Conclusions. The study showed that the expression of matrix metalloproteinases is not typical for the normal oral mucosa. Further studies are aimed at studying the expression of matrix metalloproteinases in pathologically altered mucosa with dysplastic and neoplastic changes. Because of the unclear pathogenesis of some diseases of the oral mucosa their management remains mostly symptomatic treatment. For that reason, understanding of the underlying disease pathways and identifying specific mediators remains an actual direction of research.

References


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FEATURES OF MATRIX METALLOPROTEINASE -7, -8, -13, -14 EXPRESSION IN DIFFERENT TYPES OF PERIODONTITIS

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Periodontal diseases represent a numerous and diverse group of diseases in both clinical and morphological manifestations. There is still need in studying of clear clinical and radiological signs of diagnosis of periodontitis. Further researchers of biomolecular markers in determining the prognosis of periodontitis at the early stages of the disease are in the focus of interest. Objective: to study features of metalloproteinases (MMP)-7, -8, -13, -14 expression in patients dependent upon the type of periodontitis.

Materials and Methods. Biopsy of gingival material was analyzed from 39 patients with aggressive (AP, n=13), chronic simplex (CSP, n=5), chronic complex (CCP, n=12) periodontitis, and a control group (n=9). Morphometric analysis of the MMPs expression was performed using Aperio Image Scope v9.0 software. Spearman and U-test was applied, p<0.05.

Results. Expression of MMP-7, -8, -13, -14 in the biopsy material was obtained in all cases of patients with different forms of periodontitis from mild to severe with a predominance of expression in the stromal component, and to a lesser extent the involvement of the gingival epithelium (except MMP-13, expression of which was detected only in the lamina propria). In the control group, the expression of the studied MMPs was absent or was represented by focal weak expression, the indices of which were significantly lower than in the groups of patients with different forms of periodontitis (p=0.002). The direct correlation between stromal expression of the studied MMPs (p=0.68, p=0.59, p=0.77 for MMP-7, MMP-8, MMP-14, respectively, p<0.05) with the epithelial expression of appropriate MMPs and severity of inflammation, and epithelial expression of MMPs with the severity of interepithelial infiltration of leukocytes were revealed. Also, a correlation between the stromal expression of MMP-14 with that of MMP-13 and MMP-7 (p=0.52 and p=0.56, respectively) was found. The “hot point” analysis of the MMPs expression revealed significantly lower levels of stromal expression of MMP-14 and MMP-7 (U=12.5 p=0.033 and U=30.5; p=0.02) in the group of the AP in comparison with the CSP and CCP, respectively, and higher stromal expression of MMP-8 (U=18 p=0.001) in the AP group compared to the CCP.

Conclusions. The expression pattern and indices of the studied MMPs are interrelated both with each other and with the degree of inflammation and can be considered
as informative indicators for differential diagnosis and determination of character of periodontal disease course at the manifestation stage of the disease.

INFLUENCE OF BETARGIN AND QUERCETIN ON INDICATORS OF CHRONIC SYSTEMIC INFLAMMATION IN PATIENTS WITH CORONARY HEART DISEASE CONCURRENT WITH NON-ALCOHOLIC FATTY LIVER DISEASE

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Comorbidity is one of the major issues of modern medicine, characterized by masking of pathological conditions, aggravated course and unfavorable prognosis [1].

Recently, there is a significant increase in the incidence of coronary heart disease (CHD) and non-alcoholic fatty liver disease (NAFLD) all over the world [2]. Formation of both CHD and NAFLD is associated with the intensity of chronic systemic inflammation (CSI), dyslipidemia, endothelial dysfunction, and oxidative stress [3, 4]. The detection of the leading role of low-intensity CSI in the pathogenesis of both pathological conditions underlies the scientific searches for effective and well-founded therapeutic approaches under conditions of comorbidity [5]. The aim of our research was to study the indicators of chronic systemic inflammation in patients with stable ischemic heart disease concurrent with non-alcoholic fatty liver disease and the effect of comprehensive therapy with the addition of betaine, arginine and quercetin on the detected disorders.

Materials and Methods. The study included 75 patients of both sexes, aged from 40 to 69, with the diagnosis of coronary heart disease: stable exertional angina, FC II, HF 0-I, NAFLD. Patients were divided by random sampling into 2 groups – the study group (n=27) and the comparison group (n=48). Patients were examined to determine levels of cytokines (CK) – tumor necrosis factor alpha (TNFα), interleukin-6 (IL-6) and interleukin-10 (IL-10) in the serum by immunoenzymatic method, fibrinogen content (FG) in the blood plasma by weight method, and the expression level of mRNA gene of inhibitor kappa B alpha (IkBα) of nuclear factor-kappa B (NF-kB) in mononuclear cells by PCR in real-time mode [6, 7]. All patients are prescribed standard therapy for stable coronary heart disease (β-blockers, statins, aspirin), as well as silymarin (90 mg per day) and lecithin (1200 mg per day) for NAFLD correction. Patients in the study group were additionally prescribed betargin at a dose of 2 g of arginine citrate and 2 g of betaine daily per os, as well as quercetin at a dose of 120 mg daily per os. In 2 months, patients were re-examined to the aforementioned extent of studies.

Results. All patients with coronary heart disease had elevated blood levels of TNFα (10.56±3.74 pg / ml). The levels of proinflammatory CK IL-6 and anti-inflammatory CK IL-10 were within the physiological norm and amounted to 4.69±1.21 pg / ml and 11.43±2.12 pg / ml, respectively; the FG content in the blood plasma was moderately elevated (4.65±1.04 g / l). We studied the expression level of mRNA gene of IkBα in mononuclear cells, reflecting the level of transcriptional activity of NF-kB, and, accordingly, the severity of systemic inflammation in patients with stable coronary heart disease concurrent with NAFLD, which was 0.215±0.015 c.u. Two months after initiation of treatment, a reliable decrease in TNFα levels was observed in the study group by 55.6% (p<0.001), in the comparison group – by 34.5% (p<0.01). The level of IL-6 was reliably decreased only in the study group (p>0.05). The level of anti-inflammatory CK IL-10 under the influence of therapy was significantly increased only in the