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THE SPECIAL FEATURES OF COMPREHENSIVE TREATMENT OF PATIENTS WITH GENERALIZED PARODONTITIS IN THE BACKGROUND OF CORONARY HEART DISEASE

ODMIENNOŚCI KOMPLEKSOWEGO LECZENIA PACJENTÓW Z UOGÓLNIONĄ PARODONTOZĄ I CHOROBĄ NIEDOKRWIENNĄ SERCA

Tatyana O. Petrushanko, Ivan Y. Popovych, Olga M. Bojchenko
STATE HIGHER EDUCATIONAL INSTITUTION IN UKRAINE, UKRAINIAN MEDICAL DENTAL ACADEMY, POLTAVA, UKRAINE

ABSTRACT

Introduction: According to official statistics, the incidence of coronary artery disease in the working-age population in 2015 is registered in 9.7 per 100 thousand of the population; a significant number of the patients with IHD are the elderly, although there is also a "rejuvenation" of this pathology. Ischemic heart disease tends to occupy a top position amongst the causes of temporary and permanent disability, leading to the complete disability of the population. Numerous clinical and laboratory studies revealed a direct interconnection between the severity of the course of generalized periodontitis and the functional class of stable angina pectoris.

The aim of my study was to determine effective comprehensive treatment for the patient with chronical generalized paradontitis, associated with ischemic heart disease.

Materials and methods: The examination and treatment of 42 subjects with chronic generalized periodontitis I-II grade at the age from 45 to 60 years, who suffered from coronary heart disease, with a mean duration of 1.5 to 5 years, was performed.Clinical studies have revealed that in the patients, suffering from coronary artery disease, during the period of in-patient treatment, predominantly generalized periodontitis takes place at the stage of exacerbation (89.5%).

Results and conclusions: Exacerbation of HGP I and II in the patients, suffering from stable angina pectoris, is accompanied by significant disorders of metabolic processes; changes of the condition of regional hemodynamics and microcirculation in periodontal tissues, which is proven by alteration in reopardotography rates. It is confirmed that while the administration of antihypoxic therapy in the comprehensive treatment of patients with CGP, suffering from coronary heart disease, it is advisable to take into account the sequence of violations of bioenergetic processes that begin on the substrate region of the respiratory chain with a violation of the ICFA I and extend with the increase of oxygen insufficiency to the terminal cytochrome oxidase site. On the basis of this, the protection of the body from oxygen deficiency with the help of antihypoxic drugs provides, as a mandatory condition, the restoration of the energy-absorbing function. The administration, as part of integrated therapy, of local antihypoxic and coenzyme Q_{10} in patients with coronary heart disease is accompanied by a significant improvement in the clinical manifestation of subjective and objective characteristics; positive dynamics of periodontal indicators; correction of antioxidant and nitroxydergic systems, which manifested in increasing of the antioxidant potential in the oral liquid as well as optimization of processes of regional hemodynamics and microcirculation.

KEY WORLDS: generalized parodontitis, $CoQ_{10,}$ ischemic heart disease, antihypoxic drugs

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INTRODUCTION

One of the most important tasks of the state politics in the field of Ukrainian health services is the prevention of cardiovascular diseases, the reason for the increased level of mortality. Thus, the number of such cases makes up over 78% of all deaths in Ukraine. Ischemic heart disease (CHD) is one of the main factors affecting the level of mortality in the USA, Europe, and Ukraine isn`t an exception in this list. According to the official statistics, the incidence of CHD amongst individuals of the working age in 2015 was 9,7 in 100.000 of the population, where the vast majority of patients with CHD is represented by the elderly though there is also a «rejuvenation» of this pathology. Ischemic heart disease occupies a leading place mid the reasons of temporary and persistent disability; thereby, this is the cause of consequent and complete disability[1]. Moreover, a close affiliation of periodontal diseases with the pathology of the cardiovascular system has recently been proven[2,3]. It's alleged that inflammatory periodontal diseases interconnected with coronary artery disease, as there is one and the same root for both nosologies, namely, the immune inflammatory process in the endothelium that triggers both pathologies, the complications from which in the area of coronary vessels may be one of the key reasons of the high mortality in the world[4].

According to N.L. Kuznetzova and her co-authors, the incidence of chronical generalized parodontitis (CGP) between the patients suffering from CHD, including those

ones who underwent myocardial infarction (MI), constituted 82,4 and 72,2%, accordingly[5]. During the last decade, scientific and practical interests toward this domain of research has increased significantly in the connection with the emergence of «a single theory of atherogenesis" that consider atherosclerosis, the main contributing factor in the development of CHD, as an immune inflammatory process[6,7]. The ostended way of influence of paradontal inflammatory diseases on atherogenesis affiliated with the fact that the gum and paradontal pockets, the reservoir of microorganisms, release bacterial components (endotoxins) into the bloodstream, which indirectly, with the help of proinflammatory cytokines and other mediators of inflammation, produced by the cells-respondents, lead to the alteration of the endothelium of vessels, hyperlipidemia and lipid infiltration of the vascular wall as well as the stimulation and support of inflammatory response. Thus, the atherogenesis is launched and maintained[8].

Chronic inflammatory disorders of the oral cavity may increase the risk of development and progress of cardiovascular diseases, caused by atherosclerotic changes in the vessels. Numerous scientific studies witness that there is 1,3-2 times higher risk of development of CHD in the patients with CGP, in comparison with the general population rate. The possibility of the development of coronary heart disease in the individuals suffering from paradontitis, taking into account listed risk factors, rises in 6,5 times, in comparison with the healthy population, and the increase in the risk depends on the severity of the process accordingly[9].

The general rule for all types of inflammation is the elevation of free radical oxidation (FRO) on the background of the decrease in the activity of the body physiological antioxidant (AO) system[10]. One of the main causes of activation of FRO in various pathological processes is the severe hypoxia of the body tissues, which occurs due to the violation of the ability of these tissues to absorb oxygen from the blood or due to the impairment of the efficiency of enzymatic oxidation[11]. The utilization of oxygen by the body tissues can be violated as a result of inhibition of biological oxidation by various inhibitors, a violation of the synthesis of enzymes or consequent damage to the cell structures[12].

The leading domains in the pathogenesis of HGP are associated with violations of periodontal nourishment due to the impairment of functional and organic microcirculation, the imbalance of the nervous and hormonal regulations, disfunction of the immune systems and the barrier systems[13]. The hypoxia of the periodontal tissues, which occurs as a result of those disorders and the pathogenic consequences of emotional pain, focused on these structures due to their rich vascularization and innervation, are activated by FRO[14]. Intensification of the latter aspect may be considered as a mechanism of the body adaptation to the altered level of oxygen in the tissues. The outcome of FRO is the formation of active forms of oxygen (AFO) - singlet oxygen (* O_2), superoxide anion radical (.O), hydrogen peroxide (H_2O_2), hydroxyl radical (OH), nitric oxide radical (NO)[15]. According to the modern hypothesis, hypoxia that takes place in the body owing to an inefficient supply of the tissues and organs with oxygen is a phased process, accompanied by the dysfunction of mitochondrial enzyme complexes (MFCF), which leads to the inhibition of aerobic energy synthesis, energy-dependent functions, and cell metabolism, respectively. The violation of the energy-absorbing function of mitochondria, accompanied by a decrease in oxygen consumption, is considered as tissue (bioenergetic) hypoxia. Therefore, protection and prevention from hypoxia and its consequences is the primary task of the modern medicine[16].

THE AIM

The purpose of my study was to determine effective comprehensive treatment for the patient with chronical generalized paradontitis, associated with ischemic heart disease (IHD).

MATERIALS AND METHODS

The examination and treatment of 42 subjects with chronic generalized periodontitis I-II grade at the age from 45 to 60 years, who suffered from coronary heart disease, with a mean duration of 1.5 to 5 years, was performed. The establishment and verification of the diagnosis of coronary artery disease in subjects were conducted by a cardiologist of in-patient hospital. The dental status of the patients was evaluated by a dentist and recorded in the history of the illness of the in-patient patients. The patients over 65 years old; those with myocardial infarction in history,which took place less than 6 months ago; those with insulin-dependent diabetes mellitus; those with current malignant neoplasms, and the patients with full adentia were excluded from our study.

In the first (control) group of subjects (20 persons) included patients with CGP I-II grades with no history of CHD, and those ones, who received treatment under the Protocols - standards for dental patients treatment.

The second group consisted of subjects (20 persons) with coronary artery disease with stable angina pectoris functional class (f.c.) I, CHD, f.c. II NYHA (moderate chronic heart failure) with the preserved systolic left ventricular function with chronic generalized periodontitis I-II grades; cardiologic indications and a cardiologist's recommendation for the antihypoxant 3-hydroxy-6-methyl-2-ethylpyridine succinate (in capsules 0.3-0.6 g per day) and locally injected into periodontal pockets by instillating 5% solution and applying a hardening bandage (5% solution of 3-hydroxy-6-methyl-2-ethylpyridine succinate - 2 ml , zinc oxide and dentin powder by 3.5 g, lanolin - 1.0 g) on the vestibular and oral side of the alveolar processes of the upper and lower jaw. The hardening bandage was kept on the surface of the gum until it was completely dissolved. There were 10 sessions of local treatment for one course.

The third group consisted of the subjects (22 persons) with coronary heart disease with stable angina pectoris II-III

Table I. Index evaluation of the dental condition of the subjects with CGP I-II garde, suffering from IHD, during the treatment.

Indicator	Stages of treatment	Groups of subjects		
		First (control) (n = 20)	Second (n = 20)	Third (n = 21)
GI	Before treatment	2,09 ±0,10	1,90±0,02	2,1 ± 0,02
	After 6 months	1,8±0,07	1,48±0,07*	1,58±0,01*/**
PMA	Before treatment	60,19±2,65	60,0 ± 2,4	62,2 ± 2,6
	After 6 months	50,02 ± 1,71*	49,8 ± 2,2*	47,0 ± 3,3*/**
PI	Before treatment	4,48±0,13	3,60 ± 0,07	4,05 ± 0,11
	After 6 months	4,00 ± 0,15*	2,50 ± 0,22*	3,05 ± 0,16**

Notes: 1. * – p<0,05 in comparison with the data of the same group before treatment;
2. ** – p<0,05 in comparison with the data of the first group of subjects.

Table II. Indicators of reopardontographic examination of the condition of the paradontal tissue in the subjects with CGP I-II grade.

Indicator	Stages of treatment	Groups of subjects		
		First (control) (n = 20)	Second (n =18)	Third (n = 20)
RI	Before treatment	1,50 ± 0,05	1,50 ±0,07	1,40 ± 0,09
	After 6 months	1,20 ± 0,04*	1,10 ± 0,08*	1,04 ± 0,03*/**
IE	Before treatment	46,0 ± 1,95	43,00 ± 2,37	45,01 ± 1,31
	After 6 months	57,50 ± 1,94*	64,95 ± 1,40*	72,30 ± 1,39*/**
IPR	Before treatment	238,0 ± 10,2	250,0 ± 10,1	246,0 ± 10,4
	After 6 months	168,3 ± 16,5*	142,8 ± 13,3*	135,7 ± 8,7*/**
IVT	Before treatment	23,02 ± 0,59	24,0 ± 1,6	21,00 ± 0,73
	After 6 months	21,81 ± 1,20	21,0 ± 1,7	17,30 ± 1,40*/**

Notes: 1. * – p<0,05 in comparison with the data of the same group before treatment;
2. ** – p<0,05 in comparison with the data of the first group of patients.

f.c., CH IIA, f.c. III for NYHA (pronounced chronic heart failure), with a reduced systolic function of the left ventricle with chronic generalized periodontitis I-II grades. They have prescribed 3-hydroxy-6-methyl-2-ethylpyridine succinate (in capsules of 0.3-0.6 g per day) as antihypoxants for cardiological issues and CoQ_{10} (in capsules of 0.9 g per day).

The length of treatment with antihypoxic drugs lasted for 10 days in all groups.

In order to determine the state of periodontal disease and discriminate a clinical dental diagnosis, a common set of methods for clinical examinations was used. The verification of the diagnosis of generalized periodontitis was performed, according to the classification of MF. Danilevsky[17].

For the purposes of evaluation of the nature of the contents of the periodontal pocket, a benzidine test by S.Sorrin was employed. Differential diagnostics of periodontal and aspartic pockets was conducted with the help of the formalin sample by C.Parma. Fedorov-Volodkin's index (1971) was applied, in order to estimate the hygienic state of the oral cavity. To determine the state of periodontal tissues, the papillary-marginal-alveolar index (PMA) was administered in the Parma modification (1960) and the combined periodontal index of Russell (1956). Furthermore, orthopantomography of the tooth-jaw area and intrathecal stent dental radiography were performed on all categories of subjects.

In order to study the functional state of the vascular bed of the gum, reopardontography (RPG) was used, via the application of the tetrapolar technique. Simultaneously, the rheogram together with the ECG in the second excerpt and the differential rheogram were recorded. The exposure for the records: lying position. The analysis of the data obtained was based on qualitative indicators (by the nature of the curve, the severity of the amplitude, duration and sequence of oscillations) and quantitative indicators: rheographic index (RI), vascular tone index (IVT), index of peripheral resistance (IPR), index of elasticity (IE).

For the assessment of the severity of hypoxia, the concentration of lactate was determined in the blood and oral fluid (the Hohrst method)[18] together with the pyruvate (the method of Tsok and Lamrehta)[19], with the further calculation of their ratio in the blood from the gum tissues. The blood from the gum tissues and oral fluid was used for the measurement of peroxide oxidation of lipids (LPO) and antioxidant system (AOS). The level of LPO in patients was recorded in the light of the reaction of thiobarbituric acid (TBC) with TBC-active products of colored trimethine complex, the activity of AOS via calculation of superoxide dismutase (SOD) and catalase, accordingly[19].

The condition of the nitroxydergic system was judged by the concentration of nitrite ions (NO_2-)in the oral liquid, which are the final products of oxidation of nitric oxide, performing as its depot, and according to the content of urea - a product of metabolism of L-arginine - a substrate of NO-synthase reaction[18].

Table III. The condition of peripheral circulation of the parodontium in the patients with GP I - II grade in CHD, according to reopardotography data.

Groups of subjects	Term	RI	IE	IPR	IVT
Persons without significant somatic pathology (before and after treatment n = 20, after 6 months n = 17)	Before treatment 10 days After 6 months	1,5+0,05 1,2+0,04* 1,2+0,06*	46,0+1,95 57,5+1,94* 54,3+2,07*	238,0+10,2 168,3+16,5* 184,2+17,3*	23,0+0,59 21,8+1,20 22,0+1,03
Subjects with coronary artery disease with stable angina pectoris II f.c., with the preserved systolic function of the left ventricle (before and after treatment n = 20, 6 months after n = 16)	Before treatment 10 days After 6 months	1,4+0,09 1,0+0,03* 1,2+0,11	45,0+1,31 72,3+1,39* 69,7+1,44*	246,0+10,4 135,7 + 8,7* 146,4 + 9,2*	21,0 + 0,73 17,3+1,40* 18,1+0,93
Subjects with coronary artery disease with stable angina pectoris III f.c., (with a reduced systolic function of the left ventricle (before and after treatment n = 22, 6 months after n = 15)	Before treatment 10 days After 6 months	1,5+0,06 0,8+0,06* 0,9+0,07*	50,0+1,38 73,1+2,97* 71,4+2,12*	252,0+20,3 112,7 + 5,6* 124,3 +11,8*	23,0 + 0,92 18,61+1,1* 19,1+0,97*

Note: * – p<0, 05 in comparison with the data of the same group before treatment.

All obtained data were statistically processed. The criterion of Shapiro-Vilka was employed in order to verify the distribution of normality. If the data corresponded to the normal distribution, the Student's t-criterion for independent samples was used to compare them. In the case, where the data series did not appear to be a subject to normal distribution, statistical processing was performed utilizing a nonparametric method, the Mana-Whitney test.

RESULTS AND DISCUSSION

The evaluation of the clinical efficacy of the comprehensive treatment of patients with generalized periodontitis was grounded on the analysis of the dynamics of clinical symptoms alterations of periodontal disease, including X-ray, functional and laboratory features of the subjects. In subjects with hypertension, the disappearance of pain sensations, as well as the disappearance or significant reduction of bleeding of the gums, bad breath, gum edema and hyperemia, no secretions from periodontal pockets, improvement static of moving teeth and reduction or complete disappearance of the increased sensitivity of the teeth were found as a positive outcome, straight after a 2 weeks` treatment, after 1 and 6 months, consequently.

After 7 sessions of treatment of subjects with chronic generalized periodontitis II grade, the phenomenon of inflammation in the gums almost eliminated in 73% of all patients. The gums acquired more natural color, became denser; edema and hyperemia were not observed. The Schiller-Piserev test was negative, the depth of periodontal pockets shrank noticeably. In almost all subjects,there was a decrease in bleeding gums, also, a smaller discharge from periodontal pockets, and alterations in index values of the oral cavity (Table I).

Study of the condition of regional hemodynamics and microcirculation in the periodontal tissues of the subjects with coronary heart disease on the basis of reopardotography is reflected in Table II.

Standard treatment for subjects with HGP (control group) produced a positive outcome, thus, according to RPG: the index of peripheral resistance of the periodontal vessels, the rheographic index, the index of elasticity of the blood vessels improved. However, the indicator of the tone of the periodontal vessels practically did not respond to the course of treatment (p> 0,05), which indicates the ineffectiveness of the administered therapeutic measures.

The additional local antihypoxic therapy (group 2) contributed to a possible decrease in the rheographic index of periodontal disease, the index of peripheral vascular resistance and improvement of their elasticity. In this observed group, as well as in the previous one, there was no significant improvement in the tone of the periodontal vessels, hence, it is not pertinent to consider given treatment to be sufficient.

The implementation of local antihypoxic therapy in combination with the administration of coenzyme Q10 (for the subjects in group 3) contributed to a possible improvement in the values of RI, IE of the periodontal vessels, and a decrease in IOP in comparison with a group of patients in standard treatment (Table III) in different time periods. The analysis of the studied biochemical parameters in the oral liquid of the patients from different groups allowed to state positive changes in the dynamics of treatment of the patients after 6 months (Table IV-VI). The presence of stable angina pectoris with the preserved systolic function of the left ventricle does not have a significant effect on the activity of SOD and catalase in the gum blood of subjects with CGP I-II. The presence of coronary artery disease with stable angina pectoris, following the preserved systolic function of the left ventricle, in subjects with CGP I-II grade, is accompanied by a significant rise in the concentration of lactate - up to 6,53 ± 0,22 mmol / l (15,4%, p <0,05) and the ratio of lactate / pyruvate. The content of the latter may not undergo any significant changes.

Therefore, in the subjects with CGP I-II g., suffering from coronary artery disease with stable angina pectoris, a decreased systolic function of the left ventricle, lactate concentration increased even more – up to 7.74 ± 0.38 mmol / l (36 , 7%, p <0.01), though without significant changes in the level of pyruvate.

Table IV. The activity of antioxidant enzymes in gum blood of patients with HGP I-II severity suffering from coronary heart.

Groups of subjects	The activity of antioxidant enzymes	
	COD,units. act.	Catalase, mk/l
CGP I-II grade. without CHD (control group)	1,13±0,03	2,02±0,11
CGP I-II grade in subjects with stable angina pectoris, the preserved systolic function of the left ventricle	1,16±0,03	2,26±0,12
CGP I-II grade in subjects with stable angina pectoris, a decreased systolic function of the left ventricle	0,94±0,02 *	1,69±0,08 *

Note: * – p<0,05 in comparision with the control group.

Table V. Indicators of anaerobic glycolysis in the blood from the gums in the patients with CGP I-II grade, suffering from coronary heart disease.

Groups of subjects	The activity of indicators of glycolysis, mmol / l	
	Lactate	Pyruvate
CHP I-II g. without CHD (control group)	5,66±0,31	0,167±0,007
CGP I-II g. in subjects with stable angina pectoris, the preserved systolic function of the left ventricle	6,53±0,22 *	0,178±0,011
CGP I-II g. In the subjects with stable angina pectoris, a decreased systolic function of the left ventricle	7,74±0,38 *	0,158±0,007

Note: * – p<0, 05 in comparision with the indicators of the control group.

The presence of coronary artery disease with stable angina pectoris, the preserved systolic function of the left ventricle, in the patients with CGP I-II g. does not significantly influence the concentration of TBC-reactants and their increments during the incubation of the oral fluid in the prooxidant buffer solution, in spite of the fact that during the incubation of blood gum, these indices underwent significant alterations. Nevertheless, in the patients with CGP I and II g., suffering from coronary heart disease with stable angina pectoris with a reduced systolic function of the left ventricle, the concentration of TBC-active products is denoted by an obvious increase - up to the incubation of the oral fluid in the prooxidant buffer solution by 27.1% (p <0.001), after incubation - by 35.2% (p <0.001). The growth in the concentration of TBC-active products during the 1.5-hour incubation in the iron-ascorbate buffer solution went up by a total of 49.6% (p <0.001).

The condition of the system NO was assessed in the light of the concentration in the oral liquid of the final products of its metabolism - nitrite ions (NO2-). The level of urea, a cleavage product of arginine, a precursor of NO, was also determined and taken into account.

In the subjects of the control group with CGP I-II g., without signs of CHD, the content of nitrite ions in the oral liquid was 6.04 ± 0.12 μmol / l, and urea - 59.5 ± 1.21 μmol / l. The presence of coronary artery disease with stable angina pectoris with the preserved systolic function of the left ventricle, in patients with CGP I-IIg. is accompanied by a significant rise in the concentration of nitrite ions and urea in the oral liquid to 6.67 ± 0.16 μmol / l (10.4%, p <0.01) and 68.6 ± 1.11 μmol / 15.3%, p <0.001). In patients with CGP I-II g., suffering from coronary heart disease with stable angina pectoris with a reduced systolic function of the left ventricle, indicators of the nitroxydergic system in the oral liquid are marked by an even greater rise, respectively. The content of nitrite ions went up to 6.72 ± 0.12 μmol / l (11.3%, p <0.01), urea - to 71.3 ± 1.07 μmol / l (by 19.8%, p <0.001).

Therefore, CGP I-II g., in patients suffering from stable angina pectoris are accompanied by more significant metabolic pathologies, the manifestation of which is reflected in the increase in the processes of lipid peroxidation in the blood of the gums and oral fluid; the decline of the antioxidant potential; the violation of the oxygenation of the tissues with the development of lactatemia; a rise in the concentration in the oral liquid of the components of the nitroxydergic system, namely, nitrite ions and urea.

Thus, the level of metabolic disturbances enhances in the direct correlation with the degree of severity of the current condition in subjects with coronary artery disease, stable angina pectoris, and heart failure. In the presence of clinically severe left ventricular dysfunction, there is a manifestation of decompensated peroxide oxidation of lipids with a significant decrease in the activity of antioxidant enzymes - superoxide dismutase and catalase in the blood of gums and oral fluid.

CONCLUSIONS

To sum up, upon prescription of antihypoxic therapy in the comprehensive treatment of the patients with CGP, on the background of coronary heart disease, it is advisable to take into account the sequence of violations of bioenergetic processes that occur on the substrate area of the respiratory chain, followed by violation of the MCFK I, and prolongation with the increase of oxygen insufficiency to the terminal (cytochrome oxidase) sites. Thus, the protection of the body from oxygen deficiency with the help of antihypoxic means provides, as a mandatory condition, the restoration of the energy-absorbing function and a macroergy pool. At the early (compensated) hypoxia stage, the correction of mitochondrial

Table VI. The quantity of secondary LPG products in the oral liquid of the patients with CGP I-II grade, suffering from coronary heart disease.

Groups of subjects	Concentration of TBC-reactants, μmol / l		
	Before incubation	After incubation	Growth during incubation
CGP I-II g. without CHD (control group)	4,62±0,12	7,24±0,21	2,62±0,08
CGP I-II g. in subjects with stable angina pectoris, the preserved systolic function of the left ventricle	5,21±0,28	7,94±0,32	2,73±0,11
CGP I-II g. In the subjects with stable angina pectoris, a decreased systolic function of the left ventricle	5,87±0,21 *	9,79±0,40 *	3,92±0,16 *

Note: * – p<0, 05 in comparision with the indicators of the control group.

disturbances should be directed either to restoring the electron transport function of the ICFA I, or to activate the alternative NADN-oxidase pathway of compensatory metabolic flows, as confirmed by our studies of the high efficacy of succinate-containing antihypoxants, derivatives of 3-hydroxypyridine.

With the increase in the intensity of systemic effects of hypoxia (under conditions of development of severe chronic heart failure with a reduced systolic function of the left ventricle) and the severity of hypoxic disorders in the periodontal tissues, the decompensation of energy metabolism is unavoidable, requiring an obligatory restoration of the electron transport function of the respiratory chain at the level of the coenzyme Q - cytochromes b-c_1. This claim is confirmed during our study by the obvious effectiveness of the drug coenzyme Q_{10}. Comprehensive treatment of patients with generalized periodontitis suffering from coronary heart disease with stable angina pectoris with various degrees of chronic heart failure, contributes to the normalization of clinical and paraclinical parameters, hence, increases the resistance of periodontal tissues to the action of the factors that provoke hypoxic and freely radical necrobiosis, and improves regional hemodynamics and microcirculation in the periodontal tissues.

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CORRESPONDING AUTHOR

Ivan Yu. Popovych
Krasina street 118/2, corp. 1, flat 110
36000, Poltava, Ukraine
e-mail: ivanstomat@ukr.net

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