of Baku. During the seven-year study period (2013-2020), 559 patients were diagnosed with MS. Of these, 392 (70.1%) patients were female and 167 (29.9%) were male. At the same time, in 390 (69.9%) patients the disease had recurrent (RMS), 109 (19.5%) had progressive (PMS) types of course, and 60 (10.6%) had a clinically isolated syndrome (CIS). CIS was more common in the age group of 20-29 years, RMS in the group of 30-39 years, and PMS was typical in the groups of 40-49 and 50-59 years. The manifestation of the disease in the CIS and RMS groups began at almost the same age (26.0 (24.0-34.0) and 27.0 (22.0-34.0)), with clinical manifestations of damage in one functional system, with a statistically significant difference in age with PRS (34.0 (25.0-41.0)), where the catastrophe was observed in several functional systems. Atrophic changes on MRI of the brain predominated in the group of patients with PMS type of course from the first symptoms of the disease (21.1% of cases), and with subsequent visualizations it tended to increase, reaching 57.4%. In patients treated with a drug that modifies the course of MS – beta interferon 1a, by the end of the year of treatment, EDSS in the CIS and PMS groups remained stable (3.0 (2.0-4.0) and 4.25 (4.0-5.5), respectively), while in the RMS group there was a statistically significant improvement in clinical condition (2.0 (1.0-4.0)).

Key words: multiple sclerosis, clinical features, clinical course.

ORCID and contributionship:
Guliyeva A. I.: https://orcid.org/0000-0002-7235-4444 ABCDEF

Corresponding author
Guliyeva Aygun Isa
Azerbaijan State Advanced Training Institute for Doctors named after A.Aliyev
Azerbaijan, AZ 1012, Baku, 3165 Tbilisi Avenue
Tel.: +994557621780
E-mail: Statya2021@mail.ru

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Ihnatko Ya. Ya., Derbak M. A., Chubirko K. I., Moskal O. M.

FEATURES OF THE FUNCTIONAL STATE OF THE LIVER AND INDICATORS OF ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH CORONARY HEART DISEASE COMBINED WITH NON-ALCOHOLIC FATTY LIVER DISEASE

"Uzhgorod National University" (Uzhhorod, Ukraine)
yaroslav.ihnatko@uzhnu.edu.ua

Long-term CHD comorbid with NAFLD leads to acceleration of atherogenesis, imbalance of ED and adipocytokines. To compare indicators of the liver functional state, levels of adipocytokines, C-reactive protein and marker of endothelial dysfunction in patients with CHD comorbid with and without NAFLD was the aim of research. 145 patients with CHD comorbid with NAFLD and 100 patients with CHD were examined. When analyzing the obtained laboratory parameters characterizing the liver functional state, it was established that in patients with CHD comorbid with NAFLD, a significant moderate elevation in the levels of ALT and AST was registered, which characterizes the presence of cytolysis syndrome. An imbalance of adipocytokines was also found in these patients: a decline in the concentration of adiponectin, an elevation in the level of resistin, and a decline in the ratio of adiponectin to resistin. A substantive increase in the serum level of ADMA and CRP in patients with combined pathology indicates the presence of ED against the background of activation of systemic inflammation, which may have pathogenetic significance in the growing of CHD comorbid with NAFLD.

Key words: *CHD, NALFD, adipocytokines, resistin, endothelial dysfunction.*

Connection of the publication with planned research works.

The scientific research was conducted according of the plan of the departmental topic of the Department of Therapy and Family Medicine of UzhNU “Innovative methods of the diagnostic and the treatment of pathology of internal organs in obese patients” state registration No. 0121U111773.

Introduction.

Coronary heart disease (CHD) is the common disorder of the cardiovascular system (CVD) and has dangerous consequences that cause disability and premature death. In Ukraine, the incidence of CHD has doubled over the last decade and is 68.8% [1]. Modern data on the pathogenesis of CHD prove that atherosclerosis is the result of a process of chronic inflammation of low intensity and, thanks to lipoperoxidation, causes the transformation of low-density lipoproteins (LDL) with

the acquisition of antigenic properties and the appearance of insoluble LDL-IgG immune complexes in the blood, which are actively captured by macrophages and deposited in intimate arteries. The direct damaging factor of endotheliocytes is specifically modified LDL, which causes a decrease in NO synthesis by the endothelium (ET), an elevation in the producing of endothelin-1, impart to an increase in the pro-aggregant properties of ET and its desquamation, as is the conclusion of endothelial dysfunction (ED) [2, 3, 4]. A sensitive marker characterizing ED is asymmetric dimethylarginine (ADMA), which is a methylated derivative of L-arginine and has the property of inhibiting NO synthase, which leads to a decrease in NO formation in blood vessels and other tissues.

In recent years, the potential role of ADMA in the development of CVD has attracted considerable attention of researchers. Due to the data of numerous studies, an increased level of ADMA in blood plasma was seen in patients with a combination of CHD with hypercholesterolemia, hypertension, diabetes mellitus (DM) type 2 and insulin resistance, hypertriglyceridemia, hyperhomocysteinemia [5, 6, 7]. Distinct attention is paid to the close relationship between CHD and the pathology of the hepatobiliary system, primarily with non-alcoholic fatty liver disease (NAFLD) [8, 9]. In particular, research in recent years has proven that NAFLD acts a considerable role in the formation of risk factors for CVD. On the one hand, impaired liver function is one of the weighty factors in the expansion of dyslipoproteinemia, since changes in lipid metabolism begin at the hepatocyte level, and on the other hand, the liver is a target organ in atherogenic dyslipidemia [10, 11].

The concept of NAFLD unites the spectrum of clinical and morphological changes of the liver, represented by non-alcoholic fatty hepatosis (NAFL), non-alcoholic steatohepatitis (NASH), liver fibrosis (LP), liver cirrhosis and hepatocellular carcinoma, which develop in person who do not drink alcohol in hepatotoxic doses [12]. There are data on the detection of NAFLD in patients with cardiovascular pathology, namely: widespread atherosclerosis, dyslipoproteinemia, and metabolic syndrome (MS) [13]. The presence of NAFLD can be predicted in individuals with obesity and a very high risk of developing cardiovascular diseases [14]. However, many studies have proven that patients with NAFLD, even without signs of MS, have a high risk of developing atherothrombosis [15]. Due to the guideline sof hepatological associations, the mandatory screening plan for patients with NAFLD includes not only additional examinations to detect NASH and LF, but also valuation of metabolic disorders and cardiovascular risk. Therefore, today, NAFLD can be considered as an early predictor of the development of CVD [16].

It should be noted that NAFLD is considered as a chronic inflammatory process of low intensity, which contributes in the form of additional atherogenic stimuli to the already formed pro-inflammatory status in CHD [17, 18]. Also, in the pathogenesis of the combined course of CHD and NAFLD, special importance is attached to adipose tissue hormones adiponectin, which has antidiabetic, antiatherogenic and anti-inflammatory effects [19], and resistin, which is associated with systemic inflammation and insulin resistance [20].

Since CHD and NAFLD have common pathogenetic mechanisms of development, they contribute to the mutually aggravating course of these pathologies. Therefore, an important task today remains the study of common pathogenetic mechanisms of the formation and progression of CAD and NAFLD with the aim of their early diagnostic and the forming of differentiated approaches to treatment [21].

The aim of the study.

To compare indicators of the liver functional state, levels of adipocytokin, C-reactive protein, and endothelial dysfunction in patients with CHD comorbid with and without NAFLD.

Object and research methods.

The study involved 245 patients with CHD, with whom were created two groups: 1 group (n=145) – patients with stable angina pectoris, II functional class (FC) in combination with NAFLD, and 2 group (n=100) – patients with stable angina pectoris voltage of the II FC without NAFLD, who were treated at the Communal Nonprofit Enter-prise «Central City Clinical Hospital» of Uzhhorod City Council in the cardiology department with intensive care units (ICU). The average age of the examinees was 55.3±6.3, by gender 140 (57%) men predominated, 105 (43%) women. The control group included 30 clinically and anamnestically healthy individuals aged 56.2±5.1 years, among whom there were 14 (46%) men and 16 (54%) women. The study was approved by the local ethics committee (protocol No. 7/2 dated March 16, 2023), and at the beginning, all patients signed an informed consent to participate in its conduct, in accordance with the requirements of the Helsinki Declaration of 1975.

The diagnosis of stable CHD was verified based on the results of electrocardiography (ECG), coronary angiography, history of MI and/or myocardial revascularization interventions in accordance with the unified clinical protocol “Stable coronary heart disease” (Order of the Ministry of Health of Ukraine No. 152 of 03/02/2016). The diagnosis of NAFLD was made in accordance with the unified clinical protocol “Non-alcoholic steatohepatitis” (Order of the Ministry of Health of Ukraine No. 826 of 06.11.2014) [22], in accordance with the recommendations of the European Association for the Study of the Liver (EASL), the European Association for the Study of Diabetes (EASD), the European Association on the study of obesity (EASO) [23]. The study excluded patients with positive markers of viral hepatitis in the blood (HBsAg, antiHCV, PCRHCV, PCRHBV, etc.), in which fatty liver dystrophy was caused by hepatotoxic drugs, patients with inflammatory bowel diseases, celiac disease, as well as those who drank alcohol excessively (≥30 g/day – men and ≥20 g/day – women).

The patients underwent a general clinical examination (determination of body weight, height, waist circumference), biochemical tests, and sonogrphic examination of abdomen for the diagnosis of NAFLD. Functional tests of the liver were determined (ALT, AST, gammaglutamyltransferase (GGT), alkaline phosphatase (AL), C-reactive protein (CRP), total bilirubin. Enzyme-Immuno-Sorbent-Assay (ELISA) was used in the work to determine adiponectin levels , resistin and asymmetric dimethylarginine (ADMA) in blood serum and sets of reagents manufactured by Mediagnost and “Human Asymmetrical Dimethylarginine ELISA”.

Table 1 – Clinical characteristics of the patients

Parameters, units of measurement	Group 1 CHD+NALFD n=145	Group 2 CHD n=100	Control group n=30
Age, years (M±m)	54,4±6,2	57,1±5,6	56,2±5,1
Men, abs. (%)	80/55,2	60/60	14/46,7
Women, abs. (%)	65/44,8	40/40	16/53,3
HR, beats/min (M±m)	84±6,1▪	86±3,2	76±4,8
SBP, mmHg (M±m)	160±9,6▪	155±7,6	134±5,3
DBP, mmHg (M±m)	90±10,1▪	85±10,3	70±6,8
AH, abs/%	71/49	69/69	-
Duration of hypertension, years, (M±m)	12±10,1	13±11,2	-
Duration of CHD, years, (M±m)	5±3,2	6±4,1	-
Duration of NAFLD, years, (M±m)	4±3,3	-	-
BMI, kg/m^2, (M±m)	31,5±7,7*▪	28,5±6,2	25,3±4,4
Obesity, abs/%	36/25*▪	15/15	-
Dyslipidemia, abs/%	15/10*▪	6/6	2/6
Weighted heredity, abs/%	60/41,3	63/63	1/3
Smoking, abs/%	76/52,4	48/48	14/46,7

Notes: *- reliability of the difference in indicators between groups 1 and 2 of patients; ▪ – the reliability of the difference between the indicators of the examined patients and the group of control (p<0.05).

Statistical processing of the gotting results was carried out with the help of software – the Microsoft Excel spreadsheet and the using Janovi version 2.3.28. The estimation of the probability of differences in mean values was getting with using the Student's paired t-test.

Research results and their discussion.

Analyzing the obtained results, it should be noted that the patients were comparable in age; according to the objective examination, the levels of systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR) in groups 1 and 2 did not differ statistically from each other, but a difference was found in these indicators with the group of control (p<0.05).

According to anamnestic data, no statistically significant differences in the average duration of CHD, arterial hypertension (AH), and NAFLD were seen between the groups of patients.

In the observed patients of the first and second groups, there is a statistical difference in the presence of obesity and dyslipidemia at p<0.05. When comparing the first group and the group of control, a statistical difference was established with the presence of dyslipidemia at p<0.05.

No significant difference was found between groups 1 and 2 in the frequency of identification of burdened heredity and smoking, but a difference was detected in the indicator of burdened heredity in comparison with the group of control at p<0.05 **(table 1)**.

Table 2 – Indicators of the functional state of the liver in the observed patients

Parameters, units of measurement	Group 1 CHD+NALFD n=145	Group 2 CHD n=100	Control group n=30
ALT, Units/l (M±m)	51,8±9,2* ▪	27,1±9,4	24,5±4,3
AST, Units/l (M±m)	47,3±7,1* ▪	24,7±6,4	21,3±6,4
AL, Units/l (M±m)	230,5±14,7* ▪	69,7±5,1*	58,6±5,2
GGT, Units/l (M±m)	51,7±2,3* ▪	35,6±7,4*	23,7±4,8
Total bilirubin, mmol/l (M±m)	13,4±2,4	13,1±2,1	8,8±2,6

Notes: *- reliability of the difference in indicators between groups 1 and 2 of patients; ▪ –the reliability of the difference between the indicators of the examined patients and the group of control (p<0.05).

When analyzing the obtained laboratory parameters characterizing the liver functional state, it was showed that the individuals of the first group had a moderate increase in the levels of ALT and AST, which characterizes the presence of cytolysis syndrome. This confirms the presence of nonalcoholic steatohepatitis of a moderate degree of activity in these patients, which probably potentiates the activity of chronic systemic inflammation of low intensity compared to group 2 and the control group at p<0.05. Also, in patients with CHD with NAFLD, an elevation in the levels of GGT and AP was noted in comparison with group 2 and the control group at p<0.05, which proves the presence of cholestasis syndrome. Although in patients with CHD, all indicators of the liver functional state were within the normal level, however, a significant increase in AL and GGT was found in comparison to the group of practically healthy individuals p<0.05.

Thus, in patients with CHD in combination with NAFLD, moderate impairment of the liver functional state was determined **(table 2)**.

According to the sonographic examination of the abdomen, 110 (75.8%) patients with CHD in combination with NAFLD showed ultrasound signs of hepatosis: increased echostructure, increased echogenicity, and decreased sound conductivity of the liver, and 68 (46.8%) people increased liver size (hepatomegaly), 42 (28.9%) patients were diagnosed with chronic cholecystitis (densification of the gallbladder wall). In patients with CHD without NAFLD, no increase in the size or increase in echogenicity of the liver was found.

In order to assess the blood flow in the liver vessels, pulsed Doppler imaging of the liver vessels was used **(table 3)**. This examination demonstrated a significant raise in the blood flow rate in the portal vein (p<0.05) in patients with CHD comorbid with NAFLD compared to group 2 and the group of control, as well as a significant raise in the blood flow rate in the hepatic veins (p<0.05) compared to with group 2 and the group of control.

The levels of adiponectin and resistin in the subjects are shown in **table 4**. An imbalance in the level of adipocytokines in patients with CHD comorbid with NAFLD was revealed. The serum level of adiponectin in CHD patients with NAFLD was 80% lower than in healthy subjects (p<0.05 and 41.1% lower than in group 2, while resistin was 64.9% higher, than in the group of control, and by 23.3% higher than in group 2 (p<0.05).In patients of group 1, a significant decline in the ratio of adipocytokines was observed: the adiponectin/resistin ratio was 17 times lower compared to the group of control and 5.5 times less compared to group 2 (p<0.05).

Indicators of endothelial dysfunction in the subjects are presented in **table 5**. It was shown that in patients with CHD comorbid with NAFLD, the serum level of ADMA was 79% higher than in healthy individuals, and 25.9% higher, compared to group 2 (p<0 .05). The value of the CRP indicator in patients of the first group 1 exceeded the control group by 3.1 times, patients of group 2 by 1.7 times.

Therefore, in patients with CHD in combination with NAFLD, an imbalance of adipocytokines was revealed: a decline the concentration of adiponectin, a raising in the level of resistin, and a decline in the ratio of adiponectin to resistin, compared to the control group and patients with CHD without liver pathology. A significant raise in the serum level of ADMA and CRP in patients of group 1 indicates the presence of ED against the background of activation of systemic inflammation, which may have a pathogenetic significance in the development and progression of CHD in the presence of comorbidity with NAFLD.

When studying the adipocytokine profile, we obtained data on the presence of an imbalance of adipocytokines in patients with CHD in combination with NAFLD, which coincides with a number of studies that testify to the clinical significance of hypoadiponectinemia as a risk factor for the development of CHD. Thus, Fiaschi T. proved that a low level of adiponectin is an independent risk factor for CHD with NAFLD [24]. Our data are consistent with the clinical studies of Khoramipour K. et al. (2021), which established that in patients with NAFLD, the level of adiponectin is reduced and is inversely correlated with the severity of inflammation and liver damage [25]. It has also been proven that adiponectin diminishes the degree of steatosis with a high-calorie diet, obesity, and insulin resistance [26, 27].

Table 3 – Indicators of vascular blood flow of the liver

Parameters, units of measurement	Group 1 CHD+NALFD n=145	Group 2 CHD n=100	Control group n=30
Venous blood of v. portae hepatic, m/s (M±m)	0,39±0,10*▪	0,29±0,06	0,21±0,05
Venous blood of v. hepatica, m/s (M±m)	0,22±0,05*	0,19±0,06	0,13±0,02

Notes: *- reliability of the difference in indicators between groups 1 and 2 of patients; ▪ – the reliability of the difference between the indicators of the examined patients and the group of control (p<0.05)

Table 4 – Concentration of adiponectin, resistin and their ratio in examined patients

Parameters, units of measurement	Group 1 CHD+NALFD n=145	Group 2 CHD n=100	Control group n=30
Adiponectin, μg/ml (M±m)	3,67±2,7*▪	10,83±2,4	18,4±2,1
Resistin, μg/ml (M±m)	15,4±5,8*▪	11,8±3,6	5,4±1,7
Adiponectin/resistin (M±m)	0,2±0,1*▪	1,1±0,4	3,4±0,8

Notes: *- reliability of the difference in indicators between groups 1 and 2 of patients; ▪ – the reliability of the difference between the indicators of the examined patients and the group of the control (p<0.05).

Table 5 – Markers of systemic inflammation and endothelial dysfunction

Parameters, units of measurement	Group 1 CHD+NALFD n=145	Group 2 CHD n=100	Control group n=30
CRP, mg/l	5,2±1,8*▪	3,1±0,8	1,7±0,4
ADMA, μmol/l	0,82±0,05 *▪	0,61±0,07 ▪	0,17±0,01

Notes: * – reliability of the difference in indicators between groups 1 and 2 of patients; ▪ – the reliability of the difference between the indicators of the examined patients and the group of control (p<0.05).

Conclusions.

1. In CHD patients with NAFLD, compared to CHD patients without liver pathology, a significant increasing in the serum level of resistin, ADMA, CRP, a decrease in the concentration of adiponectin and the adiponectin/resistin ratio were found.

2. The detected increase in blood flow velocity in the portal and hepatic veins in patients with CHD in combination with NAFLD may be a consequence of possible densification of the liver parenchyma against the background of a violation of its functional state and the reaction of vessels to chronic vascular inflammation of low-intensity.

Prospects for further research.

The study of common pathogenetic mechanisms of the development of CHD and NAFLD in conditions of chronic systemic inflammation and the search for new effective methods of diagnosis and treatment.

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ОСОБЛИВОСТІ ФУНКЦІОНАЛЬНОГО СТАНУ ПЕЧІНКИ ТА ПОКАЗНИКІВ ЕНДОТЕЛІАЛЬНОЇ ДИСФУНКЦІЇ У ХВОРИХ НА ІШЕМІЧНУ ХВОРОБУ СЕРЦЯ У ПОЄДНАННІ З НЕАЛКОГОЛЬНОЮ ЖИРОВОЮ ХВОРОБОЮ ПЕЧІНКИ

Ігнатко Я. Я., Дербак М. А., Чубірко К. І., Москаль О. М.

Резюме. Серцево-судинні захворювання та НАЖХП мають спільні механізми взаємообтяжуючого перебігу за рахунок спільних механізмів патогенезу. З метою порівняння показників функціонального стану печінки, рівнів адипоцитокінів, С-реактивного протеїну та ендотеліальної дисфукції було обстежено 145 хворих на ІХС комор бідну з НАЖХП та 100 хворих на ІХС. За допомогою стандартних методик проводилося загальноклінічне обстеження, УЗД ОЧП, визначали функціональні проби печінки, СРП, адипонектин, резистин та АДМА. Встановлено, що у хворих на ІХС у поєднанні з НАЖХП визначається достовірне підвищення рівнів АЛТ та АСТ, що визначає присутність синдрому цитолізу. Це підтверджує у даних пацієнтів наявність неалкогольного стеатогепатиту помірного ступеня активності, який, імовірно, впливає на активність низькоінтесивного хронічного системного запалення. Також у пацієнтів на ІХС з НАЖХП відмічено збільшення рівнів ГГТ та ЛФ, що доводить наявність синдрому холестазу. У пацієнтів з ІХС усі показники функціонального стану печінки знаходились у межах фізіологічної норми. За даними ультразвукової діагностики ОЧП у 75,8% у пацієнтів з ІХС у поєднанні з НАЖХП виявлено ультразвукові ознаки гепатозу: підсилення ехоструктури, гіперехогенність, а в 46,8% осіб – гепатомегалія, у 28,9 % хворих діагностовано хронічний холецистит. У хворих на ІХС без НАЖХП збільшення розмірів та підвищення ехогенності печінки не виявлено. Згідно даних імпульсної допплерографії судин печінки виявлено значиме підсилення швидкості кровотоку у ворітній вені та підсилення швидкості кровотоку в печінкових венах. У хворих з ІХС у поєднанні з НАЖХП виявлено дисбаланс адипоцитокінів порівняно з контрольною групою та з хворими на ІХС без патології печінки. Достовірне зростання сироваткового рівня АДМА та СРП у хворих на ІХС з коморбідною патологією показує наявність дисфункції ендотелію під час активації системного запалення, що може мати патогенетичне значення у розвитку та прогресуванні ІХС за умови коморбідності з НАЖХП.

Ключові слова: ІХС, НАХЖП, адипоцитокіни, резистин, ендотеліальна дисфункція.

FEATURES OF THE FUNCTIONAL STATE OF THE LIVER AND INDICATORS OF ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH ISCHEMIC HEART DISEASE COMBINED WITH NON-ALCOHOLIC FATTY LIVER DISEASE

Ihnatko Ya. Ya., Derbak M. A., Chubirko K. I., Moskal O. M.

Abstract. Noalcoholic fatty liver disease affects the course of CVD due to common pathogenetic mechanisms. In order to compare indicators of the functional state of the liver, levels of adipocytokines, C-reactive protein, and endothelial dysfunction, 145 patients with CHD in combination with NAFLD and 100 patients with CHD were examined . Due to standard methods, a general clinical examination was carried out, an ultrasound of abdomen, functional tests of the liver, CRP, adiponectin, resistin and ADMA were determined. It was established that in patients with CHD in combination with NAFLD, a moderate increase in ALT and AST levels is determined, which characterizes the presence of cytolysis syndrome. This confirms the presence of nonalcoholic steatohepatitis of a moderate degree of activity in these patients, which probably potentiates the activity of low-intensity chronic systemic inflammation. Also, in patients with CHD with NAFLD, an increase in GGT and AP levels was noted, which proves the presence of cholestasis syndrome. In patients with CHD, all indicators of the functional state of the liver were within the physiological norm. According to the ultrasound diagnosis of abdomen, in 75.8% of patients with CHD in combination with NAFLD, ultrasound signs of hepatosis were found: increased echo structure, hyperechogenicity, and decreased conductivity of the sound of the liver, and in 46.8% of people, the size of the liver was increased, in 28.9% of patients diagnosed chronic cholecystitis. In patients with CHD without NAFLD, no increase in the size or increase in echogenicity of the liver was found. Due to the data of pulsed dopplerography of the

liver vessels, a significant raise in the blood flow rate in the portal vein and an increase in the blood flow rate in the hepatic veins were revealed. In patients with CHD comorbid with NAFLD, an imbalance of adipocytokines was revealed: a decline in the concentration of adiponectin, an raise in the level of resistin, and a decline in the ratio of adiponectin to resistin, in comparison with the control group and with patients with CHD without liver pathology. A significant increasing in the serum level of ADMA and CRP in patients with CHD with poor ventricle pathology indicates the presence of ED against the background of activation of systemic inflammation, which may have a pathogenetic significance in the development and progression of CHD in the presence of comorbidity with NAFLD.

Key words: CHD, NAFLD, adipocytokines, resistin, endothelial dysfunction.

ORCID and contributionship:
Ihnatko Ya. Ya.: https://orcid.org/0000-0003-1618-8952 ABCD
Derbak M. I.: https://orcid.org/0000-0003-4791-4080 BDE
Chubirko K. I.: https://orcid.org/0000-0002-4379-0538 F
Moskal O. M.: https://orcid.org/0000-0001-8615-9933 E

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Corresponding author
Ihnatko Yaroslav Yaroslavovych
Uzhhorod National University
Ukraine, 88000, Uzhhorod, 20 Griboyedova str.
Tel.: 0958408087
E-mail: yaroslav.ihnatko@uzhnu.edu.ua

A – Work concept and design, **B** – Data collection and analysis, **C** – Responsibility for statistical analysis, **D** – Writing the article, **E** – Critical review, **F** – Final approval of the article.

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[1]Kalashnikov O. O., [2]Usenko O. Y., [1]Todurov I. M., [1]Kosiukhno S. V., [1]Pylypchuk O. O.

RECONSTRUCTIVE DUODENOENTEROPLASTY IN PATIENTS AFTER STRANGULATED INTERNAL HERNIA AFTER REVISIONAL ROUX-EN-Y GASTRIC BYPASS (CLINICAL CASE)

[1]SSI "CIMT of the NAS of Ukraine" (Kyiv, Ukraine)
[2]State Institute "Shalimov`s National Institute of Surgery and Transplantation" of National Academy of Medical Sciences of Ukraine (Kyiv, Ukraine)
o.plpchk99@gmail.com

Bariatric surgery is the most effective method of treatment for obesity and related metabolic disorders. Bariatric surgery leads to a sustainable loss of excess body weight and compensation for comorbidities associated with obesity, primarily type 2 diabetes and cardiovascular diseases, which generally leads to a reduction in mortality. Given the growing interest in bariatric surgery, the diagnosis and surgical correction of complications after revisional operations remains an urgent challenge for surgeons, especially in the context of overweight relapse. One of the most serious and potentially life-threatening complications for patients is a strangulated internal hernia.

The surgical approach to the correction of strangulated internal hernias is quite diverse and depends on the length of the strangulated loop and its viability. On an emergency basis, in this category of bariatric patients, the surgical intervention for this complication usually implies obstructive resection of a section of the small bowel.

The use of the reconstructive duodenoenteroplasty technique allowed to restore the physiological passage of food and enzymes through the gastrointestinal tract. Normal functioning of the gastrointestinal tract after reconstruction allowed to stabilise weight, restore normal blood biochemical parameters and, as a result, improve the quality of life.

Key words: *reconstructive duodenojejunal plication, Roux-en-Y gastric bypass surgery, obstructive resection, morbid obesity, recurrence of overweight, bariatric surgery.*

Connection of the publication with planned research works.

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Introduction.

Morbid obesity is a widely recognised public health problem. Following a National Institutes of Health (NIH) conference convened in 1991, surgical approaches were recognised as the most appropriate treatment for patients with clinically significant obesity, with a body