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Chronic Hypertrophy Etiopathoginetic Gixates Gingivitis

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Relevance of research. The clinical development of periodont diseases in adolescents has many differences from similar pathological processes in adults, and this is due to the fact that in adolescence these processes occur in morphologically and functionally immature tissues, which are insufficient and in any case cannot give the same response. There are similar causes that can cause periodontal disease in adults. In addition, in the pathogenesis of clinical forms of surinkal hypertrophic gingivitis in adolescents, functional juvenile hypertension leads to the appearance of periodontitis as a result of juvenile disorders of carbohydrate metabolism. These deviations in the condition of the parodont can be lost without a trace under the influence of minimal interventions or have an independent progressive disease character, despite the fact that the causes that cause them are eliminated [2.4.6.8].

Among the various parodont diseases, pathological processes associated with lateral proliferation of the connective epithelium of the milk sac stand out. Such diseases include chronic inflammation of the gum, edema, hyperplasia of tissues and false pockets without periodontal pockets, since we have attachment or apical migration of the epithelium. However, since the environment of false pockets is low in oxygen, it is favorable for the presence of periodontopathogenic anaerobic microorganisms. As mentioned above, gingivitis can progress to periodontitis. However, even if left untreated, it can remain stable for many years. With treatment, gingivitis is completely reversible.

In adolescents, surinkal hypertrophic gingivitis is usually a generalized process, and at first glance it appears that the parodont is involved in a restricted area. This is due to proliferative inflammation in different parts of the jaw. The positive result of the Schiller-Pisarev test confirms this. According to Vinogradova, it clinically distinguishes granulating, exofoliant (desuvamative) and fibrous forms. However, most authors [1.3.5.7.9.11.13].

Hypertrophic ginigivitis is gingival papillae due to swelling and hyperplasia of connective tissue that causes false periodontal pockets to form that or that part of the tooth crown in the form of edema. Gingival papillae are dark red, soft, not painful, bleeding or self-bleeding when brushing teeth, eating and biting. Most often, the growth of granulation tissue and the identified descvuamasia sites of the epithelium appear on the edge of the gingival papillae, which are similar in appearance to erosion (desquamative gingivitis). In a swollen (granular) form, patients complain of gum growth, itching, bleeding and pain, exacerbate during meals and significantly disrupt chewing movement, the presence of a bad smell the clinical picture of the tumor form is manifested by the patient's complaints about an aesthetic defect caused by an unusual appearance of the gums, pain when brushing teeth and during meals. Due to the significant growth of damaged gums, false periodontal pockets are detected, from which exudate is often removed, but the periodontal attachment is not broken. In the cervical area of the teeth, where there is a hypertrophy of the gums, a large amount of soft or pigmented plaque is found, which is firmly connected with the hard tissues of the tooth. The tops of hypertrophied papillae sometimes undergo necrosis. Additional research methods allow you to determine the inflammatory process of the mucous membrane of varying degrees of intensity. The fibrosis form of hypertorphic gingivitis develops well. Patients do not complain (with mild violence) or complain about the unusual appearance and shape of the gums (with moderate to severe violence) and The Associated aesthetic dissatisfaction.

In the fibrosis form of surinkal hypertrophic gingivitis, the gingival papilla and part of the attached gums are significantly enlarged, dense, pink in color, do not bleed and are painless. Granulations do not bleed or grow. The disease develops gradually. In surinkal hypertrophic gingivitis, pathologically in granular form, there is swelling of the connective tissue of the gingival papilla, increased and dilated

blood vessels, swelling and expansion of collagen fibers, infiltration of lymphocytes and leukocytes, fibroblast proliferation and a decrease in the number of cell elements. The fibrosis form is characterized by roughness and significant growth of collagen fibers, acanthosis and hyperkeratosis phenomena with pycnosis of the nuclei, and vacuolar degeneration of the cytoplasm of epithelial cells. On the inner surface of the papilla, sites of epithelial ulcers are found (positive formalin test). Inflammatory infiltration is weakly expressed [10.12.14.16.18].

Compared to the intact part of the parodont, the resistance of the capillaries is significantly reduced. Also, the hematoma formation time is 14.87 ± 1.31% per second, much lower than that of the intact parodont. A significant decrease in the resistance of capillaries in gingivitis is associated with the formation of granulation tissues rich in blood vessels. Danilevsky N.F. against the background of physiological puberty, an increase in the number of working capillaries in patients with gingivitis, a change in their shape and the nature of microcirculation were noted. This gave reason to believe that functional changes in milk vessels (spasm or dilation) are associated with the characteristics of neuroendocrine diseases and vegetative diseases in girls with pathologies of puberty.

In Surinkali hypertrophic gingivitis, it is characterized by a decrease in the initial and maximum levels of oxygen tension, an increase in oxygen saturation of the tissues of the gums and the time of extraction. Petruchin A.G. Functional disorders of periodontal hemodynamics were found, manifested by a decrease in capillary resistance, an increase in tooth gum temperature, a decrease in the rheographic index, a decrease in the elasticity of blood vessels, their tonic tension and increased peripheral resistance. blood flow [13.15.17.19]. Periodontal tissue disease is one of the diseases known to mankind since ancient times. With the development of civilization, the prevalence of inflammatory periodontal diseases increased dramatically. In the 30s and 50s of our century, this pathology was abundant in people aged 40 and older. Over the past 15-20 years, inflammatory periodontal diseases have become much more "younger" not only in our country, but all over the world. It was identified in the process of epidemiological investigations of the population, the methodology of which implies the targeted identification of indicators that characterize the condition of periodontal tissues [17]. In adolescents, periodontal disease is common. According to the WHO, about 78% of children have various periodontal diseases. They can be inflammatory, degenerative and neoplastic in nature. The largest group of Parodont diseases is inflammation (gingivitis, parodontitis). They account for 92-98% of all periodontal disease. Periodontitis and gingivitis (catarrhal or hypertrophic, swelling form) have the same causal factor. These are two interconnected forms of the disease. The inflammatory process of gingivitis is limited only to dental gums. The spread of inflammation to other periodontal tissues (periodont, root cement, alveolar bone) leads to the development of periodontitis. The prevalence of gingivitis in children is 78%, periodontitis - 2-4%. The most common periodontal disease affects children aged 9-10 years.

K. Research by Jackson (1975) found that gingivitis occurs in 36-90% of cases in England at the age of 15. According to the Harvard School of Dentistry in the United States, 1/3 of children between the ages of 6 and 11 and 1/3 of adolescents suffer from various forms of periodontal disease. In Japan, Yoshinori Takahashi (1986) reported a periodontal inflammatory disease at age 17 in 37% of cases, and at age 20-25 in 63.5% of cases, and according to Yoshinori Sasaki (1986), reported an outbreak of inflammatory forms. the age periodontal disease is 94.3%. N. Muhlenman, A. Magog (1982) found inflammatory changes in the gums in 80% of German schoolchildren between the ages of 7 and 16. Epidemiological studies of the population according to the WHO methodology in our country showed that the prevalence of inflammatory periodontal diseases in children aged 12-15 years is 57-90%, depending on climate and geographical zones. One of the periodontal pathologies that is often observed in youth, according to epidemiological data of domestic and foreign authors, is gingivitis, the prevalence of which reaches almost 100%. Depending on the age of the child, periodontitis is divided into: - for prepubertal periodontitis-up to 11-12 years old; - puberty (underage) - from 12 to 17 years old; - after puberty-from 17 to 21 years old.

The features of the development of periodont diseases in children are associated, first of all, with the development of a pathological process in tissues that develop in constantly growing tissues, forming

part of the periodontal tissues, morphologically and functionally immature, capable of adequately responding to small damaging factors. On the other hand, periodontal pathology has general functions (tooth, periodont, alveolar bone, etc.) that can develop against the background of an imbalance in the growth and maturation of tissue structures in the system. The whole organism and its adaptation to changes in the external environment (nervous, humoral, endocrine, etc.), which leads to the appearance of periodontic diseases in adolescence. In addition, the periodont state can be affected by the absence of synchronicity between the release of permanent teeth and the rate of construction of the alveolar bone, which leads to a decrease in the zone of attached (alveolar) gums, stretching of the clinical Crown. Teeth by 2-5 mm, a decrease in the vestibular depth of the oral cavity. Thus, parodont should take into account the structural features of parodont in children when assessing clinical and Radiological signs of the disease. To prevent inflammatory periodontal diseases, great attention is paid to chronic forms of gingivitis, which are defined in the classification as follows: gingivitis is an inflammation of the gums caused by the negative effects of local and general factors and that proceeds without compromising its integrity are periodont diseases. The main etiological factor in relation to inflammatory periodont diseases is microbial, which in the clinic is determined by dental plaque or soft plaque. In this regard, among all preventive dental methods, individual and professional hygiene measures occupy an important place in the oral cavity. The concept of plaque control is of particular importance [16.19].

As a result of the action of inflammatory mediators (histamine, serotonin, bradykinin), the permeability of blood vessels increases, causing redness, swelling of the gums, parodont, alveolar bones and pain in the gums. Initially, there are signs of gingivitis (catarrhal or hypertrophic, swollen form). Without prolonged treatment, there is relaxation and disruption of periodontal epithelial attachment as a result of the cytotoxic effects of microbial toxins and acidic environment, apical epithelial germination followed by bone resorption. Activation of osteoclast resorption under the influence of inflammatory mediators (lymphokins, leukotrienes, interleukins, prostaglandin). Systemic diseases (diseases of the endocrine, cardiovascular system, blood, gastrointestinal tract, hypovitaminosis, disorders of the functions of the gonads, cases of immunodeficiency, etc.) lead to changes in the immunobiological reactivity of the body, a decrease in protective and adaptive reactions that ensure the stability of the whole organism and especially the parodont. There are many studies showing a significant weakening of non-specific and specific immune factors in patients with periodontitis. In this regard, conditions are being created for the implementation of the main complexes of causal factors. The differences in the course of the periodont disease are due to the different immunity of patients. Prolonged contact of dental sediment microflora from periodontal tissues can lead to the development of optimal processes [15]. Decrease in protective and adaptive reactions, which ensure the stability of the body as a whole, and especially the parodont. There are many studies showing a significant weakening of non-specific and specific immune factors in patients with periodontitis. In this regard, conditions are being created for the implementation of the main complexes of causal factors. Differences in the course of periodontic diseases are due to the different immunity of patients.

According to grudyanova A.I. (1995) when examining students of grades 1-10 of Moscow schools, students of grades 1, inflammation of the parodont was detected, the frequency and intensity of which gradually increases with age: the average frequency of gingivitis in children. In the range of 7 years, from 12 to 20%, the intensity of inflammation - 2-6% (PMA). For graduates, they rose to 32-56% and 28-36%, respectively. At the same time, 6% of graduate students had catastrophic changes in the bone of the alveolar processes. It should be noted that in this case, the hygienic condition of the oral cavity, in both younger and older students, in general, has been described as unsatisfactory.

Conclusion. Thus, we see a clear relationship and correlation between the level of hygienic condition of the oral cavity, the quality of hygienic measures in the oral cavity and the prevalence and intensity of periodontal diseases and, accordingly, the dental condition.

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