УДК 616.311.2-056.52-085.

ETIOPATHOGENESIS AND TREATMENT OF PERIODONTAL DISEASE

[1]Navruzova U.O., [2]Jabborova E.J.

[1]Bukhara State Medical Institute named after Abu Ali ibn Sino**,** Uzbekistan
[2]Chairman of the department of dentistry of public health technical named after Afshona Abu Ali Ibn Sino

✓ ***Resume***

The review of literature on features of a current of parodontit at a metabolic disorder is provided in article. Thus, inflammation as one of the most ancient problems of medicine and also the related questions of reactivity and change of various components of a homeostasis including and a complex of metabolic changes, not only did not lose the value, but as the changed structure of incidence shows, acquires today still big relevance. Immune and inflammatory factors modulate a lipidic range. Lipoproteida render regulatory effects on the immune answer, metabolism of cages of a system of immunity and nonspecific resistance to pathogens. In relation to a parodontopatologiya issues of interaction of inflammatory process and violations of fatty exchange and their pathophysiological analysis were practically not developed.

Key words: periodontal disease, metabolic syndrome, obesity, inflammation and metabolic syndrome

PERIODONTAL KASALLIK ETIOPATOGENEZINING VA DAVOLASH USULLARI

[1]Navruzova U.O., [2]Jabborova E.J.

[1]Abu Ali ibn Sino nomidagi Buxoro davlat tibbiyot instituti
[2]Afshona Abu Ali Sino nomidagi jamoat salomatlik texnikumi stomatologiya kafedrasi mudirasi

✓ ***Резюме***

Maqolada metabolik kasalliklarda parodontit oqimining xususiyatlari bo'yicha adabiyotlarni ko'rib chiqish keltirilgan. Shunday qilib, yallig'lanish tibbiyotning eng qadimiy muammolaridan biri sifatida, shuningdek, gomeostazning turli tarkibiy qismlarining reaktivligi va o'zgarishi bilan bog'liq muammolar, shu jumladan metabolik o'zgarishlar majmuasi nafaqat o'z ahamiyatini yo'qotmadi, balki kasallanishning o'zgargan tuzilishi sifatida. ko'rsatadi, bugungi kunda ham katta dolzarblik kasb etadi. Immunitet va yallig'lanish omillari lipid diapazonini modulyatsiya qiladi. Lipoproteidalar immunitetga, immunitet tizimining hujayra metabolizmiga va patogenlarga o'ziga xos bo'lmagan qarshilikka tartibga soluvchi ta'sir ko'rsatadi. Parodontopatologiyada yallig'lanish jarayoni va yog 'almashinuvi buzilishining o'zaro ta'siri va ularning patofiziologik tahlili deyarli ishlab chiqilmagan.

Kalit so'zlar: periodontal kasallik, metabolik sindrom, semizlik, yallig'lanish metabolik sindrom

ЭТИОПАТОГЕНЕЗ И ЛЕЧЕНИЕ ЗАБОЛЕВАНИЙ ПАРОДОНТА

[1]Наврузова У.О., [2]Жабборова Э.Ж.

[1]Бухарский государственный медицинский институт имени Абу Али ибн Сино,
[2]Заведующий кафедрой стоматологии Колледжа общественного здравоохранения им. Абу Али Сино Бухара, Узбекистан

✓ ***Резюме***

В статье представлен обзор литературы по особенностям течения пародонтита при нарушении обмена веществ. Таким образом, воспаление как одна из древнейших проблем медицины, а также связанные с ним вопросы реактивности и изменения различных компонентов гомеостаза, в том числе и комплекса метаболических изменений, не только не потеряли своего значения, но и как изменившаяся структура заболеваемости шоу, приобретает сегодня еще большую актуальность. Иммунные и воспалительные факторы модулируют липидный спектр. Липопротеиды оказывают регуляторные эффекты на иммунный ответ, метаболизм клеток системы иммунитета и неспецифическую резистентность к патогенам. Применительно к пародонтопатологии практически не разрабатывались вопросы взаимодействия воспалительного процесса и нарушений жирового обмена и их патофизиологического анализа.

Ключевые слова: пародонтоз, метаболический синдром, ожирение, воспаление и метаболический синдром.

Relevance

The prevalence of periodontitis in the population, the difficulties in the prevention and treatment of the disease, the ambiguity in the interpretations of the main pathogenetic mechanisms (interdependent inflammatory, immune and metabolic) makes this problem extremely relevant in medicine. It is known that among patients with metabolic disorders (metabolic syndrome, diabetes, gout, systemic lupus erythematosus) inflammatory diseases of the periodontal complex are widespread. Periodontium, its structures are sensitive to the pathogenic effect of factors forming the proatherogenic spectrum of metabolic disorders. This aspect reflects the interdependent effect of two major pathogenetic mechanisms - inflammatory and metabolic [2, 5].

The author noted that periodontal infection can adversely affect the glucose level in diabetes. Treatment of periodontitis, which reduces the bacterial effect and, consequently, the inflammatory destruction of periodontal disease, helps to reduce the amount of glucose in the blood in patients with diabetes. According to Zvigintsev M.A. (1998) there are several mechanisms by which diabetes adversely affects periodontal tissues: vascular changes; connection of glucose with tissue proteins; a change in collagen metabolism; increased activity of matrix metalloproteinases (collagenases); an increase in glucose in the gingival fluid, which leads to impaired function of periodontal cells, tooth decalcification, and carious tooth decay; violation of the immune response, as a result of which the function of neutrophils is weakened and a hyperreactive monocytic response arises, due to which periodontal tissues are destroyed [2].

According to WHO, obesity is recognized as an epidemic of the 21st century. At the end of the 20th century, 30% (approximately 1.7 billion) of the world's population showed signs of obesity. A special epidemiological situation is recorded in highly developed countries. For example, in the US 60% of the population is overweight [3].

Factors contributing to the development of obesity include genetic factors and environmental factors (excessive consumption of high-calorie foods, low physical activity, socio-cultural characteristics f society, etc. A particularly rapid spread of obesity is associated with its nutritional-constitutional or diet-induced (DI) form.

Analyzing the results obtained and based on literature data, the author expresses a number of points explaining the relationship between the development of the inflammatory reaction and changes in lipid metabolism, and also tries to give a pathogenetic assessment of these connections - [18].

The nature of changes in immune reactivity at various stages of periodontitis has been studied by many authors - [7, 8].

This was a violation of immune defense factors and a change in immune regulation during advanced periodontitis, which is characterized not so much by an inflammatory reaction as by changes in osteoblasts and osteoclasts and bone resorption - [10, 13].

When considering the pathogenesis of periodontitis, there is a significant lack of knowledge about the genesis and mechanisms of development of tissue lesions. Treatment and rehabilitation of patients with periodontitis are a significant challenge. When gingivitis occurs, which is the initial stage of the development of periodontitis, a number of authors found a pronounced weakening of specific factors of local protective reactions of the oral cavity - [5, 11, 14].

Local therapeutic effects on the affected periodontium in such patiens are often ineffective [12,14,17].

Lymphocytes are stimulated by bacteria of V. alcalescence type, obtained from patients with gingivitis or periodontitis, a factor inhibiting macrophage migration (MIF) is released. Dental plaque and certain types of bacteria - V. alcalescence, A.viscosus, F. nucleatumn, B. melaninogenicus - are able to induce the production of MIF by lymphocytes of individuals with experimentally caused gingivitis and thus inhibit or completely suppress phagocytosis - [12, 16].

Today, scientists in many countries are developing a concept according to which inflammation in general, and subclinical inflammation in particular, are considered as the general pathophysiological basis of modern pathology, which closes the pathogenetic circles of nosological forms of diseases of civilization. The commonality of inflammation and proatherogenic metabolic disorders from a pathophysiological point of view is quite natural, since both syndromes form the same cells: endothelial and smooth muscle, fibroblasts, monocytes and macrophages, neutrophils, platelets and, to a lesser extent, T and B lymphocytes .

In inflammation and atherosclerosis, the adhesion (fixation) of monocytes and neutrophils on the surface of the endothelium is activated by the same proteins of cellular interactions: integrins on the membrane of neutrophils and monocytes, E-selectin on the membrane of the endothelium and P-platelet platelet. In both pathological processes, active infiltration (chemotaxis) of tissues by monocytes and neutrophils circulating in the blood occurs. In both situations, activated neutrophils and tissue macrophages in a respiratory explosion reaction enhance the formation of superoxide radicals and activate peroxidation of proteins and lipids, causing alteration of normal tissues - [6, 9, 10]. Since 1999, after the publication, which has already become the textbook work of R. Ross, the inflammatory nature of atherosclerosis is recognized by most scientists. Since then in all studies and published articles, the idea of inflammation as the essence of the atherosclerotic process is dominant, and it pushed into the background the importance of hypercholesterolemia as a factor of atherogenesis. However, in most recently published works, the thesis on the importance of inflammation in the pathogenesis of atherogenic metabolic disorders has acquired a declarative character, since each author puts his own meaning into this concept. Over two decades of research, reports periodically appear that an association between inflammation in periodontal tissues and cardiovascular disease is detected.

Still debatable is the question of the etiological dependence of periodontal inflammation and lipid metabolism. . In a number of modern epidemiological, clinical and experimental studies, conflicting results have been obtained regarding periodontitis and coronary heart disease (CHD), which is associated with general additional risk factors characteristic of these nosological forms (aging, male gender, socioeconomic status, smoking). Nevertheless, studies of the relationship between serum antibody levels against periodontopathogens (Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans) and risk factors for coronary heart disease (proatherogenic lipid profile) confirm this dependence - [1, 4, 11].

According to Borges P.K. et al. (2007), chronic infections with a subpclinical course (for example, periodontitis), can play a greater role in the develoment of atherosclerosis than is currently considered. The decrease in the frequency of cardiovascular diseases with statins is due not only to lipid-lowering, but also anti-inflammatory effect of these drugs - [19, 20].

Lipoproteins have regulatory effects on the immune response, the metabolism of cells of the immune system and non-specific resistance to pathogens. The relationship of the immune and lipid systems is usually analyzed when discussing several issues.

Conclusion

In relation to periodontopathology, the issues of the interaction of the inflammatory process and disorders of fat metabolism and their pathophysiological analysis have not been practically developed. This aspect seems to be completely pathogenetically justified (the interdependent effects of inflammatory and metabolic components) from the standpoint of both pathophysiology and dentistry, which is important for the development of new (pathogenetically significant) therapeutic approaches.

LIST OF REFERENCES:

1. Алявия З.А. The influence of ozone and ultrasound on the factors of local immunity of the oral cavity with periodontitis: Author's abstract. ... Candidate of Medical Science. - Tashkent, 2009. – 21 с.
2. Bitsermaster S.D. Microbiological diagnosis of periodontal tissue diseases // New in dentistry. - 2003. -№7. - С. 27-30.
3. Bubnova M.G. Obesity: causes and mechanisms of mass gain, approaches to correction // Consilium-medicum. - 2005. - Том 7. - N5.
4. Bulgakova A.I. The influence of the state of local immunity of the gums and oral cavity on the course of chronic periodontitis // New in dentistry. -2001(100). - №10. - С. 90-93.

5. 5.Butrova S.A. Metabolic syndrome: pathogenesis, clinical features, diagnosis, treatment approaches // Russian Medical Journal2001. - Т. 9, №2. - С. 56-60.
6. Вишняк Г.Н. Генерализованные заболевания пародонта. – Киев, 1999. –
7. Воложин А.И., Порядин Г.В., Казимирский А.Н. Иммунологические нарушения в патогенезе хронического генерализованного пародонтит // Стоматология. – 2005. - №3. – С.4-7.
8. Воложин А.И. Дизрегуляция иммунологических механизмов и фагоцитарной активности лейкоцитов - ведущая причина острого воспаления // Патологическая физиология и эксперим. терапия. - 1997. - №2. - С. 29-31
9. Гаража Н.Н., Гарус Я.Н., Ивашов А.В., Сакуров А.А. Эффективность антиоксидантного препарата мексидола в комплексном лечении воспалительных заболеваний пародонта // Стоматология. – 2006. - №6. – С.19-21.
10. Грачев С.В. NO-терапия – новое направление в медицине. Взгляд в будущее. - Москва: Издательский дом «Русский врач», 2001. - С.19-21.
11. Григорьян А.С. Роль и место феномена повреждения в патогенезе заболевания пародонта // Стоматология. - 1999. - №1. - С. 16-20.
12. Григорян А.С., Грудянов А.И. Ключевые звенья патогенеза заболеваний пародонта в свете данных цитоморфологического метода исследований // Стоматология. - 2001. - №1. - С. 5-8.
13. Грудянов А.И. Пародонтология (этиология, патогенез, лечение, профилактика) избранные лекции. - М.: ОАО Стоматологии, 1997. – 32 с.
14. Грудянов А.И., Дмитриева Л.А., Максимовский Ю.М. Пародонтология. Современное состояние вопроса и направления научных разработок // Стоматология. - 1999. - №1. - С. 31-33.
15. 15. Грудянов А.И., Дмитриева Н.А., Овчинникова В.В. Обоснование оптимальной концентрации препарата метрогил-дента при лечении воспалительных заболеваний пародонта // Стоматология. - 2002. - №1. - С. 44-47.
16. Грудянов А.И., Дмитриева Н.А., Фоменко Е.В. Применение таблетированных форм пробиотиков Бифидумбактерина и Ацилакта в комплексном лечении воспалительных заболеваний пародонта // Стоматология. - 2002. - №1. - С. 39-43.
17. Грудянов А.И., Барсегян Т.А., Чкония Г.Д. Комбинированное лечение воспалительных заболеваний пародонта циклофероном и метранидозолом //Российский стоматологический журнал. – 2006. - №6. – С.20-23.
18. Демуров Е.А., Моисеева Е.Г., Илларионова Т.С. Патогенетические механизмы хронизации воспалительного процесса // Развитие научных исследований на медицинских факультетах Университетов России: Материалы 1 Всерос. научной конференции. - Москва, 2001.- С.206-207.
19. Еловикова Т.М. Лечение поражений пародонта и зубов у больных сахарным диабетом 1 типа // Стоматология. -1989. - № 4. - С. 18-20.
20. Звигинцев М.А. Стоматологическая реабилитация больных сахарным диабетом: Автореф. дис. ... д-ра мед. наук. - Омск, 1998. - 43 с.
21. Rakhimova G.Sh., Kadirova L.V. “The condition of some endocrine glands of white rats after an experimental traumatic brain injury. The new day in medicine”. – 2021. - № 5(37), p. 189-191.
22. Кадырова Л.В., Рахимова Г.Ш. «Некоторые Аспекты Состояния Эндокринных Желёз Белых Крыс После Черепно-Мозговой Травмы» CENTRAL ASIAN JOURNAL OF MEDICAL AND NATURAL SCIENCES. – 2021. – С. 254-257.
23. 23.Кадирова Л.В., Тўраев У.Р., Абдуллаева М.А. Changes of Indicators of Immune Status in Patients with Nonspecific AortoArteritis on the Base of Combined Therapy// The Pharmaceutical and Chemical Journal. 2020. - №7(1) .-P. 35-38.
24. Абдуллаева М.А. Цитокиновый профиль у больных неспецифическим аортоартериитом на фоне терапии.// Биология ва тиббиёт муаммолари. 2019. - №116. – С.7-10.
25. У.О., Каримова Г.К., Ихтиярова Г.А.- Современные диагностика патологии шейки матки // Тиббиёт ва спорт -2020 №1. С. 74-773.
26. Наврузова У.О., Хамидова Н.Қ., Юсупов Ш.А- Evropean journal of pharmaceutical and medical research Journal. 2019 №3. С-108-113.
27. Косимова Д.С.,Каримова Н.Н. Акушерские кровотечения: ранние и отдаленные результаты. // Оналик ва болаликни мухофаза килишнинг долзарб муоммолари. –Республика илмий –амалий анжумани материаллари. Бухоро, №1 2015. – С. 75-76 .
28. Косимова Д.С., Ихтиярова Г.А. Гемодинамические изминения у родилниц перенесших тяжелую преэклампсию и ее реаблитация.// Монография. Бухоро 2015й 215б.
29. 29.Negmatullayeva M.A., Navruzova U.O., Inoyatov A.SH., Jabboroba O.I-Ways to solve the incidence of covid-19 as a global problem //Annals of the Romanian for cell biology.2021

Entered 09.05.2022