

Porphyromonas gingivalis and non-alcoholic fatty liver disease as combined factors of periodontitis

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The periodontium is constantly influenced by various exogenous pathogenic factors, and microorganisms are only one of them. It is impossible to imagine oral cavity without microflora, because it is one of the body biotopes, which is formed 2-3 hours immediately after the birth, and therefore the periodontium has certain adaptive and protective capabilities to such an effect. The integrity of epithelial barrier, constant movement of gingival fluid, local immunity control, the necessary partial pressure of oxygen and the minimum stack layering preserve periodontal health, even if concurrently influenced by several exogenous factors.

The presence of NAFLD in a dental patient is an endogenous factor in reducing the adaptive capacity of the periodontium, initiating and maintaining the inflammatory process through systemic circulation as a source of pro-inflammatory cytokines. Thus, somatic disease facilitates increased activity of specific periodontopathogens, and the occurrence and development of pathological changes in the periodontium.

One of the main order 1 periodontopathogens is *Porphyromonas gingivalis*, which has powerful virulence factors. However, with NAFLD, protective capabilities of the periodontium go down and quantitative and qualitative changes begin to occur with *Porphyromonas gingivalis* i.e. infectivity (increase and growth of microcolonies) and invasion (intratissue penetration). Hypoxia phenomena in the periodontium backed by NAFLD contribute to the growth of anaerobes. *Porphyromonas gingivalis* has direct and indirect mechanisms of periodontal tissue destruction. By secreting special enzymes i.e. gingipains, the bacterium destroys interepithelial contacts, reduces the viscosity of the main substance of the connective tissue, which makes it easier for it to invade.

Purpose: The purpose of this study is to determine the quantitative level of *Porphyromonas gingivalis* in the periodontal pocket and its gingipain virulence factor in patients with non-alcoholic fatty liver disease.

Methods: The study recruited patients with a verified NAFLD diagnosis and somatically healthy patients from the control group. Dental examination was carried out according to the standard methods with the determination of the OHI-S hygienic index, the Papillary-Marginal-Alveolar index (PMA), the Papilla-Bleeding Index (PBI), the Periodontal Index PI (Russell), and the loss of attachment and the depth of gingival probing. The quantitative composition of *Porphyromonas gingivalis* was determined by the method of Quantitative Polymerase Chain Reaction (PCR) in Real Time (qRT-PCR) using universal primers. Gingipain K concentration in oral fluid was determined by immunoenzymatic method using HUMAN GINGIPAIN K (KGP) ELISA KIT (DRG Instruments GmbH, Germany); Statistical data processing was performed using the SPSS statistical program package (version 17.0 for Windows; SPSS, Chicago, IL). The Kolmogorov-Smirnov test was used to test the sign for normality.

Results: The majority of patients with NAFLD (65.7%) were diagnosed with moderate (grade B) Generalized Periodontitis (GP). According to the level of severity of periodontal pathology, it was found that the typical clinical form for 39.9% of patients was grade II GP, while 26.7% of people had grade III GP, and 32.6% did not have inflammatory periodontal pathology at all. In the control group, only one subject had grade II GP, while others had healthy periodontium.

Its highest level was recorded in patients with NAFLD, which was characteristic on the background of decreased adaptation capabilities of the periodontium and the activity of local immune system, as well as an unsatisfactory level of oral hygiene. The proteolytic enzyme gingipain, which is secreted only by *P. gingivalis*, also had the highest values in the main group.

When analysing the correlation interaction, a positive dynamic was found between *P. gingivalis* and the presence of GP ($r=0.652$; $p=0.000$) and between gingipain and GP ($r=0.510$; $p=0.006$).

We've found a significantly positive correlation between *P. gingivalis* and PBI ($r=0.457$; $p=0.002$) and between *P. gingivalis* and probing depth ($r=0.391$; $p=0.009$).

Indeed, an increase in the number of this periodontopathogen, a change in its phenotype and virulence, the secretion of gingipain contributes to the onset of an inflammatory reaction in periodontal tissues, as well as vascular permeability, and therefore the PBI index increases. Conversely, when treating GP, decreased PBI index facilitates the normalization of the *P. gingivalis* value and its subsequent remission. It should be noted that reduced depth of gingival pocket when treating GP helps to eliminate a favourable anaerobic environment for the quantitative growth of *P. gingivalis*.

Conclusion: The analysis and determination of *P. gingivalis*/gingipain correlation ratio with special indices available at a regular dental appointment allows the dentist to monitor the dynamics of inflammation in GP patients and predict the onset and course of remission with subsequent stabilization.

Biography

Emelyanova Natalia is a professor of medicine and together with Dmitry Emelyanov (PhD) work as researchers at a research institute and deal with the problems of manifestations of somatic diseases in the oral cavity. Their research has been featured in many well-known journals and conferences.

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