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Chronic systemic inflammation in nonalcoholic fatty liver disease

The aim — establishment of subclinical inflammation markers in NAFLD patients, associated with obesity.

Materials and methods. The study included 78 NAFLD patients (27 men and 51 women) undergoing therapy in the State Institution «L. T. Mala National Therapy Institute of the National Academy of Medical Sciences of Ukraine». The patients' age ranged from 29 to 78 years old, and the mean age was 54.38 ± 11.88 years old. Other causes of development of liver fatty dystrophy were excluded.

Results. Systemic inflammation is one of main pathonetic mechanisms of NAFLD. Mean CRP levels in NAFLD patients reliably exceeded the values for reference groups and comprised (2.67 ± 0.42) mg/l for nonobese patients and (5.42 ± 1.09) mg/l for obese patients $(p < 0.05)$. It was noteworthy that maximum levels of this acutephase protein were detected in patients with NAFLD associated with obesity, and reliably exceeded the same parameter in other groups $(p < 0.05)$. One of key cytokines involved in development and maintenance of systemic inflammation in NAFLD patients is interleukin6. NAFLD patients showed increased plasma concentration of this cytokine. In nonobese NAFLD patients, IL6 mean concentration was (7.74 ± 0.76) pg/ml and reliably exceeded the relevant parameter in apparently healthy volunteers $(p < 0.05)$. In its turn, mean IL6 level in a group of NAFLD patients with concomitant obesity reached (11.05 ± 1.22) pg/ml, achieving maximum values both versus nonobese NAFLD patients and versus the control group $(p < 0.05)$.

Conclusions. The obtained results are in agreement with literature data regarding subclinical inflammation activity in patients suffering from nonalcoholic fatty liver disease confirmed by elevated CRP and IL6 concentrations. The obtained data are indicative of progression of chronic inflammation in nonalcoholic fatty liver disease associated with obesity.

Key words: nonalcoholic fatty liver disease, obesity, chronic inflammation.

Nonalcoholic fatty liver disease (NAFLD) is the most prevalent liver disease worldwide, which includes a range of liver disorders starting from benign steatosis (i.e. triglyceride accumulation in liver > 5.5 % according to magnetic resonance tomography data) to more severe liver lesion, i.e. lobular inflammation, ballooning degeneration of hepatocytes, fibrosis — nonalcoholic steatohepatitis (NASH) [12, 15]. Potentially, NAFLD may progress to cryptogenic liver cirrhosis and hepatocellular carcinoma [14].

About 30 % of adult population of USA and other Western countries suffer from NAFLD [5]. Nevertheless, true prevalence of this disease is unknown, as NAFLD frequently remains undiagnosed. Still, it is generally known that the risk of NAFLD development is considerably higher (~ twice as high) in case of concomitant obesity compared to healthy nonobese patients [13]. Thus, NAFLD prevalence increases to 57 % in obese patients, to 70 % in patients with diabetes mellitus type 2 (DM 2) and to 90 % in individuals with morbid obesity [8].

In Europe, according to the data by European Association for the Study of the Liver (EASL), NAFLD prevalence in general population is 2—44 % (including children with obesity) and 42.6—69.5 % in patients with DM 2 [3].

In Russian Federation, NAFLD is rather prevalent as well: thus, according to the results of clinical-epidemiological study DIREG L 01903, conducted in Russian Federation in 2007, NAFLD prevalence among patients (n = 30 754) referring to therapists of outpatient clinics, was 26.1 % [1]: among them, liver cirrhosis was identified in 3 % of patients, steatosis — in 79.9 %, and steatohepatitis — 17.1 %. In the age group below 48 years old,

NAFLD was diagnosed in 2305/15 336 (15 %) patients; in the age group above 48 years old, it was diagnosed in 5638/15 095 (37.4 %) patients. Significance of all evaluated risk factors has been established subsequent to the study results; the most prevalent factors in NAFLD population were as follows: arterial hypertension, dyslipidemia, abnormal cholesterol, and abdominal obesity. By each risk factor, percentage of patients in NAFLD population was higher compared to the percentage of patients with the same risk factor in general population of patients included to the analysis.

NAFLD morbidity is related with increased risk of general mortality and mortality related with cardiovascular diseases (CVD) and liver pathology. In a Danish study, with correction for sex, diabetes mellitus and cirrhosis co-morbidity, standardized parameters of NAFLD-related mortality were 2.3 (95 % CI 2.1—2.6) for all-cause mortality, 19.7 (95 % CI 15.3—25.0) for hepatobiliary pathology, and 2.1 (95 % CI 1.8—2.5) for CVD [9]. In SHIP study, risk ratios of all-cause mortality and CVD-related mortality in men with steatosis signs, according to ultrasonic examination data and gamma-glutamyl transpeptidase elevation, were 1.98 (95 % CI 1.21—3.27) and 2.41 (95 % CI 1.05—5.55), respectively. In addition to liver complications, NAFLD patients are at increased risk of developing diabetes mellitus type 2 (DM 2) [6]. For these reasons, NAFLD is considered as a hepatic component of metabolic syndrome (MS) [11].

Moreover, NAFLD is a cause of essential economic loss worldwide, in particular, in Europe: economic analysis of a patient cohort of SHIP project has shown that healthcare expenses in 5 years in a group of patients with NAFLD signs diagnosed by US examination data and elevated alanine aminotransferase (ALT) data exceeded the same parameter for the general population by 26 % [4].

Wide prevalence and considerable economic losses turn the study of NAFLD clinical course particulars and pathogenetic links in combination with obesity into an urgent issue of modern medicine. Results of recent years' studies have shown that NAFLD pathogenesis associated with MS is not limited to insulin resistance; one of novel NAFLD development mechanisms is the presence of systemic inflammation in the body [7].

Thus, the goal of this study was establishment of subclinical inflammation markers in NAFLD patients, associated with obesity. The study was conducted in liver and gastrointestinal diseases department within the scope of research project «Establishment of genetic polymorphism of ADIPOR2 gene and clinical course particulars of nonalcoholic fatty liver disease in patients at cardiovascular risk» (State Registration N 0113U001139).

The study was approved by bioethics commission of the SI «L. T. Mala National Therapy Institute of the NAMS of Ukraine», and conducted in accordance with principles stated in the Declaration of Helsinki. Before the study commencement, all the patients signed the informed consent.

Materials and methods

The study included 78 NAFLD patients (27 men and 51 women): 27 patients with concomitant obesity and 51 non-obese NAFLD patients. The patients' age ranged from 29 to 78 years old, and the mean age was 54.38 ± 11.88 years old. All patients were undergoing therapy in the SI «L. T. Mala National Therapy Institute of the NAMS of Ukraine».

Other causes of development of liver fatty dystrophy — alcohol abuse (consumption of more than 50 g of ethanol a week for men and more than 30 g of ethanol a week for women during the last year); infection with HBV-, HCV-, HDV-viruses; presence of autoimmune or drug-induced hepatitis, Wilson's disease, idiopathic hemochromatosis and congenital $\alpha 1$-antitrypsin deficiency were excluded in the study subjects. The study also did not include patients with severe liver fibrosis and cirrhosis, patients with diabetes mellitus decompensation and those requiring insulin therapy.

The main group included 78 NAFLD patients. The control group included 8 healthy volunteers of comparable sex and age. NAFLD verification was performed based on ultrasonic examination of abdominal cavity organs and clinical-instrumental data. Obesity was diagnosed in accordance with classification of WHO International Obesity Task Force (1997) by calculated body mass index (BMI). Patients of the main group were divided into subgroups of reciprocal sex and age depending on the presence of obesity.

All patients underwent clinical examination with measurement of anthropometric parameters and determination of anatomic fat distribution type (height, body weight, calculation of body mass index (BMI), waist circumference (WC), hip circumference (HC) and waist-to-hip ratio (WHR)).

Studies of pigment and enzymatic metabolism using conventional generally accepted methods were conducted in order to evaluate liver functional state.

Concentrations of total cholesterol (TC) and its fractions — high density lipoproteins TC (HDL TC) and triglycerides were measured using enzymatic method by biochemical analyzer *Humalayzer* (N 2106-1709, Germany) using a reagent kit (*Human*, Germany). Concentration of low density lipoproteins

TC (LDL TC) was calculated using a conventional formula (Friedewald WT). TC concentration in the composition of very low density lipoproteins (VLDL TC) was calculated using the ratio TC/2.22.

Carbohydrate metabolism condition was evaluated in patients by fasting glycemia level, glycated hemoglobin, and insulin concentration. Glucose concentration in venous blood samples was evaluated by photometric method (automated biochemical analyzer — general purpose photometer *Humalyzer 2000*, plant N 183005397, Germany). In order to evaluate long-term carbohydrate metabolism compensation, glycated hemoglobin concentration (HbA_{1c}) was measured using a kit *Reagent* (Ukraine) by reaction with thiobarbituric acid and total hemoglobin using the spectrophotometer *Specol-11* (Germany). Besides, radioimmunological method was applied to measure immunoreactive insulin levels using standard kits *РИО-ИНС-ПГ125I* from *Khopibokh* (Belarus).

In order to evaluate systemic inflammation, serum C-reactive protein (CRP) concentrations were measured using a reagent kit *HS — CRP ELISA KIT — DRG Instruments GmbH* (Germany), and interleukin-6 (IL-6) was measured using a reagent kit *Interleukin-6 — IFA-BEST* from *Vector-BEST* CJSC (Russian Federation).

Statistical processing of the results was performed using a software package SPSS 11.0. All quqantitative data were represented in the form of descriptive characteristics: mean, standard deviation.

Linear regression analysis and Pearson correlation coefficient were used for establishment of relationship between dependent and independent variables. Statistical significance of differences between the mean values for groups were assessed by Student's t-test for odd samples; the differences were considered as statistically significant at $p < 0.05$.

Results and discussion

Evaluation of anthropometric parameters of NAFLD patients has allowed establishing statistically significant differences in BMI and fatty tissue distribution nature. The main anthropometric characteristics of patients included to the study are shown in Table 1.

At evaluation of BMI parameters of examined individuals, increase of this parameter in NAFLD groups was noteworthy (34.12 ± 0.07 kg/m^2 and 27.07 ± 1.12 kg/m^2), which reliably exceeded the relevant parameters in the control groups (24.52 ± 0.88 kg/m^2). The relevant changes were also detected in WC and HC parameters. The said circumferences were reliably higher in patients of NAFLD groups compared to the parameters of apparently healthy volunteers. Mean values of WHR ratio reflecting the presence of abdominal obesity exceeded 1.0 in all NAFLD patients and achieved maximal values in a group of patients with concomitant obesity. It should be mentioned that comparison of mean values of WHR ratio for NAFLD groups showed that this parameter reliably exceeded the relevant parameter in the control group. The obtained data are in agreement with results of other studies showing that NAFLD prevalence increases with BMI increase, and the presence of obesity and abdominal fatty tissue distribution type promote NAFLD formation and represent independent cardiovascular risk factors.

We have identified lipid and carbohydrate metabolism disorders in NAFLD patients compared to healthy individuals. Fasting glycemia elevation occurred in majority of NAFLD patients, both in obese patients (5.71 ± 0.10 mmol/l), and non-obese patients (5.24 ± 0.09 mmol/l). Besides, we have detected reliable increase of insulin concentrations in examined groups of NAFLD patients compared to apparently healthy individuals ($p < 0.01$) (Table 2).

Study of lipid metabolism parameters of NAFLD patients has revealed proatherogenic serum lipid profile. Thus, in obese NAFLD individuals, mean parameters were as follows: TC — (5.69 ± 0.18) mmol, TG — (1.75 ± 0.12) mmol/l, LDL TC — up

Table 1. **The main anthropometric characteristics of examined patients**

Parameter	Control group (n = 18)	Non-obese NAFLD patients (n = 51)	Obese NAFLD patients (n = 27)
BMI, kg/m^2	24.52 ± 0.88	27.07 ± 1.12*	34.12 ± 0.07*#
WC, cm	85.66 ± 3.11	96.88 ± 5.67	112.91 ± 3.91*#
HC, cm	96.61 ± 2.93	96.73 ± 1.45	110.21 ± 4.20*#
WHR, conventional units	0.89 ± 0.11	1.01 ± 0.24	1.02 ± 0.12*

Notes. * $p < 0.05$ versus the control group.
# $p < 0.05$ versus non-obese NAFLD patients.

Table 2. **Lipid and carbohydrate metabolism parameters of examined patients**

Parameter	Control group (n = 18)	Non-obese NAFLD patients (n = 51)	Obese NAFLD patients (n = 27)
Fasting glycemia, mmol/l	4.72 ± 0.35	5.24 ± 0.09*	5.71 ± 0.10*#
Insulin, µU/ml	6.82 ± 1.12	9.72 ± 1.20*	16.39 ± 1.24*#
Total cholesterol, mmol/l	4.76 ± 0.18	5.24 ± 0.12*	5.69 ± 0.18*#
Triglycerides, mmol/l	0.68 ± 0.01	1.51 ± 0.14*	1.75 ± 0.12*
Lipoproteins, mmol/l			
High density	1.66 ± 0.03	1.06 ± 0.02*	0.96 ± 0.02*#
Very low density	0.31 ± 0.01	0.67 ± 0.07*	0.79 ± 0.10*
Low density	2.79 ± 0.03	3.5 ± 0.12*	4.93 ± 0.17*#
Atherogenicity factor	1.87 ± 0.03	3.94 ± 0.13*	3.94 ± 0.15*

Notes. * $p < 0.05$ versus the control group.
# $p < 0.05$ versus non-obese NAFLD patients.

to (4.93 ± 0.17) mmol and low HDL TC — (0.96 ± 0.02) mmol/l, which reliably exceeded the parameters in the reference group. The relevant dynamics was typical of patients with normal body weight and NAFLD (Table 2). No lipid metabolism disorders were detected in the control group: TC — (4.76 ± 0.18) mmol/l, TG — (0.68 ± 0.01) mmol/l, LDL TC — (2.79 ± 0.03) mmol/l, HDL TC — (1.66 ± 0.03) mmol/l.

The detected changes are suggestive of the development of lipid and carbohydrate metabolism disorders in NAFLD patients, conforming to pathogenetic links of insulin resistance syndrome formation.

The study of the role of low grade chronic systemic inflammation in development and progression of metabolic syndrome as a whole and its components, NAFLD in particular, remains an urgent issue. Establishment of relationship between subclinical inflammation markers, pathogenetic links of insulin resistance syndrome, and excessive fat accumulation in liver is essential.

Recently obtained data are indicative of the presence of associative links between numerous inflammation mediators, in particular, interleukin-6 (IL-6), pro-inflammatory markers such as CRP, and

formation of excessive body weight and insulin resistance syndrome [10].

In order to assess the intensity of systemic inflammation in patients of examined groups, concentrations of such inflammation markers as highly sensitive CRP and IL-6 were measured (Table 3).

Evaluation of CRP concentration (Table 3) has established that this parameter was (0.11 ± 0.03) mg/l in the control group. Mean CRP levels in NAFLD patients reliably exceeded the values for reference groups and comprised (2.67 ± 0.42) mg/l for non-obese patients and (5.42 ± 1.09) mg/l for obese patients (p < 0.05). It was noteworthy that maximum levels of this acute-phase protein were detected in patients with NAFLD associated with obesity, and reliably exceeded the same parameter in other groups (p < 0.05).

One of key cytokines involved in development and maintenance of systemic inflammation in NAFLD patients is interleukin-6. It plays a special role of hepatocyte activating factor. This cytokine is capable of activating the synthesis of many acute-phase proteins, in particular, C-reactive protein, elevation of which is a well-known cardiovascular disease risk factor. Comparative analysis has re-

Table 3. **Systemic inflammation markers in NAFLD patients**

Parameter	Control group (n = 18)	Non-obese NAFLD patients (n = 51)	Obese NAFLD patients (n = 27)
C-reactive protein, mg/l	0.11 ± 0.03	2.67 ± 0.42*	5.42 ± 1.09*#
Interleukin-6, pg/ml	3.86 ± 0.34	7.74 ± 0.76*	11.05 ± 1.22*#

Notes. * $p < 0.05$ versus the control group.
# $p < 0.05$ versus non-obese NAFLD patients.

vealed that the lowest IL-6 concentration was observed in the control group and comprised (3.86 ± 0.34) pg/ml. NAFLD patients showed increased plasma concentration of this cytokine. In non-obese NAFLD patients, IL-6 mean concentration was (7.74 ± 0.76) pg/ml and reliably exceeded the relevant parameter in apparently healthy volunteers $(p < 0.05)$. In its turn, mean IL-6 level in a group of NAFLD patients with concomitant obesity reached (11.05 ± 1.22) pg/ml, achieving maximum values both versus non-obese NAFLD patients and versus the control group $(p < 0.05)$.

The obtained results are in agreement with literature data regarding subclinical inflammation activity in patients suffering from nonalcoholic fatty liver disease [2]. The presence of chronic inflammation can be explained by increased volume of fatty tissue and its re-distribution, which, in its turn, is associated with cytokine synthesis dysregulation: increased synthesis of pro-inflammatory mediators secreted by visceral fatty tissue promotes increased synthesis of acute phase proteins by liver. On the other hand, formation of insulin resistance, in its turn, results in disturbance of essential mechanisms allowing insulin to exert selective influence on protein synthesis in liver, increasing albumin synthesis and decreasing CRP and fibrinogen synthesis. Thus, increased synthesis of acute phase proteins, in particular, CRP, occurs as a result of hyperinsulinemia, caused by decreased tissue sensitivity to insulin [2].

Conclusions

Nonalcoholic fatty liver disease is among the most prevalent liver diseases. Anthropometric changes detected are suggestive of formation of abdominal obesity type and development of insulin resistance syndrome. NAFLD pathogenesis is associated with development of subclinical chronic inflammation confirmed by elevated CRP and IL-6 concentrations. The obtained data are indicative of progression of chronic inflammation in nonalcoholic fatty liver disease associated with obesity. Potential further studies include those aimed at advanced understanding of inflammation process drivers and factors capable of influencing its course, which represent an urgent medical problem.

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Хронічне системне запалення при неалкогольній жировій хворобі печінки

Мета — визначити маркери субклінічного запалення у хворих на неалкогольну жирову хворобу печінки (НАЖХП), асоційовану з ожирінням.

Матеріали та методи. У дослідження залучено 78 хворих на НАЖХП (27 чоловіків і 51 жінка), які перебували на лікуванні в ДУ «Національний інститут терапії імені Л. Т. Малої НАМН України», з них 27 пацієнтів із супутнім ожирінням. Вік пацієнтів — 29—78 років, середній вік — $(54,38 \pm 11,88)$ року. В усіх пацієнтів виключено інші причини формування жирової дистрофії печінки.

Результати. Системне запалення — один з основних патогенетичних механізмів розвитку НАЖХП. Концентрація С-реактивного білка у хворих на НАЖХП була достовірно вищою ($p < 0,05$), ніж у практично здорових осіб: $(2,67 \pm 0,42)$ мг/л у пацієнтів з НАЖХП без ожиріння і $(5,42 \pm 1,09)$ мг/л — у пацієнтів з НАЖХП з ожирінням, при цьому в останніх цей показник достовірно ($p < 0,05$) перевищував такий у хворих на НАЖХП без ожиріння. Вміст інтерлейкіну-6 (ІЛ-6) у хворих на НАЖХП і супутнім ожирінням достовірно перевищував показник групи хворих на НАЖБП без ожиріння (($11,05 \pm 1,22$) і $(7,74 \pm 0,76)$ пг/мл відповідно), при цьому обидва значення були достовірно вищими ($p < 0,05$), ніж рівень ІЛ-6 у практично здорових осіб.

Висновки. Отримані результати відповідають даним літератури про активність субклінічного запалення у хворих на НАЖХП, про що свідчить підвищення рівня С-реактивного білка та ІЛ-6. Установлено прогресування хронічного запалення у разі НАЖХП за наявності ожиріння.

Ключові слова: неалкогольна жирова хвороба печінки, ожиріння, хронічне запалення.

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Хроническое системное воспаление при неалкогольной жировой болезни печени

Цель — определить маркеры субклинического воспаления у больных неалкогольной жировой болезнью печени (НАЖБП), ассоциированной с ожирением.

Материалы и методы. В исследование включены 78 больных НАЖБП (27 мужчин и 51 женщина), находившихся на лечении в ГУ «Национальный институт терапии имени Л. Т. Малой НАМН Украины», из них 27 пациентов с сопутствующим ожирением. Возраст пациентов — 29—78 лет, средний возраст — $(54,38 \pm 11,88)$ года. У всех пациентов исключены другие причины формирования жировой дистрофии печени.

Результаты. Системное воспаление — один из основных патогенетических механизмов развития НАЖБП. Концентрация С-реактивного белка у больных НАЖБП была достоверно выше ($p < 0,05$), чем у практически здоровых лиц: $(2,67 \pm 0,42)$ мг/л у пациентов с НАЖБП без ожирения и $(5,42 \pm 1,09)$ мг/л — у пациентов с НАЖБП с ожирением, при этом у последних данный показатель достоверно ($p < 0,05$) превышал таковой у больных НАЖБП без ожирения. Содержание интерлейкина-6 (ИЛ-6) у больных НАЖБП с сопутствующим ожирением достоверно превышал показатель группы больных НАЖБП без ожирения (($11,05 \pm 1,22$) и $(7,74 \pm 0,76)$ пг/мл соответственно), при этом оба значения были достоверно выше ($p < 0,05$), чем уровень ИЛ-6 у практически здоровых лиц.

Выводы. Полученные результаты соответствуют данным литературы об активности субклинического воспаления у больных НАЖБП, о чем свидетельствует повышение уровня С-реактивного белка и ИЛ-6. Установлено прогрессирование хронического воспаления при НАЖБП при наличии ожирения.

Ключевые слова: неалкогольная жировая болезнь печени, ожирение, хроническое воспаление.

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