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INTERRELATION BETWEEN INFLAMMATORY PERIODONTAL DISEASES AND FATTY LIVER DYSTROPHY: MODERN CONCEPTS AND PATHOGENETIC MECHANISMS

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Аннотация

В настоящем обзоре обобщены современные знания о взаимосвязи между воспалительными заболеваниями пародонта (ВЗП) и жировой дистрофией печени (ЖДП). По данным Всемирной организации здравоохранения (ВОЗ), заболевания пародонта остаются одним из наиболее распространенных хронических воспалительных состояний, ухудшающих качество жизни из-за боли, кровоточивости, галитоза и преждевременной потери зубов. В то же время жировая дистрофия печени, неалкогольное метаболическое заболевание печени, характеризующееся чрезмерным накоплением жира в гепатоцитах, все чаще признается частью системных воспалительных и метаболических синдромов. В данном обзоре обсуждаются взаимные патогенетические механизмы, связывающие пародонтальное воспаление и нарушения липидного обмена в печени, с акцентом на роли окислительного стресса, дисбаланса цитокинов, иммунной дисрегуляции и микробиоты полости рта. Также рассматриваются современные диагностические подходы и терапевтические перспективы. Взаимосвязь между здоровьем полости рта и системным здоровьем подчеркивает важность комплексного подхода к ранней диагностике, профилактике и лечению обоих заболеваний.

Ключевые слова: пародонт, воспалительное заболевание пародонта, жировая дистрофия печени, окислительный стресс, системное воспаление, патогенез

Annotatsiya.

Ushbu sharhda parodontning yallig‘lanish kasalliklari (PYAK) va jigarning yog‘li distrofiyasi (JYOD) o‘rtasidagi bog‘liqlik haqidagi zamonaviy bilimlar umumlashtirilgan. Jahon sog‘liqni saqlash tashkiloti (JSST) ma’lumotlariga ko‘ra, parodont kasalliklari og‘riq, qon ketish, galitoz va tishlarning erta yo‘qolishi tufayli hayot sifatini yomonlashtiradigan eng keng tarqalgan surunkali yallig‘lanish holatlaridan biri bo‘lib qolmoqda. Shu bilan birga, jigarning yog‘li distrofiyasi, gepatotsitlarda ortiqcha yog‘ to‘planishi bilan tavsiflangan jigarning alkogolsiz metabolik kasalligi, tizimli yallig‘lanish va metabolik sindromlarning bir qismi sifatida tobora ko‘proq tan olinmoqda. Ushbu sharhda parodontal yallig‘lanish va jigarda lipid almashinuvi buzilishlarini bog‘lovchi o‘zaro patogenetik mexanizmlar muhokama qilinib, oksidlovchi stress, sitokinlar muvozanati buzilishi, immunitet buzilishi va og‘iz bo‘shlig‘i mikrobiotasining roliga e’tibor qaratilgan. Shuningdek, zamonaviy diagnostika yondashuvlari va davolash istiqbollari ko‘rib chiqilmoqda. Og‘iz bo‘shlig‘i

salomatligi va tizimli salomatlik o‘rtasidagi bog‘liqlik ikkala kasallikni erta tashxislash, oldini olish va davolashga kompleks yondashuvning muhimligini ta’kidlaydi.

Kalitli so’zlar: parodont, parodontning yallig‘lanish kasalligi, jigarning yog‘li distrofiyasi, oksidlanish stressi, tizimli yallig‘lanish, patogenez

Abstract

The present review summarizes current knowledge about the relationship between inflammatory periodontal diseases (IPD) and fatty liver dystrophy (FLD). According to the World Health Organization (WHO), periodontal diseases remain one of the most common chronic inflammatory conditions affecting the quality of life through pain, bleeding, halitosis, and premature tooth loss. At the same time, fatty liver dystrophy, a nonalcoholic metabolic liver disorder characterized by excessive fat accumulation in hepatocytes, is increasingly recognized as part of systemic inflammatory and metabolic syndromes. This review discusses the mutual pathogenetic mechanisms linking periodontal inflammation and hepatic lipid metabolism disorders, highlighting the roles of oxidative stress, cytokine imbalance, immune dysregulation, and oral microbiota. Modern diagnostic approaches and therapeutic perspectives are also considered. The interrelation between oral and systemic health emphasizes the importance of an integrated approach to early diagnosis, prevention, and management of both conditions.

Keywords: periodontium, inflammatory periodontal disease, fatty liver dystrophy, oxidative stress, systemic inflammation, pathogenesis

Introduction. Inflammatory periodontal diseases (IPD) represent one of the most prevalent chronic infectious-inflammatory conditions affecting the supporting tissues of the teeth. According to the WHO, the prevalence of periodontal pathology among adults reaches 85–98%, depending on population and diagnostic criteria [1–3]. The consequences of periodontal disease extend beyond the oral cavity, leading to pain, halitosis, bleeding gums, tooth mobility, and eventual tooth loss.

In parallel, fatty liver dystrophy (FLD) — often referred to as nonalcoholic fatty liver disease (NAFLD) or hepatic steatosis — is one of the most common chronic liver conditions worldwide. It is associated with metabolic disorders such as obesity, insulin resistance, and dyslipidemia [12–20]. Increasing evidence suggests a bidirectional relationship between oral inflammation and hepatic lipid metabolism, mediated by systemic inflammatory and metabolic pathways.

The aim of this review is to summarize current data on the interrelation between inflammatory periodontal diseases and fatty liver dystrophy, describe shared pathogenetic mechanisms, and outline diagnostic and therapeutic perspectives relevant for dental practitioners.

Periodontal disease is a multifactorial chronic inflammation of the supporting tissues of the teeth, caused primarily by bacterial biofilm (dental plaque). The disease involves both local and systemic immune responses to microbial invasion. Under normal conditions, the inflammatory response resolves through programmed regulatory mechanisms, restoring tissue homeostasis [4–9].

When this balance is disrupted, inflammation becomes chronic and destructive, leading to attachment loss, alveolar bone resorption, and eventual tooth loss. The pathogenesis of IPD is characterized by excessive production of pro-inflammatory mediators such as interleukin-1β, tumor necrosis factor-α, and prostaglandin E2, as well as activation of matrix metalloproteinases [10–11]. In addition to microbial factors, host susceptibility plays a major role. Genetic predisposition, smoking, diet, systemic diseases, and impaired immune regulation contribute to disease progression. Chronic periodontitis is now recognized not only as a localized oral disease but also as a systemic inflammatory disorder influencing distant organs.

Fatty liver dystrophy (FLD) is a metabolic liver disorder characterized by accumulation of triglycerides in hepatocytes exceeding 5–10% of liver weight. It may occur due to increased fatty acid uptake, reduced β-oxidation, or impaired lipoprotein export from the liver [12–20].Several mechanisms contribute to fat accumulation: Excessive dietary fat intake, leading to elevated chylomicron remnants absorbed by hepatocytes. Disturbance of fatty acid oxidation in mitochondria and peroxisomes, resulting in triglyceride synthesis. Defective triglyceride export due to impaired formation of very-low-density lipoproteins (VLDL). High carbohydrate intake, enhancing de novo lipogenesis [14, 21–26].

Clinically, FLD is more frequently diagnosed in middle-aged and elderly individuals, with a male predominance [12, 17, 27–30]. However, pediatric cases are also increasingly reported [31–33]. The condition may progress to steatohepatitis, fibrosis, and cirrhosis if left untreated.

Recent research views fatty liver dystrophy as part of a systemic metabolic-inflammation continuum, sharing pathogenic mediators with other chronic inflammatory conditions, including periodontitis.

Result.

Pathogenetic Interrelation Between Periodontal Disease and Fatty Liver Dystrophy A growing body of evidence supports a bidirectional link between IPD and FLD. Both conditions are characterized by chronic low-grade inflammation, oxidative stress, and immune dysregulation. Periodontal inflammation results in the release of cytokines (IL-1β, IL-6, TNF-α) and bacterial endotoxins into systemic circulation. These mediators can influence hepatic metabolism, promoting lipid accumulation and insulin resistance. Conversely, the inflamed liver releases acute-phase proteins such as C-reactive protein and serum amyloid A, further aggravating systemic inflammation [5–9, 14]. Both IPD and FLD are associated with elevated oxidative stress markers, including malondialdehyde (MDA) and decreased antioxidant enzymes (superoxide dismutase, catalase, glutathione peroxidase). Reactive oxygen species (ROS) contribute to hepatocellular injury and periodontal tissue destruction [17, 23, 25]. Dysbiosis of the oral microbiota in periodontitis leads to increased permeability of the gingival epithelium and systemic dissemination of bacterial components such as lipopolysaccharides (LPS). These molecules activate hepatic Kupffer cells, initiating inflammatory cascades that exacerbate fatty degeneration of the liver [14, 18, 22].

Common risk factors — obesity, diabetes mellitus, smoking, poor oral hygiene, and sedentary lifestyle — contribute to both diseases. Impaired neutrophil function and overproduction of proinflammatory cytokines are typical in both pathologies [20, 27, 30].

Thus, chronic periodontitis may aggravate hepatic steatosis, while liver dysfunction can impair immune regulation in periodontal tissues, creating a vicious cycle of inflammation.

Discussion. Limitations, Challenges, and Perspectives

For patients with FLD, oral examination should include periodontal indices such as OHI-S, PMA, and SBI to assess hygiene and inflammation. Similarly, in patients with chronic periodontitis, evaluation of liver enzymes (ALT, AST) and lipid profile is advisable, since subclinical liver dysfunction may accompany advanced periodontitis [1, 3, 10].

Assessment of oxidative stress biomarkers (MDA, total antioxidant capacity), proinflammatory cytokines (IL-1β, IL-6, TNF-α), and lipid spectrum (HDL, LDL, triglycerides) provides valuable information on systemic involvement. Imaging methods such as ultrasound and elastography are used for liver evaluation.

Treatment should be comprehensive and interdisciplinary. Local therapy: scaling and root planing, antiseptic mouth rinses, laser therapy, and professional hygiene.

Systemic therapy: correction of metabolic disorders, antioxidant supplementation, hepatoprotective drugs, and anti-inflammatory

agents. Lifestyle modification**:** diet optimization, reduced fat and sugar intake, regular physical activity, and smoking cessation. Collaborative management between dentists, hepatologists, and endocrinologists ensures better control of both conditions and reduces the risk of progression.

Despite significant advances, the molecular and causal links between periodontal inflammation and fatty liver dystrophy remain incompletely understood. Future studies should focus on: identifying biomarkers predictive of both oral and hepatic inflammation; evaluating the effects of periodontal therapy on liver function and vice versa; exploring the role of microbiota-derived metabolites and immune signaling pathways in the "oral–liver axis."

Development of integrated diagnostic protocols and preventive programs will enhance early detection and improve patient outcomes.

Conclusion

Inflammatory periodontal diseases and fatty liver dystrophy are interconnected chronic inflammatory disorders sharing common risk factors, pathophysiological mechanisms, and clinical implications. Periodontal inflammation may contribute to hepatic lipid accumulation through systemic cytokine release, oxidative stress, and endotoxemia, while hepatic dysfunction can exacerbate periodontal tissue destruction. An interdisciplinary approach to diagnosis and treatment, involving dental and medical specialists, is essential for effective management. Recognition of this interrelation highlights the systemic nature of oral diseases and supports the concept of comprehensive patient care.

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