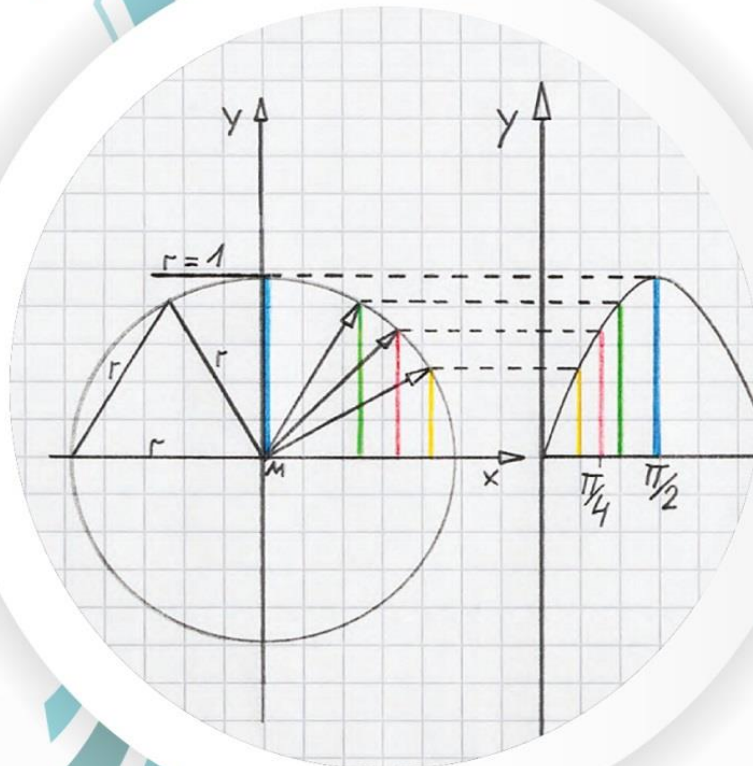


INTERNATIONAL JOURNAL OF

ENGINEERING MATHEMATICS: THEORY AND APPLICATION



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Hungary Academic Member of ATINER

Athens E-mail: kurdics@nyf.hu

CONTACT

Professor of Computational Engineering

Mathematics and Numerical Analysis

Faculty of Engineering

Zagazig University

Zagazig

P. O. 44519

Egypt

<http://iejemta.com/>

Email: sgamil@zu.edu.eg

STUDY OF CHRONIC PSYCHOEMOTIONAL STRESS ON PERIODONTITIS AND GINGIVAL RECESSION

Sabirov Elyor Ergashevich- assistant (Phd)

Boymuradov Shukhrat Abdujalilovich- D.M.Sc, professor

Khaitmurodov Davron Ergashevich- assistant

Ruzikulova Munira Shukhratovna- assistant

Tashkent Medical Academy (Tashkent, Uzbekistan)

Abstract. The problem of restoring lost tissues in periodontal diseases is relevant due to the high prevalence of the pathology and difficulties in treatment. According to WHO data, functional disorders of the dentoalveolar system caused by tooth loss due to periodontal diseases develop 5-6 times more frequently than in cases of caries complications.

Keywords: periodontium, emotional stress, gingival recession

Psycho-emotional tension has become a constant companion in modern life. Information overload, accelerated pace of life, and unfavorable working and living conditions create or significantly intensify chronic stress. Numerous contemporary studies have demonstrated that emotional factors play a leading role in the development of cardiovascular pathologies, neurotic states, and ulcerative processes in the digestive tract. Periodontal tissues are characterized by high sensitivity to stress stimuli. In the mechanism of cellular damage to the periodontium during stress, as mentioned above, a significant role is attributed to the activation of lipid peroxidation and hemostasis. Periodontium - a complex of closely interconnected tissues (gums, periosteum, alveolar bone, periodontal ligament, and cementum covering the tooth root) surrounding and fixing the teeth. Periodontal tissues represent an embryological, physiological, and pathological unity. There is a close relationship between the development, functions, and diseases of the periodontium, despite the different structures of its constituent elements. The embryological connection is indicated by the fact that all elements of the periodontium (except for the gums) develop from the connective tissue surrounding the tooth germ and have a common blood supply. The physiological connection manifests itself in the fixative function of periodontal tissues. When teeth are lost, the entire periodontium is resorbed. The pathological connection lies in the fact that pathological processes arising in Changes in individual tissues of the periodontium typically spread quickly to its other parts. The periodontium is considered more of a functional, physiological, and pathological concept rather than an anatomical one[1].

Gingival recession is the apical displacement of the gingival margin below the cemento-enamel junction (CEJ) relative to its physiological position, resulting in gradual exposure of the tooth root on the oral or vestibular side. Gingival recession is classified as a pathological condition of periodontal tissues in the International

Classification of Diseases ICD-C according to ICD-10. In the new classification of periodontal tissue conditions and diseases, adopted in Chicago on November 9-11, 2017, under the co-chairmanship of the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP), it was decided to consider gingival recession as mucogingival deformations and conditions. There is evidence that the prevalence of this pathology of varying degrees occurs in approximately 58-62% of people. Gingival recession can be localized or generalized and affect one or more tooth surfaces. Today, the exact mechanism of gingival recession occurrence has not been fully studied, and it is assumed that it has a multifactorial etiology. Clarification of the etiology and pathogenesis of gingival recession is the focus of many researchers. Authors differently assess the degree of importance of various etiological factors. The specific structure of the vestibular and oral walls of tooth sockets, their functional significance in the perception of masticatory loads necessitate focusing attention on further study and clinical assessment of their condition[2]. In conditions of chronic emotional-pain stress, histological studies of soft tissues revealed deeper dystrophic changes in cellular, epithelial, and connective tissue elements compared to OEBS [3].

In the gingival epithelium, the contours are unclear. The boundary between the basal and spinous layers is weakly defined. Cells with pyknotic and lysed nuclei, as well as isolated micronecrosis of the epithelium, are encountered. Deorganization of the periodontal connective tissue is observed, manifested in the disorganization of collagen fibers. The latter appear swollen and swollen. Fibroblasts are located along the collagen fibers. Blood capillaries and small veins The blood vessels are dilated and engorged with blood. Very rarely, destruction of capillary walls is observed. In larger vessels, endothelial swelling is noted. Mast cells filled with granules are present, as well as those in a degranulated state. Examination of patients with generalized periodontitis of the 1st degree of development, who are in a state of chronic emotional stress, also revealed a tendency towards worsening clinical manifestations of the disease compared to patients working under reduced stress conditions. They experienced a deterioration in oral hygiene and an increase in the prevalence of inflammatory processes in the periodontium. A similar trend was observed in patients with moderate generalized periodontitis. These patients showed a significant increase in the level of general collagenolytic activity in the gums and oral fluid, with this indicator being higher in women than in men [4]. The author concludes that chronic emotional stress intensifies the collagenolysis process and contributes to the depolymerization of the main substance of periodontal connective tissue. Furthermore, a close correlation was found between the level of collagen activity in the gums and oral fluid and the excretion of hydroxyproline in urine, which proves increased catabolism of collagen in periodontal tissues of patients. Finally, studies of individual typological features of nervous regulation in patients with generalized periodontitis showed medium and high levels of general anxiety. In the group of female workers experiencing chronic emotional stress, an increase in the level of general anxiety was established compared to those working under normal

conditions. The identified close correlations between the level of collagen activity in the gums, oral fluid, blood serum, and the level of general anxiety indicate a dependence of metabolic changes in periodontitis on individual typological features of nervous regulation. The provisions arising from our data regarding the acceleration of periodontal involvement under conditions of chronic emotional stress, loss of molars, and increased resorption of jaw bone tissue can be considered as evidence of the essence of aging as accumulation and damage at all levels of living organism organization [5,6]. L.M. Tarasenko argues that emotional stress participates in the regulation of metabolism and jaw bone repair. Thus, examination of patients with open jaw fractures showed that the presence of a psychotraumatic situation leads to a longer healing of the wound, which proceeds with complications. Thus, periodontal tissues are characterized by high sensitivity to stressors, which manifests as gingival destruction, impaired blood circulation, decreased phosphorus content, increased resorption of the alveolar process of the jaw bones, and tooth elimination. The severity of periodontal damage during stress depends on the functional state of the body's regulatory systems. At the same time, it has been established that the symptom complex of pathological changes during stress is counteracted by adaptation reactions of the periodontium. This manifests as an increase in anti-aggregation activity, which prevents thrombogenesis and the development of hypoxia, replacing the resorption sites of jaw bone tissue with connective tissue, which contributes to the preservation of the supporting and chewing function of the periodontium. The decrease in parathyroid hormone production, characteristic of the stress syndrome, which weakens calcium mobilization from bone tissue, also plays an adaptive role in stress. The leading links in periodontal damage during stress and the initiation of a whole series of pathological reactions are the activation of the processes of CRP, lipids, and hemostasis, which entails a whole symptom complex of changes aimed at regulating homeostatic functions in periodontal tissues.

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