteinases by M. Kunitz [34] . The statistical analysis was performed using parametric and nonparametric criteria (Student, Pearson) on the RS AMD Athlon 64 using Statistica 5.1 software (StatSoft, Inc., USA) and SPSS 10.0.5. Standard Version.

Results and discussion. Analysis of the results of the 2nd phase of the coagulation hemostasis showed that the PT was significantly lowered in patients of all groups of observation (Table 1). The maximum decrease in the rate was observed in patients with NASH and CKD - 1.9 times compared with the indicator in the PHPs ($p < 0.05$) with the presence of intergroup difference; in patients with NASH without CKD, PT was 1.6 times lower than that in PHPs ($p < 0.05$). In patients with NAS, less intensive changes were observed: PT in the group without comorbidity was 1.2 times lower ($P < 0.05$), in patients with NAS with CKD - 1.4 times ($p < 0 , 05$). In patients with isolated CKD, the decrease in PT was 1.4 times ($p < 0.05$) (Table 1). The study of the

3rd phase of coagulation hemostasis suggests that in patients the content of fibrinogen in the blood was reduced: in patients with NASH and NASH with CKD - respectively, in 1,4 and 2,0 times (p <0,05) against growth in 1, 2 times in patients with isolated CKD (p <0.05); in patients with NAS - the decrease was 12.7% and 17.1% (p <0.05), the indicator was significantly different in comparison with the intergroup aspect (p <0.05). Reducing the fibrinogen content in the blood of patients with NAFLD with CKD and obesity suggests a lack of synthesis of Factor I of coagulation in the liver and / or activation of the hemostasis system in response to inflammation, the development of hypercoagulation, the formation of microthrombus and the addition of a certain amount of fibrinogen in this process.

Table 1

Indicators of hemostasis and fibrinolysis in patients with non-alcoholic liver steatosis and steatohepatitis depending on comorbidity with CKD, in its isolated course (M ± m)

Indicators, units measurement	PHP, n=30	Groups of patients examined				
		NAS, n=32	NAS, CKD, n=110	NASH, n=52	NASH, CKD, n=160	CKD, n=90
PT, sec.	22,12±0,46	18,41± 0,32*	15,73± 0,23 */**	13,56± 0,21 */**	11,38± 0,25 */***/#	16,37± 0,29 */***/##
Fibrinogen, g/l	3,81±0,12	3,38±0,15*	3,15±0,11 *	2,69±0,17 */**	1,87±0,10 */***/#	4,35±0,09 */***/##
TT, sec	16,95±0,87	15,75± 0,36	12,31± 0,27 */**	11,84± 0,23*/**	10,25± 0,15 */***/#	13,27± 0,20 */***/##
AT III, %	95,48±2,01	82,81± 3,18*	78,33± 3,21*	73,38± 2,86*	67,27± 2,24 */***	80,27± 3,28 */##
EFS, E440/ml/hour	1,69±0,02	1,58±0,02*	1,47±0,01*	1,40± 0,01 */**	1,37± 0,004 */***/#	1,52±0,01 */***/##
NEF, E440/ml/hour	0,49±0,02	0,60±0,01*	0,63± 0,003*	0,69± 0,004*/**	0,75±0,01 */***/#	0,57± 0,002 */***/##
EF, E440/ml/hour	1,20±0,01	0,98±0,01*	0,84±0,01 */**	0,71± 0,004*/**	0,62±0,01 */***/#	0,95±0,01 */***/##
Hageman-dependent fibrinolysis, min.	19,45±0,19	22,52± 1,33*	30,21± 1,18*/**	34,53± 1,15*/**	37,31± 1,28 */***	29,39± 1,07 */##
XIII Factor, %	99,91±2,45	97,32± 2,41	82,43± 1,12*	70,82± 1,13*/**	68,18± 1,29 */***	80,25± 2,34 */##
PAP, min.	15,23±0,27	18,31± 0,21*	22,20± 0,18 */**	26,38± 0,13 */**	30,15± 0,12 */***/#	24,01± 0,11 */***/##

Notes: * - the difference is probable compared to the indicator in the PHP (p <0,05);
** - the difference is probable in comparison with the indicator in patients with NAS (p <0,05);
*** - the difference is probable compared with the index in patients with NASH (p <0,05);
# - the difference is probable in comparison with the index in patients with NAS with CKD (p <0,05); ## - the difference is probable compared with the index in patients with NASH with CKD (p <0,05).

Registration of low content of fibrinogen in patients with obesity and obesity is indicative of the development of coagulopathy of consumption, that is, the use of fibrinogen in the processes of intravascular blood coagulation with the simultaneous exhaustion of the circulating pool of this factor, as well as the initial manifestations of PCN. At the same time, the increase in the fibrinogen content in patients with CKD without comorbid pathology indicates activation of blood clotting due to chronic inflammation.

In analyzing the anticoagulation potential of blood, a reduction in TT was found in all groups of patients, with a maximum percentage reduction in patients with NASH with CKD - 1.7 times (p <0.05) compared with the PHP group, but in patients with NASH TT also is likely decreased by 1.5 times (p <0,05) with the presence of a probable intergroup difference (p <0,05). In patients with NAS with CKD, TT was reduced by 1.4 times (p <0.05), and in patients with CKD without comorbid conditions - 1.2 times (p <0.05). In patients with NAS, changes were unlikely (p> 0.05).

Changes in the activity of AT III (Table 1) indicate an insufficiency of the anticoagulation potential of the blood. In particular, the inhibition of AT III activity in all groups of comparison with the maximum inhibition of patients with NASH with CKD was determined 1.4 times (p <0.05) versus a decrease of 1.3 times in patients with NASH (Table 1). In the groups of patients with NAS and NAS with CKD, a moderate difference was not established. It should also be noted that in patients with CKD without comorbid conditions, the activity of AT III was significantly reduced by 1.2 times (p <0.05).

The study of fibrinolytic activity of blood showed that EFS of blood plasma in patients of all groups was significantly lower than the control indexes: in patients with NAS - by 7,1%, patients with NAS with CKD - by 14,9%, patients with NASH - by 17 , 2%, patients with

NASH with CKD - by 18.9%, patients with CKD - by 10.6% (p <0.05) with the presence of a probable intergroup difference between groups with comorbidity and isolated course of CKD (p <0, 05). The suppression of EFS occurred at the expense of the decrease of EF: in patients with NAS the index is significantly lower than the control in 1,2 times, in patients with NAS with CKD - in 1,4 times, in patients with NASH - in 1,7 times, in the group of patients with NASH and CKD - by 1.9 times, while in the group of patients with CKD, the suppression of EF was registered - 1,3 times (p <0,05). At the same time, the NEF in patients of all groups increased in comparison with the PHP group: in patients with NAS, in 1,2 times, in patients with NAS with CKD - in 1,3 times, in patients with NASH - in 1,4 times, in the group of patients with NASH with CKD - 1.5 times, while in the group of patients with CKD the activation of NEF was registered 1.2 times (p <0.05), with the presence of a probable difference between the groups with comorbidity and isolated course of CKD (p <0,05). That is, at patients with NASH with CKD NEF acquired compensatory maximum intensity (p <0,05). At the same time, there was a probable decrease in the activity of Hageman-dependent fibrinolysis: respectively, in patients with NAS - 1.2 times, in patients with NAS and CKD - 1.6 times, in patients with NASH - 1.8 times, in the group patients with NASH with CKD - 1.9 times, while in the group of patients with CKD decrease in Hageman-dependent fibrinolysis activity was 1.5 times (p <0.05) with the probable difference between groups with comorbidity and isolated flow of CKD (p <0.05). The activity of the fibrin stabilizing factor in patients with NASH and NASH with CKD decreased respectively by 1.4 and 1.5 times (p <0.05), indicating a violation of the postcoagulation phase of blood coagulation. In groups of patients with NAS - changes were unlikely, and in patients with NAS with CKD and isolated CKD - reduction was 1.2 times (p <0.05) (Table 1).

Patients with CKD had a probable reduction in PAP: in patients with NAS - 1.2 times, patients with NAS with CKD - 1.5 times, patients with NASH - 1.7 times, patients with NASH with CKD - in 2.0 times, in the group with CKD without comorbidity - the decrease was 1.6 times (p <0.05) with the presence of a probable difference between the groups with comorbidity and the isolated course of CKD (p <0.05) (Table 1).

Analysis of hemostasis and fibrinolysis indices in examined patients with NASH, depending on the stage of CKD showed that with the growth of the CKD stage, the activity of the cohort increases, with the exception of the fibrinogen content (most likely due to consumption coagulopathy), the activity of the anti-coagulation factors decreases, the total and enzymatic activity of fibrinolysis is reduced, and non-enzymatic compensator increases. Thus, metabolic intoxication, oxidative stress, which accompany the flow of NAFLD with obesity and CKD, promote the activation of the calicreatinkinin system, the formation of plasma and thrombin, with subsequent disturbance of equilibrium between them, the development of stasis, slag phenomenon, the formation of platelet and erythrocytic aggregates in blood circulation system. The consequence of significant activation of hemocoagulation against the suppression of EFS is the local clotting of blood in the arteries. The function of Hageman-dependent fibrinolysis is the regular deprivation of the circulatory system from fibrin clots formed under conditions of inflammation. The results of our study indicate a decrease in the rate of enzymatic, Hageman-dependent fibrinolysis, which causes the compensatory activation of NEF. Slowdown of blood circulation in the liver and kidneys due to the formation of microthrombi in the microcirculatory system promotes progression of hypoxia, formation of AOF and free radicals with subsequent damage to cellular membranes of hepatocytes, cytolysis, reduction of GFR and closure of the "vicious" circle of the progression pathogenesis of NAFLD and CKD.

Conclusions The role of chronic inflammation in CKD in the formation of hemostasis disorders and in the pathogenesis of progression of NASH on the background of obesity, which in general can be characterized as hypercoagulation syndrome due to significant inhibition of anti-coagulation factors and fibrinolytic systems and activation of plasma coagulation factors (fibrinogen) due to chronic inflammation.

The prospect of further scientific research in this direction is the development of a method for correction of hemostasis and fibrinolysis indices in patients with NAFLD depending on the stage of CKD.

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РОЛЬ ЕНДОТЕЛІАЛЬНОЇ ДИСФУНКЦІЇ У ВИНИКНЕННІ ТА ПРОГРЕСУВАННІ НЕАЛКОГОЛЬНОЇ ЖИРОВОЇ ХВОРОБИ ПЕЧІНКИ ТА ХРОНІЧНОЇ ХВОРОБИ НИРОК

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THE ROLE OF ENDOTHELIAL DYSFUNCTION IN THE DEVELOPMENT AND PROGRESSION OF NON-ALCOHOL FATTY DISEASE OF LIVER AND CHRONIC DISEASE

Анотація.

У статті наведено теоретичне узагальнення дослідження особливостей функціонального ендотелію за коморбідності хронічної хвороби нирок (ХХН): хронічного пієлонефриту з ожирінням та неалкогольної жирової хвороби печінки (НАСЖХП) залежно від її форми та від стадії ХХН, який характеризується наростаючої дисфункції ендотелію, яка виникає внаслідок впливу метаболічної інтоксикації (гіперліпідемія, постпрандіальна гіперглікемія, зростання ступеня ІР, гіперлептинемія, дефіцит адипонектину, посилення оксидативного та нітрозитивного (гіперактивація iNOS) стресу, і полягає у зниженні ендотелійзалежної вазодилятації плечової артерії, дефіциті синтезу та ліберації монооксиду нітрогену (ендотелійрелаксуючого фактора) внаслідок дефіциту ендотеліальної NO-синтази, істотного зростання пулу злущених ендотеліоцитів, які циркулюють у крові.

Abstract.

The article presents a theoretical generalization of the study of the functional endothelium in the comorbidity of chronic kidney disease (CKD): chronic pyelonephritis with obesity and nonalcoholic fatty liver disease (COPD) depending on its form and the stage of CKD, which is characterized by increasing dysfunction. intoxication (hyperlipidemia, postprandial hyperglycemia, increased IR, hyperleptinemia, adiponectin deficiency, increased oxidative and nitrositive (hyperactivation of iNOS) stress, and is to reduce endothelium-dependent vasodilatation of the brachial artery monocytosis, deficiency and deficiency). synthase, a significant increase in the pool of exfoliated endothelial cells circulating in the blood.

Ключові слова: *хронічна хвороба нирок, неалкогольна жирова хвороба печінки, ендотеліальна дисфункції.*

Keywords: *chronic kidney disease, non-alcoholic fatty liver disease, endothelial dysfunction.*