and more then 10 years diabetes duration group. ADMA concentration was determined by ELISA method and ADMA®-ELISA kit (DLD Diagnostika GmbH, Hamburg, Germany) was used as reagent. For the comparisons between groups, ANOVA test was used.

Results. Study included 60 DMT2 patients (30 male, 30 female) and 60 apparently healthy control subjects (30 male, 30 female). Serum ADMA concentration was significantly higher in DMT2 patients with diabetes duration more then 10 years (1,81±0,12 μ mol/L) compared to serum ADMA concentration in DMT2 patients with diabetes duration up to 10 years (1,38±0,08 μ mol/L; p<0.001) and compared to serum ADMA concentration in the control group of subjects (0,62±0,02 μ mol/L; p<0.001).

Conclusion: Obtained results suggest an increase in serum AMDA concentration with the progression of DMT2 duration. Since ADMA is a marker of endothelial dysfunction that contributes to DMT2 vascular complications development, determination of serum ADMA levels in different stages of DMT2 duration could assist in prevention and treatment of endothelial dysfunction in DMT2.

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CLINICAL ANALYSIS OF TREATMENT RESULTS FOR IMMUNOHISTOCHEMICAL MARKERS

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Cancer of ENT organs are on the seventh place in the world in prevalence. Combined or complex treatment were given to 35% of patients and the results are considered unsatisfactory. The questions of the clinic, diagnosis, prognosis, prevention of such patients are widely represented in the literature of different years (Garyuk G.I., 1997; Popovich V.I., 1999; Timchuk S.M., 1999; J.Shah, 2000; Paches A.I., 2000; Reshetov I.V., 2005, Protsik V.S., 2006; Palamarchuk V.V., 2008; Kashirin V.O., 2009). However, thoughts about the effectiveness of different treatment methods diverge, the issue of prognosis of the course of the tumor process after radical treatment methods is not developed.

Materials and Methods. The study is based on an analysis of the treatment and monitoring results of 90 patients with squamous cell carcinoma of the oropharynx and larynx from the second to third stage of the process. The patients did not differ by sex, age, localization of the primary tumor, the form of its growth, the stage of the disease, the duration of the disease, neglect. Patients received chemo-radiation therapy according to the proposed scheme. The determination of the levels of expression of the protein bands was used as differential diagnostic criteria.

Results. The analysis of the results of the relapse and prognosis of the oncological process, depending on the expression of oncological markers mp53, Bcl-2, Kl-67, was carried out. Analysis of the relapse of the disease depending on the expression of KI-67 oncoprotein: in year I 26 (46.43%) of patients died with expression of $56.8 \pm 2.6\%$. Il year: 22 (39.29%) patients died with an expression of 58.8 ± 2.7%. In the third year, 2 (3.57%) died with expression of 54.0%. For three years 5 patients survived (8.93%) with 45.0% expression. Summing up: with expression above 55% recurrence occurred in 1-2 years of observation. Tumor regression: a complete response in 6 (10.71%) patients with expression 54.3 ± 3.2%. Partial response was in 5 (8.93%) patients with expression 51.4 ± 5.04%. Stabilization in 8 (14.29%) patients with expression was 51.9 ± 3.0%. Progression at 37 (66.07%) with expression 59.9 ± 2.9%. Positive effects of treatment: complete response, partial, stabilization in patients where expression is > 55.0%, and $\square 60.0\%$ is progression. Investigation of relapse depending on the expression of the mark mp53. Most patients had a relapse for the 1st year after treatment: 26 (46.43%) with mp53 expression $61.2 \pm 5.5\%$. In the 2nd year - 22 (39.29%) patients with an expression of $59.4 \pm 1.5\%$. In the 3^{rd} year, relapse in 2 (3.57%) patients with expression was 64.0%. For the third year 5 (8.93%) patients survived with 54.8 ± 4.4%. Early relapse in patients with mp53 53.0%, and with an incidence below 55.0%, recurrence is late (after 3 years). Tumor regression: the complete response in 6 (10.71%) with the expression of mp53 56.70 \pm 3.3%, partial in 5 (8.93%) with the expression of mp53 59.4 \pm 1.8%. Stabilization: 8 (14.29%) of patients with mp53 expression - 57.6 ± 3.3%. Progression of the process in 37 (66.07%) with expression mp53 - 62.5 ± 1.9%. Full response and stabilization of the process for expression mp53 53.2%. Partial response and progress of the process with mp53 - 60,95%. It is assumed that the express of mp53 changes and depending on the effectiveness of neoadjuvant treatment. Expression of Bcl-2 was studied: And the year of supervision there was a recurrence in 26 (46.43%) patients with expression of Bcl-2 18.8 ± 1.1%. The second year added relapse of 22 (39.29%) to patients with an expression of 21.3 \pm 1.2%. In the 3rd year - 2 (3,57%) cases with Bcl-2 - 23,8 \pm 0,5%. The expression level of Bcl-2 probably did not affect the reciprocal activity. Partial in 5 (8.93%) for expression of Bcl-2 19.8 ± 1.4%; Stabilization of 8 (14.29%) with Bcl-2 19.5 \pm 1.4%; progression in 37 (66.07%) with expression in 19.6 \pm 1.1%. The high expression of Bcl-2 (\square 20.61%) is a vivid answer to the question. The indicator below this figure gives the progression of the process and the negative result of treatment. Positive BcI-2 - the probability of development of reccurance in the first year, low sensitivity to conducting chemo-radio therapy.

Prospects for further research: the lack of common medical protocols, the inability to predict the course of the tumor process give grounds for further work in this direction.

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DIAGNOSTIC MARKERS OF SYSTEMIC INFLAMMATION IN CORONARY HEART DISEASE IN COMBINATION WITH NON-ALCOHOLIC FATTY LIVER DISEASE

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According to the available research, coronary heart disease (CHD) ranks first among the causes of mortality in the world. In Ukraine, about 350 thousand people annually die of CHD [1]. The main pathogenetic cause of CHD is atherosclerosis (AS) of vessels, which is accompanied by disruption of lipid metabolism. It is hepatocytes that take part in the maintenance of lipid homeostasis. Thus, functional changes in the liver contribute to the potentiation of dyslipidemia (DL) and, accordingly, the development of coronary heart disease. The question about formation of fatty liver infiltration, namely, non-alcoholic fatty liver disease (NAFLD) (steatohepatosis) is also very relevant. NAFLD is a stage-by-stage process involving benign fatty liver infiltration with subsequent development of steatohepatitis, hepatocellular carcinoma and liver cirrhosis [2].

Recent scientific researches have shown that it is chronic systemic inflammation (CSI) that plays a leading role in the onset and progression of AS [7], the pathogenetic basis of which is endothelial dysfunction (ED) and DL that causes oxidative stress due to the formation of products of lipid peroxidation (LPO) [3]. Meanwhile, the nuclear factor kappa B (NF-kB) is the key link of CSI, which triggers the synthesis of proinflammatory cytokines (CK): interleukin 6 (IL-6) and tumor necrosis factor a (TNFa) [3, 4]. The foregoing provides the basis for searching the indicative markers of CSI in this combined pathology to optimize diagnostics and assessment of the course of the disease, as well as to ensure the effectiveness of therapeutic measures. The aim of the research was to analyze the relationship between the parameters of CSI, endothelial dysfunction, lipid spectrum, antioxidant defense and blood flow rate in the portal and hepatic veins in order to detect important diagnostic markers of CHD concurrent with NAFLD.

Materials and methods. An open clinical trial (single group study) was conducted. The study involved 62 subjects of both sexes with the diagnosis of coronary heart disease: stable exertional angina, FC I-II, HF 0-I concurrent with NAFLD. The inclusion criteria for the study were the age of 40-70 years, the presence of coronary heart disease: exertional angina of FC I-II in the absence of the course destabilization for at least two months, as well as the presence of concomitant NAFLD (steatohepatosis), patient's informed consent to participate in the study. The exclusion criteria were the presence of hypertension above the stage II, unstable angina, chronic heart failure (CHF) above stage I, diabetes mellitus type I, chronic and acute viral hepatitis, alcoholic liver disease, autoimmune hepatitis, Wilson-Konovalov's disease, rheumatic diseases, anemia, chronic renal failure, oncological diseases. To achieve this aim, patients were tested for blood levels of IL-6 and TNFa by the immunoenzyme method, the number of circulating endothelial microparticles (CEM) in peripheral blood with expression of CD32 and CD40 antigens by flow cytometry [9], total cholesterol (CH) and low density lipoprotein cholesterol (LDL cholesterol) by sedimentation method; expression of the mRNA gene of the kappa inhibitor Ba (IkBa) of the nuclear transcription factor kappa B (NF-kB) in peripheral blood mononuclear cells by the polymerase chain reaction method, the level of ceruloplasmin (CP) by biochemical method. The determination of blood flow rates in the portal vein (v.p.) and hepatic veins (v.h.) was carried out by ultrasonic pulsed dopplerography.

Results. Patients in the study group displayed increased proinflammatory CK TNFa -10.7 pg/ml, CD32 CD40 - 2.69 x 10^7 / I at the rate of 1.3 (1.05-2.11) x 10^7 / I [5], blood flow rate in the portal vein (v.p.) - 0.386 m / s and hepatic veins (v.h.) - 0.195 m / s.As a result of correlation analysis, a direct relationship was found between IkBa NF-kB and TNFa (r = 0.365, p <0.05), as well as between IkBa and IL-6 (r = 0.381, p<0.05), which demonstrates the key role of IkBa NF-kB in CSI. A direct correlation was found between IkBa and total cholesterol (r = 0.494, p<0.01), as well as between IkBa and LDL cholesterol (r = 0.462, p<0.01), thus demonstrating the relationship between dislipoproteinemia and CSI in this category of patients. Blood flow rate in v.p. had a direct correlation with IkBa (r = 0.597, p <0.001) and IL-6 (r = 0.534, p <0.001). We also detected a direct correlation between the blood flow rate in v.h. and CD32 CD40 (r =