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ETIOLOGICAL TREATMENT FEATURES INFLAMMATORY PERIODONTAL DISEASE

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ABOUT ARTICLE

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Abstract: Epidemiologic studies show that only 12% of all people have healthy periodontal tissue, 53% have early inflammatory changes in the supporting tissues of the teeth, and 23% have destructive changes. Of these patients, 12% have moderate to severe changes.

INTRODUCTION

Epidemiologic studies show that only 12% of all people have healthy periodontal tissue, 53% have early inflammatory changes in the supporting tissues of the teeth, and 23% have destructive changes. Of these patients, 12% have moderate to severe changes. These figures also apply to patients not undergoing orthodontic treatment, but the presence of dentures in the mouth contributes to the appearance and development of inflammatory and destructive processes in periodontal tissue due to poor oral hygiene [2]. Most periodontal diseases are inflammatory. Inflammation occurs under the influence of microbial plaque products, but at the same time the role of local trauma, including iatrogenic causes, should be noted. Statistics show that endodontic lesions are responsible for the premature removal of denture crowns in 17% of cases, mechanical destruction of cermet crowns and abutment teeth in 27%, and esthetic defects due to destructive changes in the periodontal ligament in 64% of cases [3]. To date, most non-removable dentures are fabricated without biting prepared teeth as abutments, which naturally leads to chronic injury and rapid deterioration of hygiene due to the preservation of food residues and dental deposits at the edges of non-removable elements (crowns, linings, etc.). Incorrect modeling of the occlusal surfaces and the formation of non-functional occlusions lead to overloading of the supporting and antagonistic teeth, violation of limb

attachment, and integrity of orthodontic structures (inlays, onlays, veneers, metal-free crowns on the edges), often resulting in the development of caries on the supporting teeth. This is due to the exposure of permeable dentin after enamel removal. Techniques are now used to prepare the supporting teeth by forming ledges, reducing the potential for periodontal damage and improving hygiene according to the principle of minimal invasion. Micro- and macro-sealing techniques (filtration with fluorinated compounds and surface coating with photopolymer sealants) are used to increase the resistance of the hard tissues of the supporting teeth to microbiological and aseptic demineralization. The main difference between deep fluorination (microfluorination) is that the CaF_2 crystals are only 50 Å in size. This allows the CaF_2 crystals to penetrate the pores of loose enamel at distances of up to 100 Å, creating a fluoride ion concentration approximately five times greater than that of simply fluoridated (macrofluoridated) calcium fluoride. To cover the surface of the prepared tooth, a composition is used in which microcrystals of fluoride are formed by immersion in a silicate gel to protect it from mechanical impact. This creates an optimal environment for fluoride ions, which, together with the mineral salts in the saliva, sustain remineralization for a longer period of time and increase the remineralization rate 100-fold. Copper ions in the composition of remineralizers contribute to protection from microorganisms by alkaline copper fluoride ($\text{Cu}(\text{OH})\text{F}$). The bactericidal power of the copper compounds is constantly being restored by their interaction with oxygen in the aqueous solution. Deep fluoridation does not damage the mineral tissues of the teeth, since tooth calcium is not removed [4].

Role of Oxidation by Free Radicals Experimental and clinical results have shown that periodontal tissue overload leads to complex pathological changes, primarily inflammation and dystrophic phenomena [6]. This, combined with risk factors, leads to the first background - relative insufficiency of the physiological antioxidant system. First, this is due to the leakage of superoxide anion radicals during the "respiratory explosion" of phagocytosed leukemic cells. The latter accumulate in the gingival fluid and gingival tissue under the influence of local factors (plaque, dental plaque, microflora) [1]. Free radicals damage lipids, proteins, enzymes, nucleic acids, and other compounds in periodontal tissue cell membranes. Antioxidant mechanisms protect cells from damage. One natural antioxidant is reduced glutathione (GSH). This substance contributes significantly to the function of the antioxidant system. Glutathione helps maintain membrane spontaneity and integrity, the normal course of various membrane processes, influences nucleic acid and protein biosynthesis, and plays an important role in protecting against xenobiotics. In general, the glutathione system is an important protective mechanism for cells, increasing their resistance to both chemical and physical factors. Cells respond to many undesirable influences that decrease GSH levels by increasing GSH production [5]. The normal concentration of glutathione in oral fluid is 717 nanomoles/ml [7]. It is natural to assume that this

concentration is increased by periodontal inflammation. If this positioning is proven, it will broaden our understanding of the pathogenesis and have significant implications for the overall diagnosis of periodontal disease. Therefore, there is an urgent need to investigate changes in the glutathione system in the oral fluid of patients diagnosed with abutment periodontitis before and after treatment. The aim of this study was to determine the periodontal status of the abutment teeth of patients wearing non-removable dentures in the mouth, which is an initial step necessary for further clinical and laboratory studies on the changes of the glutathione system in the oral fluid.

METHODS

The periodontal status of 38 patients (18 females and 20 males) who sought restorative treatment after 6-8 years of non-removable metal-ceramic bridges at the Department of Orthodontics, Samarkand State Medical University was investigated. The age of the examined patients ranged from 27 to 68 years.

RESULTS AND DISCUSSION

44.7% of the patients showed symptoms of gingivitis localized to the abutment teeth, with congestive hyperemia of the gingival margin, thickening of the gingival papillae, and bleeding during probing. Destructive changes in the periodontal tissue of the molars characteristic of localized periodontitis (periodontal pocket depth up to 3.5 mm, radiographic signs of resorption mainly in the interdental space and alveolar crevice up to 1/4 the length of the root, mobility of the molars after prosthesis removal up to grade I II, and induced bleeding) were detected in 18.4% of patients. Generalized periodontitis was found in 5.2% of patients. After prosthetic removal, hard tissue demineralization foci were found in 65% of the supported teeth. We performed a comprehensive treatment, which could be divided into general and localized treatments. Since it is very important to increase the restorative (reconstructive) capacity of the tissue, general restorative therapy was used according to the indications. Vitamin therapy and phytotherapy were used for this purpose. Local therapy was used to prepare for reconstruction. First, the oral cavity was disinfected to reduce inflammatory phenomena. The oral cavity was treated with aseptics and disinfectants. Ultrasonic curettage of the periodontal pockets was performed, followed by root surface polishing and excision of granulation tissue. Restorative treatment included local physiotherapy and the use of a keratostimulating jelly ("Solcoseryl"). Physical therapy included massage, vitamin electrophoresis, and analgesia. Clinical stability was good due to the combined treatment measures, including alleviation of inflammatory changes, removal of damaged bridges, and temporary splinting of the teeth, which were performed jointly by the periodontist, dental surgeon, and therapist. The patient subsequently underwent

remolding of the non-removable prosthesis according to clinical requirements. A total of 43 ceramic-metal prostheses were fabricated. Hard tissue indentation was used in this patient group. To reduce postoperative sensitivity and prevent hard tissue demineralization, deep fluoridation of the hard tissue of the prepared abutment teeth was performed twice, after tooth preparation and during frame placement. Inflammatory changes in periodontal tissues were due to the lack of compliance with the rules of modern periodontal preparation, the lack of a functioning occlusal relationship between the prosthesis and the opposing teeth, the neglect of the possibility of sealants and deep fluoridation, and the premature detection of the formation of pathological processes. Early diagnosis of inflammatory periodontal disease is necessary, along with adherence to modern methods of preparation of abutment teeth for prosthetics.

CONCLUSIONS

Therefore, new opportunities for early diagnosis of inflammation of the periapical tissues of the abutment tooth need to be developed. That is to study the changes in the oral fluid glutathione system before and after treatment of patients diagnosed with periapical periodontitis of the abutment.

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