Volume 3, Issue 2, February 2025 ISSN (E): 2938-3765

COMARBIDITY OF PERIODONTAL PATHOLOGY AND THE GASTROINTESTINAL TRACT

Saidova Mukhlisa Akhrorovna
Tashkent State Dental Institute, PhD, Doctoral Candidate in the Department of Hospital Therapeutic Dentistry
Mukhlisa_saidova95@mail.ru, +998914438293
https://orcid.org/0000-0003-1803-6194

Abstract
The peculiarities of the spread and course of inflammatory dystrophic processes in the periodontium are directly related to gastrointestinal tract diseases. The pathology of various parts of the digestive tract determines the clinical picture of periodontal tissue damage. In turn, damage to the alimentary tract contributes to the development of exacerbation or causes damage to the periodontium.

Keywords: Periodontal tissue damage, gastrointestinal tract diseases, gastroduodenal zone, chronic gastritis, ulcer disease.

Introduction

Periodontal diseases represent a group of inflammatory and metabolic-dystrophic conditions characterized by the destruction of gingival tissues, periodontal ligaments, and alveolar bone [9].
Inflammatory diseases of periodontal tissues are currently extremely prevalent worldwide. Their occurrence is primarily and decisively attributed to the active interaction of multiple factors, with a predominance of periodontal pathogenic microflora and disruption of local humoral immunity factors [10].
In more than 80% of cases, periodontal diseases occur in conjunction with gastrointestinal disorders, which undoubtedly influences the clinical manifestations and course of periodontitis. This necessitates a comprehensive approach for this category of patients, including both dental treatment and the identification and management of systemic pathology [10].
Today, periodontal diseases represent one of the most significant problems of dentistry.
Gastric ulcer disease (GUL) and duodenal ulcer disease (DUL) are often associated with gingivitis, periodontitis, aphthous lesions of the mucous membranes of the cheeks, lips, and tongue. Nitrogen oxide, produced by the constitutive NO synthases of the digestive tract mucosa, has a wide range of biological effects, affecting gastric motility, secretion, microcirculation, and cytoprotection [10].
Periodontal diseases represent one of the most important problems in dentistry, which is explained by several reasons. Firstly, the prevalence of these diseases among the world's population is high, which, according to WHO data, reaches 98%; among the population of Ukraine, the prevalence of

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

periodontal pathology, depending on age and region, is 85-95%. Secondly, periodontal diseases are the main cause of dental loss in the adult population worldwide, leading to the loss of dentofacial system functions, and subsequently, the digestive system, while deteriorating patients' quality of life.

Concomitant pathology, against which periodontal diseases develop, may be pathology of the cardiovascular, endocrine, genitourinary systems, and, above all, the gastrointestinal tract (GIT). Diseases such as diabetes mellitus, hypertension, subacute septic endocarditis, rheumatoid arthritis, urolithiasis, peptic ulcer of the stomach and duodenum, as well as diseases of the hepatobiliary system are combined with periodontal damage with absolute regularity [3].

The greatest attention is paid to periodontal pathology in cases of impaired digestive system function, since the involvement of the oral cavity organs in pathological processes in the gastrointestinal tract is beyond doubt, which is explained by the commonality of embryonic development, neurohumoral regulation, and morphofunctional similarity of the oral cavity and the gastroduodenal zone [3, 7.].

According to various authors, among patients with diseases of the gastrointestinal tract, pathological changes in periodontal tissues are detected in 87.7-91.8% of cases, including chronic periodontitis in 76.1% of those examined, while inflammatory and destructive changes in periodontitis are generalized and more active [11].

Against the background of gastrointestinal diseases, functional disorders of the salivary glands develop, there is a shift in the system of remineralization and demineralization of enamel, which contributes to the emergence and active course of the carious process [11].

According to modern concepts, the development and progression of inflammatory periodontal diseases is considered not only as a local inflammation of the parotid tissues caused by the microflora of the "dental plaque", but also as an organism's reaction to a bacterial infection. All researchers recognize that the imbalance between bacterial invasion and local resistance of the oral cavity is the main factor determining the development and course of periodontal lesions [6].

Microbial plaque and its waste products are an important link in the chain of factors that cause inflammation and destructive changes in periodontal tissues. There is an opinion about the existence of colonies of associative periodontopathogenic microbial flora, which is most active in the conditions of the gingival groove and periodontal pockets. Gram-negative anaerobes, bacteroids, fusobacteria, spirochaetes, actinomycetes, and anaerobic cocci are particularly important [6].

Most researchers consider periodontitis as an integral component of the whole organism and recognize the close pathogenetic relationship between periodontal diseases and somatic pathology. Diabetes mellitus, urolithiasis, subacute septic endocarditis, hypertension, peptic ulcer of the stomach (GCC) and duodenum (duodenum), liver diseases, etc. are combined with periodontal damage with absolute regularity [6].

Chronic gastritis, pancreatitis, chronic pancreatitis, chronic hepatitis and cirrhosis of the liver are often associated with gingivitis, periodontitis, aphthous lesions of the mucous membrane of the cheeks, lips, tongue [6,7,8]. A stable relationship is noted between periodontal pathology and inflammatory bowel diseases, and in some forms of inflammatory bowel diseases (Crohn's disease), morphological changes pathognomonic for colorectal pathology are found in

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

periodontitis — lymphoid granulomas. Against the background of diseases of the digestive system, the functional activity of the salivary glands and the dynamic balance of enamel de- and remineralization processes are disrupted, which contributes to the emergence and active course of the carious process [6].

At least 4 million people worldwide suffer from peptic ulcer disease every year, and 5% of them have ulcer perforation. In 2014, 19,331 patients with gastric or duodenal ulcer perforation were registered in Russia. Of these patients, 1,9007 were operated on. Russian researchers note that perforation of a chronic ulcer occurs in 15% of patients with peptic ulcer disease. 0.6 – 5.5% of patients have repeated perforations [2].

Peptic ulcer is one of the most common diseases among the working–age population and accounts for about 20-30% of all gastrointestinal diseases, which, in turn, rank third in the world in terms of prevalence after diseases of the cardiovascular system and cancer pathology. Nowadays, in different countries, from 3 to 20% of the entire adult population suffers from this pathology during their lifetime, and 15-30% of patients with this pathology have various complications requiring surgical intervention within 5-10 years of the disease [4].

Inflammatory periodontal diseases are an urgent medical and socio-economic problem of modern society. According to the latest data, there is a tendency for periodontal tissue damage to increase, which is detected in about 60% of the population of European countries. In addition, this topic is relevant not only for the etiological, pathogenetic and diagnostic features of inflammatory periodontal diseases, but also for functional disorders of the dental system due to tooth loss and the adverse effect of foci of chronic periodontal infection on the entire body [12].

According to the latest data, there is a tendency for periodontal tissue damage to increase, which is detected in the population of European countries. In addition, this problem is relevant not only for the etiological and pathogenetic diagnostic features of inflammatory periodontal diseases, but also for functional disorders of the dental system due to tooth loss and the adverse effect of foci of chronic periodontal infection on the entire body [12].

A number of authors attach great importance to the development and progression of gingivitis and periodontitis by both plaque microorganisms, which is the main etiological factor, and the presence of concomitant somatic pathology, especially pathology of the gastrointestinal tract. Gastrointestinal pathology helps to reduce the nonspecific resistance of the body and increase the frequency of acute and chronic diseases of infectious and inflammatory origin. As a result of immune disorders, an imbalance of conditionally pathogenic microflora of the oral cavity leads to the development of inflammatory destructive processes in periodontal tissues [12].

The severity of periodontal inflammatory diseases varies depending on the location and extent of the pathological processes in the gastrointestinal tract. Thus, the pathology of different parts of the gastrointestinal tract determines the clinical picture of periodontal tissue damage [12].

The main cause of periodontal disease is the microorganisms of plaque and plaque, which trigger the mechanisms of sensitization, immune inflammation and eventually the development of destructive changes in periodontal tissues. But at the same time, systemic factors play an important role in the development of the inflammatory process in periodontitis, in particular, concomitant pathology of various organs and systems, which leads to profound changes in the internal environment of the body and structural damage to periodontal tissues. Concomitant pathology,

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

against which periodontal diseases develop, may be pathology of the cardiovascular, endocrine, genitourinary systems, and, above all, the gastrointestinal tract (GIT). Diseases such as diabetes mellitus, hypertension, subacute septic endocarditis, rheumatoid arthritis, urolithiasis, peptic ulcer of the stomach and duodenum, as well as diseases of the hepatobiliary system are combined with periodontal damage with absolute regularity [4].

Conclusion

The oral cavity and the gastrointestinal tract are inextricably linked. Not only diseases of the gastroduodenal zone can cause the development of periodontal pathology, but also periodontal diseases, being a focus of chronic infection, can contribute to the development or exacerbation of chronic human diseases, including the gastrointestinal tract. In this case, conditions are created for the development of the "mutual burden" syndrome with the development of a pathological process in the oral cavity and digestive organs.All of the above necessitates an integrated approach in the treatment of patients with combined pathology of the oral cavity and gastrointestinal tract – a dentist together with a gastroenterologist.

References

1. Аль-Кофиш Мохаммед Али Мохаммед. Оптимизация ранней диагностики, профилактики и лечения воспалительных заболеваний пародонта у лиц молодого возраста. Автореферат.Уфа.2019.
2. Антропов И.В. Прогнозирование агрессивности течения перитонита у пациентов с перфоративной язвой Двенадцатиперстной кишки. Автореферат. Самара 2018.
3. Бабеня А.А. Особенности проявления стоматологической патологии у лиц с заболеваниями желудочно-кишечного тракта (обзор литературы) // Інновації в стоматології. – 2015. – №1. – С. 72-75.
4. Богату С.И. Сочетанная патология: заболевания пародонта и гастродуоденальной зоны (обзор литературы) / С.И.Богату // Инновации в стоматологии. -2017.-N 3-4. - С.40-46.
5. Гасанлы Нигяр Субхан кызы,распространение воспалительных заболеваний пародонта на различных этапах лечения, проводимого несъемными ортодонтическими аппаратами. Современная стоматология • N1 2021. Стр.81-84.
6. Еремин О.В., Лепилин А.В., Козлова И.В. и соавт. Коморбидность болезней пародонта и желудочно-кишечного тракта. Саратовский научно-медицинский журнал, 2009, том 5, № 3. Стр.393-398.
7. Косюга С.Ю., Варванина С.Э. Особенности проявления Helicobacter pylori на слизистой оболочке полости рта // Соврем. пробл. науки и образования. – 2015. – №1. URL: http://www.science-education.ru/121-18335.
8. М.А. Осадчук, Л. Ю. Островская, Е. А. Исламова. Особенности лечения пациентов с язвенной болезнью желудка и двенадцатиперстной кишки, сочетанной с воспалительными заболеваниями пародонта. Вестник Вол.Г.М.У. Выпуск 1 (29). 2009. Стр.76-80.
9. Олейник О.И., Кубышкина К.П., Олейник Е.А.Оптимизация лечения и профилактики заболеваний пародонта путем применения лечебных адгезивных пластин. The Journal of scientific articles “Health and Education Millennium”, 2018. Vol. 20. No 5. Стр.84-87.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

10. Орлова Е. С., Брагин А. В. Комплексный подход к лечению воспалительных заболеваний пародонта у пациентов с Helicobacter pylori-ассоциированной гастродуоденальной патологией. Проблемы стоматологии 2016, Т. 12. № 2, Стр. 36-42.
11. Шадиева Ш.Ш., Гиязова М.М. сочетанная патология: заболевания пародонта и гастродуоденальной зоны. Журнал стоматология. № 2 2021. Стр.73-76.
12. Ярова С.П., Алексеева В.С. Особенности распространения и течения воспалительно дистрофических процессов в пародонта на фоне заболеваний желудочно-кишечного тракта. // Український стоматологічний альманах. - 2014. - № 2. - Стр. 105-107.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5