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METHODS OF DENTAL EXAMINATION OF HYPERTROPHIC GINGIVITIS IN PATIENTS OF THE EXAMINATION GROUP

Abstract. *This article presents hypertrophic gingivitis, causes, etiopathogenesis, pathological changes in gum tissue in hypertrophic gingivitis, dental examination methods for hypertrophic gingivitis, and data on the objective and subjective conditions of patients with hypertrophic gingivitis.*

Keywords. *methods of dental examination for hypertrophic gingivitis, etiopathogenesis, pathological changes in gum tissue in hypertrophic gingivitis.*

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ТЕКШИРУВ ГУРУХИ БЕМОРЛАРИДА ГИПЕРТРОФИК ГИНГИВИТЛАРНИ СТОМАТОЛОГИК ТЕКШИРУВ УСУЛЛАРИ

Аннотация. *Ушбу мақолада гипертрофик гингивитлар, келтириб чиқарувчи сабаблар, этиопатогинези, гипертрофик гингивитларда милк тўқимасидаги патологик ўзгаришлар, гипертрофик гингивитларни стоматологик текширув усуллари, гипертрофик гингивит билан касалланган беморларда объектив ва субъектив ҳолатлари тўғрисида маълумотлар келтирилган.*

Калит сўзлар. *гипертрофик гингивитларни стоматологик текширув усуллари, этиопатогинези, гипертрофик гингивитларда милк тўқимасидаги патологик ўзгаришлар.*

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МЕТОДЫ СТОМАТОЛОГИЧЕСКОГО ОБСЛЕДОВАНИЯ ГИПЕРТРОФИЧЕСКИХ ГИНГИВИТОВ У ПАЦИЕНТОВ ОБСЛЕДОВАТЕЛЬНОЙ ГРУППЫ

Аннотация. *В этой статье гипертрофический гингивит, причины, этиопатогинез, патологические изменения тканей десен при гипертрофических гингивитах, стоматологические методы обследования на гипертрофический гингивит, при гипертрофическом гингивите представлены данные об объективных и субъективных состояниях больных.*

Ключевые слова. *методы стоматологического обследования на гипертрофический гингивит, этиопатогинез, патологические изменения тканей десен при гипертрофических гингивитах.*

Relevance of the study. Parodontal diseases are widespread worldwide and affect various age groups and population segments. According to the WHO, they occur in 30-80% of cases in adolescence and 64-98% in adults. Hypertrophic gingivitis accounts for 5.2 to 41.6% of Parodontal diseases overall, with its incidence during hormonal changes reaching 40-100% in pregnant women, 5.2-41.6% during puberty, 14.5-83% in individuals taking calcium antagonists, 40-50% in anticonvulsants, and 30% in immunosuppressants. According to literary data, the treatment tactics for hypertrophic gingivitis should be agreed upon with general specialists: during puberty - with a pediatrician, in case of endocrine diseases - with an endocrinologist, when taking anticonvulsants - with a neuropsychiatrist. According to modern concepts, conservative treatment of patients with edematous hypertrophic gingivitis includes: professional oral hygiene; elimination of factors that aggravate the pathogenic effect of microorganisms (occlusal trauma, pathology of frenulum attachment in the vestibule area); local use of antibacterial and anti-inflammatory drugs (antiseptics, NSAIDs, agents affecting microcirculation), antioxidants. Taking into account modern concepts of the pathogenesis of Parodontal diseases, conservative treatment includes antibacterial and immunomodulatory therapy; they take into account the individual characteristics of the patient, the severity of general and local manifestations, the nature and type of pathogen, its resistance to antibiotics, the general condition of the patient, his age, and the stage of development of the process.

According to literary sources, pathogenetic therapy of inflammatory Parodontal diseases includes several mechanisms: normalization of vascular tissue permeability; inhibition of lysosomal enzymes; Decreased activity of histamine, kinins, and prostaglandins; improved metabolic processes; de-

creased hypoxia and free radical oxidation; cytostatic effect; stimulation of endogenous glucocorticoid secretion. Synthetic drugs for the treatment of edematous hypertrophic gingivitis have been used for a long time; however, they have disadvantages such as toxic and irritating effects on the body. Currently, the use of herbal remedies that do not disrupt the oral microbiome is promising in dental practice. This makes it possible to use all the components of the medicinal plant and combine herbal remedies to enhance the therapeutic effect.

Results and analysis. Extracts of medicinal plants are represented by a wide range: plant antiseptics, antioxidants, amino acids, vitamins, macro- and microelements, compounds with immunocorrective properties. Based on the above, we set ourselves the goal of developing a new method for treating the edematous form of hypertrophic gingivitis using a herbal preparation. Hypertrophic gingivitis Parodontal diseases are widespread throughout the world and, according to WHO data, occur in childhood and adolescence in 30-80% of cases, and in adults - in 64-98% of cases. In the overall structure of Parodontal diseases, hypertrophic gingivitis accounts for 5.2 to 41.6% of cases, while its frequency during hormonal changes in the body is 40-100% in pregnant women, 5.2-41.6% - during puberty; in individuals taking calcium antagonists - 14.5-83%, anticonvulsants - 40-50%, immunosuppressants - 30%.

Parodontal diseases can develop due to local factors, including plaque microorganisms, occlusal trauma, poorly sealed fillings, and inadequate prosthetics. These factors can also contribute to the combined effects of local and systemic factors, coupled with changes in the body's reactivity. The manifestation of Parodontal disease depends on the state of the body and its individual systems, so it is important to consider the overall health of the body when treating diseases of the oral organs and tissues. Diseases that can lead to Parodontal disease include endocrine disorders, cardiovascular, gastrointestinal, and pulmonary diseases, nervous system disorders, pregnancy, and others. The basis for the pyugenesis of Parodontal disease is the interaction of periodontopathogenic anaerobic microflora and factors of the body's immunological reactivity. Numerous studies have demonstrated the role of phagocytic cell activation in the development of Parodontal disease, particularly the importance of their respiratory metabolism.

One of the local etiological factors in the development of Parodontal diseases is the lack of comprehensive oral hygiene. Biofilm, a structured community of microorganisms encapsulated in a self-producing polymer matrix that adheres to a living or inert surface, plays a key role in their development. Biofilm is characterized by surface attachment, a heterogeneous structure, genetic diversity, complex intrasystemic interactions, and an extracellular matrix of polymeric substrates. The oral microflora is vital, but its development should not lead to the emergence and development of clinically evident disease. The mouth normally contains a balanced ratio of microorganisms; disruption of this balance leads to the development of pathological processes in the periodontium. Certain microorganisms play a leading role in the development of the inflammatory process in the periodontium: Porphyromonas. gingivalis, Actinobacillus actinomycemtemcommitans, Prevotella intermedia, fusobacteria, spirochetes, trepopema and others.

Pathogenic microflora in dental plaque acts as a trigger for the activation of Parodontal macrophages and their induction of a cascade of anti-inflammatory cytokines, which causes structural damage and disruption of microcirculation in the peripheral vessels of the periodontium. The aggressiveness of this microflora is associated with the presence of proteolytic enzymes, endo- and exotoxins, which directly and through immune reactions damage Parodontal tissue and cause negative changes in the local oral immune system. According to S. Asikainen, after stopping toothbrushing, patients experience pronounced inflammatory processes in Parodontal tissues within 7 days. Some microorganisms secrete endotoxins, which are lipid- polysaccharide-nucleic acid complexes that have an autolytic effect, causing pronounced vasomotor reactions and leading to disruption of cellular metabolism. Another factor in the pathogenicity of microorganisms is their enzymatic activity: microbial enzymes can increase capillary permeability, disrupt the permeability of the epithelial membrane, and penetrate the subgingival connective tissue. Proteolytic enzymes in dental plaque stimulate the formation of highly active polypeptides called kinins, which cause the main clinical symptoms of gingivitis.

The pathogenetic mechanisms for the development of Parodontal inflammation include the excessive formation and accumulation of free radicals, which are highly reactive and capable of causing oxidative modification of proteins, nucleic acids, and carbohydrates. This leads to disruption of tissue respiration in the inner mitochondrial membrane and hydroxylation processes in microsomes. This damages the structures of DNA and ribosomal RNA, as well as cellular organelles, and increases the activity of intracellular enzymes. The end result of these processes is the development of oxygen-dependent pathological processes such as hypoxia, chronic inflammation, and intoxication.

In early pregnancy, a woman experiences a deterioration in the condition of dental hard tissues and the periodontium due to poor oral hygiene and changes in the composition of oral fluid. In the second trimester, gingivitis severity increases. Specific estrogen and progesterone receptors have been identified in gingival tissue, which functions as target organs for sex hormones. According to numerous studies, high levels of progesterone and estrogen-17

during pregnancy influence the focal inflammatory process by stimulating inflammatory mediators, particularly prostaglandin E2. It has also been found that progesterone and estrogen-17 reduce the regulatory properties of interleukin, which consequently reduces gingival resistance to bacterial products, affects vascular permeability and exudation, and completely stops microcirculation, reducing the capacity for keratinization and cellular regeneration. Hormones stimulate the growth of various bacteroides, in particular Prevotella Pregnancy is characterized by systemic effects that extend not only to the reproductive organs but to the entire body. These effects are the result of complex hormonal, immune, and nutritional changes.

The periodontium is an indicator of functional and morphological changes in the body. Most patients develop Parodontal disease when innervation and circulation are impaired. The autonomic nervous system plays an integrating role in regulating metabolic processes and continuously adjusting to a constantly changing rhythm. Relationships have been established between Parodontal diseases and various systemic pathologies, and there are factors that may underlie this relationship. The effects of different factors on Parodontal tissues vary and depend on the state of the autonomic nervous system. Some scientific studies have shown a link between Parodontal disease and changes during puberty during adolescence. During this period, sympathetic impulses flow to the body's systems, which reduces the adolescent's resistance to adverse external factors.

According to modern sources, the proliferation of gum tissue appears with drug treatment. Taking certain groups of drugs provokes the development of hypertrophic gingivitis: calcium channel blockers - nifedipine, used in the treatment of hypertension, heart failure, coronary vascular spasms, angina; immunosuppressants - cyclosporines, which are used in organ transplants, the treatment of psoriasis, ringworm and other autoimmune disorders, antispasmodic drugs that reduce the frequency and duration of seizures in patients with epilepsy. The mechanism of action of cyclosporine on gum tissue is associated with a selective and reversible change in lymphocyte function by suppressing the differentiation and proliferation of T- cells. Hucke et al. described a model in which gingival overgrowth is associated with increased activity of 17a-hydroxylase caused by excessive secretion of ACTH in response to the suppression of zona Calcium channel blockers for glomerulolytic therapy. Pathophysiology of gingival overgrowth associated with Dilantin use. Multifactorial: the drug has a direct effect on fibroblast proliferation, and the increase in the intermediate/fiber ratio is also a result of stimulation of the intermediate.

Currently, a link has been established between cardiovascular disease and Parodontal disease, which increases with aging. Desvarieus et al. discovered a correlation between bacterial colonization and subclinical atherosclerosis. A link was also found between cerebrovascular lesions and Parodontal disease, as well as a deterioration in the patient's hygiene status. The researchers also established a relationship between the amount and duration of smoking and the severity of Parodontal pathology. Both local and systemic mechanisms have demonstrated the negative impact of tobacco on oral health. Smoke and high temperatures can increase tartar formation; nicotine reduces collagen synthesis and protein secretion, which leads to a delay in wound epithelialization, increasing susceptibility to Parodontal disease. Smoking reduces immunological function and negatively affects immunoglobulin levels, which increases susceptibility to specific and non-specific microbial pathogens.

Scientists have proven that morphological changes differ in the edematous and fibrous forms of hypertrophic gingivitis. In the edematous form, morphological changes manifest themselves as increased acidic mucopolysaccharides, vasodilation, and increased lymphocyte and plasma cell counts. In the fibrous form, acanthosis, fibrobladder proliferation, enlarged collagen fiber bundles, and small foci of inflammatory infiltration are observed. Data obtained from gingival sulcus fluid analysis allow us to determine the nature of Parodontal changes and their dynamics during treatment. Currently, the choice of treatment for hypertrophic gingivitis is particularly important, depending on the etiology, pathogenesis, clinical course, and degree of gingival inflammation. According to the literature, treatment for hypertrophic gingivitis should be coordinated with general practitioners: a pediatrician during puberty, an endocrinologist for endocrine disorders, and a neuropsychiatrist when taking anticonvulsants. Surgical treatment, gingivecgomy, is effective; however, it is not always feasible. Gingivectomy is not recommended for certain systemic conditions, including blood disorders (Werlhof's disease, hemophilia A and B, congenital and acquired thrombocytopenia, von Willebrand -Jurgens syndrome), endocrine disorders (diabetes mellitus, thyroid disease), and chronic liver disease. Surgical treatment of hypertrophic gingivitis may not be effective in children, during the period of relative morphological and functional immaturity of the genital area, due to relapses and further development of gingival hyperplasia, which contributes to the destruction of the Parodontal ligament and the development of deeper Parodontal lesions. Absolute indications for gingivectomy include grade III hypertrophic gingivitis and the failure of conservative therapy. Indications for cryotherapy include grade II and III hypertrophic gingivitis and papillitis, gingival fibromatosis, and epulis.

Normally, gingival fluid is produced in small quantities. During inflammation, its volume increases due to increased intratissue pressure and

vascular permeability in the lamina propria. Various inflammatory mediators, enzymes, and other biologically active substances, as well as products of enzymatic tissue breakdown, penetrate the gingival crevicular fluid. Analysis of these substances can be clinically significant for diagnosing the nature, stage, and activity of the pathological process. Endotoxins, amines, butyrate, various enzymes, and their inhibitors are of microbial origin. The content and composition of cellular elements in the gingival crevicular fluid are of important diagnostic value, as they can indicate the nature and activity of the inflammatory process. The most typical cells in this fluid under normal conditions are neutrophilic granulocytes and monocytes. In hypertrophic gingivitis, the gingival crevicular fluid contains granulocytes (70-80%, mainly neutrophils), monocytes/macrophages (10-20%), mast cells (5%) and T-lymphocytes (5%); B-lymphocytes are usually not detected.

Conclusion. Thus, data on the high prevalence of hypertrophic gingivitis demonstrate the relevance of this pathology. The main aspects of the etiology of hypertrophic gingivitis have been studied by numerous authors; however, its pathogenic mechanisms, and therefore the choice of treatment method, may differ depending on the underlying etiological factor.

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