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IDIOPATHIC PERIODONTAL DISEASES. CLINIC, COMPARATIVE DIAGNOSTICS

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Annotation: The problem of diseases of a parodontal tissues is very extend and progressive as to find unambiguous approach to diagnostics and treatment of this disease. Examination of a parodontal diseases is implex problem according diverse from etiology, a range, nature of the arising problems, individual health and genetiks factors. Histological research is very importent for full examination to find a effective treatment of paradontal deseases.

Keywords: parodontal tissues, examination of a parodontal diseases, histological research

Idiopathic periodontal diseases are usually a consequence of general systemic diseases of the body. In this regard, their treatment requires simultaneous treatment by several specialists in addition to the periodontist.

With idiopathic periodontal disease, the same signs are observed as with periodontitis:

• Bleeding gums

• Inflammation of periodontal tissues

• High gum sensitivity

In addition, the appearance of periodontal pockets is characteristic; inflammation is accompanied by purulent discharge.

All periodontal tissues, as well as bone tissue, are systemically affected, which leads to loosening of teeth and their rapid loss.

Patients typically complain of pain when chewing, when pressing, general discomfort and swelling in the mouth, loose teeth and bad breath.

Symptoms develop quite rapidly and it is very important to consult a specialist in time. Otherwise, there is a high risk of partial or complete adentia and the need for more complex and expensive orthopedic treatment and prosthetics.

General characteristics of periodontolysis: steady progression of the processes of destruction of all periodontal tissues; rapid formation of periodontal pockets, which determines the release of pus, tooth mobility, displacement and dystrophy; in a relatively short period of time, bone pockets appear, destruction of the vertical type predominates, then lacunae are formed, osteolysis processes lead to complete resorption of periodontal bone tissue and tooth loss within 2-3 years. The etiology of the disease is unknown, the role of a hereditary factor or enzymopathies is assumed [3]. Adolescents during puberty and young women are more likely to get sick. This process is observed in children with severe uncompensated diseases and genetically determined syndromes: uncompensated diabetes mellitus, blood diseases, Papillon-Lefevre syndrome, histiocytosis X, etc.

Clinical picture. There are two stages of the disease:

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— the first stage of desmodontosis is characterized by the absence of symptoms of inflammation. Early symptoms include deformation of the dentition; in particular, fan-shaped divergence of teeth. As a result, diastemas and trema appear. The lesion is characterized by symmetry (central incisors and molars on both sides of the jaw). The gingival margin is not changed throughout, however, bleeding occurs. Dental plaque is scanty or absent. As for indices, IG is within normal limits, RMA is 0%. In the area of mobile teeth, narrow and deep periodontal pockets without discharge are observed. Radiographs show vertical bone resorption with deep bone pockets;

- the second stage of desmodontosis - pathological mobility of teeth progresses, inflammation occurs, pain in the gums, swelling, bleeding appears. Upon examination, periodontal pockets of varying depths with purulent discharge are determined, an exacerbation of the process and abscess formation are noted. Pathological mobility varies from high degree to complete stability and vice versa. Secondary traumatic occlusion occurs, the electrical excitability of the pulp of mobile teeth is reduced, radiographs reveal diffuse destruction of bone tissue in the area of the first molars and incisors, bone resorption in the form of a cup. Clinical signs of periodontal disease in children are detected at the age of 1.5-2 years. After a year or two, the child first loses his incisors, then his other milk teeth. The same thing happens with permanent teeth: lysis of bone tissue occurs, leading to the formation of deep periodontal pockets, then displacement, mobility and loss of first the incisors, then the first molars, then other teeth.

Histologically: no structural changes are detected in the epithelium; hypervascularization, thickening of capillary walls, lymphoplasmacytic infiltration, thickening of collagen fibers, and hyaline sclerosis occur in the submucosal layer. In the periodontium, swelling, disorganization, hyalinosis of collagen fibers occurs, cement is resorbed. In the bone, thinning of the compact lamina and osteolysis are detected. In this case, there is no osteoclastic reaction. The radiograph reveals a significant loss of bone tissue in the interdental septa, including in areas where gum inflammation is not visually detected or is mild.

Differential diagnosis. Periodontolysis must be differentiated from diabetes mellitus. With diabetes mellitus, changes in the periodontium corresponding to the concept of “periodontolysis” are observed in children and young adults, especially in cases of the disease not being diagnosed in a timely manner or in its uncompensated course. Patients I complain of dry mouth, severe bleeding gums, rapidly increasing pathological mobility of teeth, changes in their position, suppuration, the appearance of bad breath and tooth loss.

Upon examination, a picture is usually observed identical to severe periodontitis, sharp hyperemia of the gums, disruption of the configuration of the interdental gingival papillae due to edema, infiltration and proliferation of granulation tissue. Periodontal pockets of varying depth are usually detected, and pathological tooth mobility ranges from insignificant in some areas to degree II-III in others. Changes in the position of teeth in the dental arch and traumatic occlusion are always pronounced[3]. Radiographs reveal significant destruction of bone tissue with a predominance of lacunar, cystic resorption.

Changes in the periodontium, such as lysis of all structures, can also be observed in adults. This is possible in cases of long-term undiagnosed and, therefore, untreated diabetes. It cannot be assumed that in diabetes mellitus changes in the periodontium develop according to the type of periodontolysis alone. With timely diagnosis, treatment, and a compensated type of exchange, the periodontium may remain intact; changes may correspond to gingivitis or periodontitis. The morphological picture of the gums in diabetes mellitus has signs of specific microangiopathies: damage to the microvasculature with exudation of plasma proteins and neutral polysaccharides into

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the walls of blood vessels, proliferation of the vascular endothelium and dystrophic changes in it, up to the formation of hyaline thrombi in the arterioles. Sclerosis and hyalinosis of the vessel walls are noted until their lumen is completely obliterated. Severe vascular changes are accompanied by pronounced changes in the gum stroma: the phenomena of sclerosis, hyalinosis with poor cellular reactions. In the epithelium, the phenomena of acanthosis and focal atrophy with the disappearance of glycogen are observed. Under these conditions, inflammation is much more severe, which also leads to more severe prognostic changes in the periodontium. Osteoclastic resorption, oncolysis phenomena and other disorders characteristic of pronounced dystrophic and destructive changes predominate in bone tissue.

With uncompensated diabetes mellitus, the gingival margin is severely swollen, brightly hyperemic with a cyanotic tint, and bleeds when touched. Periodontal pockets with purulent contents and juicy granulations that bulge outside the pocket. The teeth are highly mobile, displaced along the vertical axis and covered with soft plaque; there are supra- and subgingival stones. A distinctive feature of radiological changes in the jaws is the funnel- and crater-like type of destruction of the bone tissue of the alveolar process, which does not extend to the body of the jaw.

In all forms, lesions (together or separately) of internal organs (liver, spleen), lymph nodes, skeletal bones, skull and jaw bones are observed. The diffuse histiocytic proliferation observed in this case is accompanied by hemorrhagic edema, necrosis and leukocyte reaction. The presence of eosinophils depends on the form and stage of the disease, which can change from one to another. The general condition is disturbed, the body temperature is increased. X-rays of bones reveal single or multiple bone defects of a round or oval shape with clear contours. A tissue biopsy of the affected area reveals a large number of eosinophils or xanthoma cells. Changes in the oral cavity depend on the severity of the disease. Localized histiocytosis X (eosinophilic granuloma, Taratynov disease) is a localized reticulohistiocytosis[3]. It most often affects children and young people (20-25 years old). During the course of the disease, a prodromal period and a pronounced stage are distinguished. In the prodromal period, single foci of destruction appear in the skeleton, manifested by minor pain, itching, and swelling in the affected area. The process is more often localized in flat bones, affecting the skull and lower jaw (alveolar part and ramus). In the oral cavity, swelling and cyanosis of the gingival papillae are observed (usually in the area of premolars and molars), then the papillae hypertrophy, pathological mobility of one or two molars appears, bleeding gums, there may be ulceration of the gingival margin, deep periodontal pockets. In the blood - an increase in the number of neutrophils, accelerated ESR. Often the process is two-way. The course is rapid - 1.5-2 months. With generalized damage to the skeletal system or damage to it together with the lymphatic system, the disease develops slowly, with periods of remission. Ulcerative gingivitis is preceded by severe general symptoms of the disease. Then the roots are exposed and the teeth become mobile, and deep periodontal pockets filled with granulations are formed.

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