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Research Article

MORPHO-FUNCTIONAL CHANGES IN PERIODONT TISSUES IN CHRONIC GENERALIZED PERIODONTITIS ASSOCIATED WITH CHRONIC VIRAL HEPATITIS B AND C

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ABSTRACT

Viral hepatitis (VH) with parenteral transmission of pathogens is one of the most serious and urgent problems of medical science and practical healthcare. . Currently, at least 9 types of human CV are known (A, B, C, D, E, G, F, TT), among which parenteral viral hepatitis deserves special attention, characterized by severe and chronic forms with an outcome in liver cirrhosis and hepatocellular carcinoma . More than 350 million people are currently carriers of this infection, and about 2 million people die every year from hepatitis-related diseases.

KEYWORDS

Chronic viral hepatitis B and C, periodontal disease, chronic generalized periodontitis, chronic gingivitis.

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INTRODUCTION

Chronic hepatitis is a systemic pathology in which the oral cavity is affected with a high frequency [8]. The spectrum of extrahepatic pathology in chronic hepatitis includes damage to the salivary glands and oral mucosa [4]. At the same time, information about the frequency of development, mechanisms of formation of inflammatory periodontal diseases in chronic hepatitis of various etiologies>: features of the course of comorbidity, the effect of treatment are few and contradictory [3,4] This determines the relevance of studying the clinical features and diagnostic criteria for periodontal diseases in patients with chronic hepatitis B and C.

The development of inflammatory periodontal diseases is closely related to disorders of the immune defense of the oral cavity and the body as a whole [8], therefore, the assessment of the state of the immune system by analyzing the content of cytokines becomes important both in studying the mechanisms of formation and in developing methods for treating gingivitis and periodontitis against the background of chronic diseases. hepatitis There is no information on the diagnostic and prognostic value of indicators of apoptosis and proliferation of gingival epithelial cells and in chronic hepatitis of various etiologies

Obviously, approaches to the treatment of inflammatory periodontal diseases against the background of chronic hepatitis should be built taking into account the universal pathogenetic mechanisms of liver and periodontal damage. It seems promising to study the effectiveness of the use of ursodeoxycholic acid (UDCA) in the complex treatment of inflammatory periodontal diseases in patients with chronic hepatitis, which has numerous effects, among which cytoprotective, antiapopgotic, and immunomodulatory effects have been proven [1,3].

The purpose of this study is to substantiate the clinical and morphological state of periodontal tissues in chronic hepatitis B and C.

MATERIAL AND RESEARCH METHODS

To study clinical and morphological changes in periodontal tissues, an in-depth periodontal examination of persons with periodontal diseases against the background of chronic hepatitis B and

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C was carried out in the amount of 35 people - the main group, as well as 20 patients who do not have somatic pathology. They were taken as a comparison group.

Diagnosis of periodontal diseases was carried out in accordance with the terminology and classification of periodontal diseases approved at the XVI Plenum of the All-Union Society of Dentists (1983). Patients underwent a comprehensive clinical and radiological examination of periodontal tissues.

Changes in the color of the gingival mucosa, the degree of gum bleeding [Muhlemann, 1971], the depth of periodontal pockets (WHO, 1989), and pathological tooth mobility were assessed [Fleszar T J et al, 1980].

An index assessment of the state of periodontal tissues was also carried out, using a simplified Green-Vermilion hygiene index (1965), papillary-marginal-alveolar index (PMA) [Parma G, 1960], periodontal index (PI), [Rüssel A, 1967] . X-ray examination of the dentoalveolar system included intraoral contact images of individual groups of teeth and orthopantomography. Assessment of bone density of the skeleton (densitometry) was performed by dichromatic X-ray absorptiometry on a Prology densitometer (USA).

RESULTS AND DISCUSSION

At the first stage of the study, we studied the dental status of patients with chronic hepatitis of viral and non-viral etiology. Extrahepatic manifestations of chronic hepatitis were diagnosed in 17% of patients, with significantly more ($p<0.05$) in chronic HCV hepatitis (26.9%) than in steatohepatitis (7.4%).

Dental extrahepatic manifestations of chronic HCV infection are mainly represented by xerostomia within the framework of Sjögren's syndrome, which was diagnosed in 7.7% of patients. There were complications of xerostomia cheilitis (7.7%), glossitis (5.8%), stomatitis (5.8%). Sjögren's syndrome was accompanied by generalized severe periodontitis. Multiple dental caries was noted in 78.8% of patients with chronic HCV hepatitis and in 61.1% of patients with chronic steatohepatitis.

The clinical and instrumental analysis of the state of periodontal tissues made it possible to establish that the clinical course and severity of periodontal damage in chronic hepatitis are

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associated with the etiology and degree of clinical and laboratory activity of liver damage. Inflammatory periodontal diseases are more severe against the background of chronic active hepatitis of viral or alcoholic etiology pronounced cytolysis and are characterized by the development in most patients of chronic generalized periodontitis of moderate severity (46.2-50%), less often generalized periodontitis of mild (26.8-27.8%) or severe (13.5-16.5%) degree and chronic generalized catarrhal gingivitis (13.5-5.5%).

Chronic non-alcoholic steatohepatitis was more often associated with mild chronic periodontitis (52.8%), less often with generalized moderate periodontitis (22.2%), chronic generalized catarrhal gingivitis (16.7%) or severe periodontitis (8.3%) .

Perhaps the development of more severe forms of periodontal disease against the background of chronic hepatitis of viral and alcoholic etiology is associated with the immunosuppressive effect inherent in chronic HCV infection and ethyl alcohol [6,7]. Damage to the periodontium in conditions of impaired liver function due to the toxic effects of ethanol is not excluded.

A relationship has been established between the severity of inflammatory periodontal diseases and the activity of hepatitis. With a high activity of the pathological process in the liver, the signs of periodontal damage are more pronounced than with low activity [1].

Cholestatic syndrome is accompanied by more pronounced changes in the periodontium and bone destruction of the alveolar processes of the gums. In cholestasis in patients with chronic hepatitis and inflammatory periodontal diseases, the level of systemic decrease in bone mineral density of the axial skeleton and peripheral skeleton (osteoporosis and osteopenia) correlates with the severity of the clinical condition of the periodontium and the degree of resorption of the alveolar processes (r = 0.683) [4].

When studying the processes of cell renewal, it was found that chronic periodontitis, in contrast to gingivitis, is characterized by a predominant increase in the proliferative activity of gingival epithelial cells with a moderate increase in apoptosis. In chronic gingivitis, there were no significant changes in the proliferation and apoptosis of gingival epitheliocytes [5].

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In chronic HCV-hepatitis, more pronounced changes in the proliferation and apoptosis of gingival epithelial cells (I Shch-b7 -38.0 ± 1.7%, Iapopt - 0.72 + 0.06) are observed than in steatohepatitis (I K1.67 - 31.5+1.5%, Iapopt - 0.71±0.05, $p<0.05$). Obviously, these changes are determined by the nature of the inflammatory-destructive changes in the periodontium, which is more severe against the background of viral liver damage.

At the second stage of our study, patients with chronic generalized periodontitis against the background of chronic hepatitis were divided into two groups, equal in age, the activity of the pathological process in the liver and gums.

Changes in the proliferative activity and apoptotic death of gingival epitheliocytes in inflammatory periodontal diseases are based on a violation of local regulatory mechanisms, primarily cytokine homeostasis.

The study of the content of cytokines in the oral fluid showed that in chronic gingivitis there is an increase in the content in the oral fluid of both pro-inflammatory (IL-f, INFU) and anti-inflammatory mediators (RAIL, IL-10), which reflects the maintenance of a balance between populations of immunocompetent cells mediating cellular and humoral immune response, and indicates the simultaneous triggering of the inflammatory process and repair mechanisms [6].

Chronic periodontitis is characterized by a local cytokine imbalance with a predominance of a mediator with immunosuppressive properties (IL-10) and a high concentration of IL-1 r. It should be emphasized that in periodontitis, the increase in the concentration of IL-ip exceeded the increase in the level of RAIL, which is undoubtedly important in the progression of periodontal disease.

The increase in the content of the studied cytokines in the oral fluid correlated with the severity of periodontitis (gyl-ip ~ 0.633, rhyl = 0.518, hil_yu = 0.582), the depth of periodontal pockets (hyl-ip - 0.558), the PMA index (hyl-1p = 0.620), the PI index (hil-ip =0.593) and the degree of gum bleeding (hil.10=0.604).

This indicates that the change in the concentration of IL-lp, RAIL and IL-10 in the oral fluid can be considered as a criterion for the severity of chronic periodontitis.

Therefore, a significant factor in the morphogenesis of chronic periodontitis is a

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violation of the processes of proliferation and apoptosis of epitheliocytes of the gingival mucosa. Against the background of perverted local cytokine regulation, the processes of cellular renewal of the gingival epithelium shift towards proliferation, a chronic recurrent inflammatory-destructive process is formed in the periodontium.

When assessing the clinical picture on the basis of index indicators in the dynamics of the disease, it was found that on the 15-16th day from the start of therapy in the 1st group, almost all parameters reflecting the severity of periodontal pathology were significantly better than in the group with traditional methods of therapy.

There were no side effects when using cycloferon liniment in patients of the 1st group, the drug was well tolerated.

Clinical examination of patients for 6 months made it possible to state that exacerbation of periodontitis was observed in the 1st group in 12% of cases, in the 2nd - in 48%.When studying the processes of cell renewal, it was found that chronic periodontitis, in contrast to gingivitis, is characterized by a predominant increase in the proliferative activity of gingival epithelial cells with a moderate increase in apoptosis. In chronic gingivitis, there were no significant changes in the proliferation and apoptosis of gingival epitheliocytes [5].

In chronic HCV hepatitis, there are more pronounced changes in the proliferation and apoptosis of gingival epithelial cells (I Shch-b7 - 38.0 ± 1.7%, Iapopt - 0.72 + 0.06) than in steatohepatitis (I K1. 67 - 31.5+1.5%, Iapopt - 0.71±0.05, $p<0.05$). Obviously, these changes are determined by the nature of the inflammatory-destructive changes in the periodontium, which is more severe against the background of viral liver damage.

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Therefore, a significant factor in the morphogenesis of chronic periodontitis is a violation of the processes of proliferation and apoptosis of epitheliocytes of the gingival mucosa. Against the background of perverted local cytokine regulation, the processes of cellular renewal of the gingival epithelium shift towards proliferation, a chronic recurrent inflammatory-destructive process is formed in the periodontium.

CONCLUSIONS

When assessing the clinical picture on the basis of index indicators in the dynamics of the disease, it was found that on the 15-16th day from the start of therapy in the 1st group, almost all parameters reflecting the severity of periodontal pathology were significantly better than in the group with traditional methods of therapy.

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periodontitis was observed in the 1st group in 12% of cases, in the 2nd - in 48%.

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