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EVALUATION OF METABOLIC AND HUMORAL BLOOD FACTORS IN PATIENTS WITH CHRONIC GENERALIZED PERIODONTITIS ASSOCIATED METABOLIC SYNDROME

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ABSTRACT

The aim of this study is to evaluate the characteristics of indicators of carbohydrate, lipid metabolism and humoral factors of immunity in the blood plasma in patients with chronic generalized periodontitis (CGP) of moderate degree of combined background metabolic syndrome. The study was conducted in 58 patients with chronic generalized periodontitis of moderate severity combined metabolic syndrome aged 35 to 63 years, as well as 14 volunteers (practically healthy people) with intact periodontal age 25–35 years, who formed the control group (CG). It was revealed that in patients with MS combined with inflammation in periodontal tissues on the background of hyperinsulinemia and dyslipidemia, marked changes in the indices of cellular and humoral immunity are observed. The marked increase in IgG in the blood against the background of a decrease in IgA, in our opinion, is due to increased synthesis of secretory IgA in the oral fluid.

KEYWORDS: Chronic generalized periodontitis; metabolic syndrome; inflammation.

INTRODUCTION

The actual problem of modern dentistry today is the problem of the influence of systemic disorders characteristic of the metabolic syndrome on the organs and tissues of the oral cavity. Numerous studies have shown that among patients with metabolic disorders (metabolic syndrome, diabetes, systemic lupus erythematosus) inflammatory diseases of the periodontal complex.[1,2,10,11] are very common. Parodont, its structures are sensitive to the pathogenic effect of the factors that form the proatherogenic spectrum of metabolic disorders.[5,6] The commonness of inflammation and pro-atherogenic metabolic disorders from a pathophysiological point of view is quite natural, since both syndromes form the same cells: endothelial and smooth muscle cells, fibroblasts, monocytes and macrophages, neutrophils, platelets, and to a lesser extent, T and B lymphocytes.[3,4,7,8] In inflammation and metabolic syndrome, the adhesion (fixation) of monocytes and neutrophils on the surface of the endothelium activates the same proteins of cell interactions: integrins on the membrane of neutrophils and monocytes, E-selectin on the membrane of the endothelium and P-selectin - platelets. In both syndromes, active infiltration (chemotaxis) of tissues by monocytes and neutrophils circulating in the blood occurs.[9,14] In both situations, activated neutrophils and tissue macrophages in the "respiratory blast" reaction

intensify the formation of superoxide radicals and activate peroxidation of proteins and lipids, causing alteration of normal tissues.[12,13] On this basis, the study of the clinical manifestations of periodontitis in the initial stage of the development of the metabolic syndrome for the timely and successful conduct of pathogenetic therapy should be recognized as relevant and necessary.

The purpose of this study: was to evaluate the parameters of carbohydrate, lipid metabolism and humoral immunity factors in the blood plasma of patients with chronic generalized periodontitis (CGP) of moderate degree combined with the background of metabolic syndrome.

MATERIALS AND RESEARCH METHODS

We carried out a comprehensive survey of 58 patients with chronic generalized periodontitis of moderate severity at the age of 35 to 63 years, as well as 14 volunteers (almost healthy people) with an intact periodontal at the age of 25–35 years who formed the control group (CG). In the experimental group, in accordance with the criteria for the diagnosis of "metabolic syndrome", in 100% of cases the central type of obesity was determined (the average value of BMI was 36.1+2.9). 31 % (18 people) patients were diagnosed with 1 degree of obesity, 50% (29 people) - 2 degree, and

19 (11 people) - 3 degree. In the control group, there were patients without abdominal obesity (the average value of BMI was 23.2+0.9). The study of the dental status of patients included a survey and examination. During the survey, the following indices were determined: Green hygiene index - Vermillion (I964), PMA, PI by A. Russel (1967), IC by Muchlemann (1971). X-ray examination was carried out using orthopantomograms and targeted intraoral images. The glucose content was determined in capillary blood on an empty stomach and after glucose loading on a biochemical analyzer using Lachema reagents. The content of insulin in the serum of venous blood using standard sets of the company "HUMAN". The concentration of C-peptide was determined by ELISA that using sets of reagents of the company BioChemMak. The criteria for hyperinsulinemia were considered the fasting insulin level of more than 12.5 MCU/ml and above, 2 hours after the glucose load - 28.5 MCU/ml and above. The level of C-peptide was considered elevated at a basal concentration of more than 3.6 ng/ml and stimulated - 4.2 ng/ml. The level of low-density lipoprotein (LDL) was calculated by the difference between the concentration of total cholesterol (TC) and LDL.

LDL = TC - (HDL+VLDL), where VLDL is a very low-density lipoprotein. The HDL level was determined by the formula PONP = TG Π 0.46. To calculate the coefficient of atherogenicity (CA) used the formula.

CA = (LDL+VLDLP) / HDL.

The total cholesterol content was taken as the norm - 3.5–5.2 mmol / l, LDLAP - 0.04–0.35 mmol/l, LDL - 2.6–3.6 mmol/l, HDL - 0.91–1.95 mmol/l. The state of general immunity was assessed by the number of T- and B-lymphocytes, the content of IgA, IgG, and IgM in peripheral venous blood using an enzyme immunoassay analyzer from ROSH.

RESEARCH RESULTS AND DISCUSSION

When examining patients, the following complaints were noted: bleeding of the gums during tooth brushing and solid food intake, itching and discomfort in the gums, bad breath, discoloration of the gums, tooth mobility. During an objective examination, the gingival papillae and marginal gum were swollen, cyanotic, and bled during probing. The depth of the periodontal pocket reached 2–3 mm. Pathological mobility was determined within the I – II degree. During radiological examination in patients with moderate CGP, predominance of the vertical type of bone tissue resorption, the absence of a compact plate and destruction of the interalveolar septum from 1/3 to 1/2 of the root length was observed, which corresponds to the second degree of destruction of the bone tissue of the alveolar jaw. The hygiene index was very high; its average value was 2.52 ± 0.25 points, which indicates poor oral hygiene. The PMA index was 40.87 ± 2.64%, which indicates that these groups of

patients have an inflammatory process in the periodontal complex. The average value of PI in the I-th group was 4.96 ± 0.21 points. The bleeding index is 2.19±0.1 4 points. The survey showed that patients had lesions of periodontal tissues corresponding to a moderate degree of periodontitis, clinically manifested by symptomatic gingivitis and bleeding phenomena.

According to the results of laboratory data, the fasting blood glucose level in all patients was within the normal range: 4.2±0.3 mmol / l in the control group, 6.1±0.24 mmol/l in patients with MS. But 2 hours after glucose loading, their level was different and amounted to 4.85±0.24 mmol/l in the control group and 5.95±0.24 mmol/l (p<0.001) in patients with MS. The insulin level was within the normal range, although MS patients had higher than the control group: 14.8±0.9 and 10.1±1.2 mC/ml, respectively (p <0.001). The level of stimulated insulin 2 hours after the glucose load in the control group of patients remained within the normal range: 17.2±1.4 µEU/ml, and in patients with MS it increased by 3 times compared with the control group - 49.9 ± 3.46 MCED/ml, indicating that hyperinsulinemia. An elevated level of insulin contributes to the delay in the body sodium, which leads to hypervolemia and vasoconstriction of blood vessels, increased platelet aggregation. In this case, adipose tissue is characterized by excessive lipolysis, impaired circulation of lipoproteins, glucose, which leads to increased hyperinsulinemia, dyslipidemia. The level of stimulated C-peptide in patients with chronic periodontitis on the background of MS exceeded the level of the control group by 39% p <0.01. The level of TC in all patients with MS naturally exceeded the control group and was 6.2±0.4 mmol/l. When analyzing the blood lipid spectrum, a regular increase in total cholesterol was noted. This was due to an increase in the level of LDL to 4.1±0.4 mmol/l (p<0.05). At the same time, there was a decrease in HDL to 0.9±0.1 mmol/l. The coefficient of causation (CA) was 5.1 ± 0.3, which is 41% higher than the control group - 3.7±0.3. High concentrations of free fatty acids formed on the background of dyslipidemia inhibit insulin absorption by the liver, which leads to hyperinsulinemia and relative insulin resistance. At the same time, free fatty acids inhibit key enzymes - pyruvate dehydrogenase and phosphokinase, reducing glucose oxidation (Rendl cycle), and participate in the mechanism of maintaining insulin resistance. The state of general immunity was assessed by the number of T and B lymphocytes, the content of IgA, IgG, IgM in peripheral venous blood. When analyzing the immunograms of patients with chronic generalized periodontitis on the background of MS, significant immune changes were recorded compared with those in the control group. Thus, the content of T-lymphocytes was significantly reduced and amounted to 41.2±2.21% (control 58.6±1.36%) (p<0.05). The insufficiency of the T-system of immunity is manifested by a chronic prolonged course, inflammatory-destructive process in the periodontium. Analysis of humoral immunity in

patients with chronic generalized periodontitis revealed a statistically significant increase in the number of B-lymphocytes by 1.7 times as compared with the control group and was $40.9 \pm 1.40\%$ (control $24.11 \pm 0.65\%$) ($p < 0.05$). These results indicate a pronounced activation of the B-cell component of the immune system in patients with moderate CGP, especially when combined with the metabolic syndrome. The concentration of Ig classes G, A in the serum of patients with metabolic syndrome was significantly higher than in the control group. The content of IgG was especially high: 1876.0 ± 9.61 mg/dl (control 1156.0 ± 0.18 mg/dl) ($p < 0.05$), the amount of which always increases with the chronic form of inflammation. The amount of IgA was 98.7 ± 7.31 mg / dl (control 150.8 ± 9.14 mg/dl) ($p < 0.05$). In the dynamics of IgM concentration in the blood, no significant changes were noted.

FINDINGS

1. In patients with MS combined with inflammation in periodontal tissues on the background of hyperinsulinemia and dyslipidemia, marked changes in the indices of cellular and humoral immunity were established.

2. Against the background of MS, an increase in IgG was observed in patients with CGP, with a decrease in IgA, which is apparently due to an increased synthesis of secretory IgA.

REFERENCES

1. Kirsanov A.I., Orekhova L. Yu. Somatic aspects of the pathogenesis of generalized periodontitis // Dentistry, 2001; 1: 26–34.
2. Sarah G. Grossi. Inflammatory processes in the oral cavity and cardiovascular diseases // Clinical dentistry, 2006; 1: 40–44.
3. Starikov I.V. Efficiency of using efferent detoxification methods in the complex treatment of chronic generalized periodontitis in patients with metabolic syndrome: author. dis. Cand. Med. sciences. - Volgograd, 2009; 31.
4. Alekseeva O.A. The role of the correction of general and local munus status and the biochemical parameters of oral fluid in the complex therapy of periodontitis for diabetes mellitus: author. dis. Cand. Med. sciences. - Ryazan, 2001; 21.
5. Ohanyan E.S. Periodontal status in patients with insulin-dependent diabetes mellitus (clinical and laboratory research): Author's abstract. dis. Cand. Med. St. Petersburg, 2001; 18.
6. Shamolina E.A. Assessment of the microcirculation in arterial hypertension with metabolic disorders: author. dis. Cand. Med. sciences. –Ivanovo, 2005; 109.
7. Alberti K.G., Zimmet P.Z. for the WHO Consultation. Definition, diagnosis, andclassification of diabetes mellitus and its complications, part I: diagnosis and classificationof diabetes mellitus: provisional report of a WHO consultation // Diabet Med, 1998; 15: 539–553.
8. D'Aiuto F., Sabbah W., Netuveli G. et al. Association of the metabolic syndromeьwith severe periodontitis in a large U.S. population-based survey // J. Clin. Endocrinol.Metab, 2008; 10(93): 3989–3994.
9. Shokhsanam Khurramova & Bekhzod Abdullaev, "Development of Periodontitis after Antihelicobacter Therapy in Patients with Ulcer Disease," Journal of Diseases, Conscientia Beam, 2017; 4(1): 1-7.
10. Han D.H., Lim S.Y., Sun B.C. et al. The association of metabolic syndrome withperiodontal disease is confounded by age and smoking in a Korean population: the Shiwha-Banwol Environmental Health Study // J. ClinPeriodontol, 2010; 7(37): 609–616.
11. Khader Y., Khassawneh B., Obeidat B. et al. Periodontal status of patients withmetabolic syndrome compared to those without metabolic syndrome // J. Periodontol, 2008; 79(11): 2048–2053.
12. Kowalski M., Brocka E., Barylski M. et al. Assessment of the periodontal state insubjects with metabolic syndrome // Pol. Merkur. Lekarski, 2009; 26(156): 620–625.
13. Shimazaki Y., Saito T., Yonemoto K. et al. Relationship of metabolic syndrome toperiodontal disease in Japanese women: the Hisayama Study // J. Dent. Res., 2007; 863: 271–275.
14. Timonen P., Niskanen M., Suominen-Taipale L. et al. Metabolic syndrome, periodontalinfection, and dental caries // J. Dent. Res., 2010; 10(89): 1068–1073.