ORAL MUCOSA INFLAMMATION AND METABOLIC SYNDROME SEVERITY: A CLINICAL-EXPERIMENTAL CORRELATION

Beknazarov B.B
Termez Branch of Tashkent State Medical University, Termez, Uzbekistan

Abstract. This study evaluated the relationship between the intensity of oral mucosa inflammation and the severity of metabolic syndrome (MetS) using a combined clinical and experimental design. In 92 adolescents, gingival inflammation (PMA index), oral hygiene (Green–Vermillion index), periodontal and alveolar bone status (Fuchs index), and enamel electrical conductivity were assessed alongside anthropometric and metabolic parameters; in an experimental rat model, inflammatory infiltration of the gingival mucosa was scored at 30, 60, and 90 days. Inflammation intensity increased in parallel with metabolic disturbance both clinically and experimentally. Significant correlations were found between BMI and PMA ($r=0.61$), waist circumference and the Fuchs index ($r=-0.68$), and blood glucose and enamel conductivity ($r=0.57$) (all $p<0.05$). The results confirm that oral mucosa inflammation is directly linked to MetS severity.

Keywords: metabolic syndrome, oral mucosa, gingival inflammation, PMA index, correlation, severity, periodontal tissues.

Introduction. In metabolic syndrome, chronic low-grade inflammation, microcirculatory disturbance, and oxidative stress create a favorable background for periodontal and mucosal disease. Adipose tissue functions not only as an energy reserve but also as a biologically active endocrine organ that secretes inflammatory mediators. Although clinical studies report a high prevalence of gingivitis and periodontitis in MetS, the quantitative link between inflammation intensity and the severity of metabolic disturbance — and its confirmation in a controlled experimental model — has been insufficiently characterized, particularly in adolescents. This work quantifies that link through parallel clinical and experimental data.

Materials and methods. The clinical part included 92 adolescents (aged 12–18 years; mean 14.8±1.6) with MetS confirmed by internationally accepted clinical and laboratory criteria. Anthropometry (BMI, waist circumference), blood pressure, glucose, and lipid profile were determined. Oral assessment used the PMA index (gingival inflammation), Green–Vermillion index (hygiene), Fuchs index (alveolar bone and periodontal status), and enamel electrical conductivity. The experimental part

used male rats with diet-plus-fructose-induced MetS examined at 30, 60, and 90 days; inflammatory infiltration of the gingival mucosa was graded on a semi-quantitative scale. Correlations were assessed by Pearson's coefficient; results were expressed as M±m and compared with Student's t-test, with significance at $p<0.05$.

Results and discussion. Clinically, oral inflammation was pronounced: periodontal tissue damage was found in 88% of adolescents, and the PMA index rose from 5.9±0.7% in controls to 19±1.2% and 37.1±4.2% in the main group, indicating widespread mild-to-moderate gingivitis. Correlation analysis linked the components of MetS to oral inflammation and destruction: a direct correlation between BMI and PMA ($r=0.61$), a strong inverse correlation between waist circumference and the Fuchs index ($r=-0.68$), and a direct correlation between blood glucose and enamel electrical conductivity ($r=0.57$) (all $p<0.05$). Rising BMI and visceral adiposity were thus associated with intensified gingival inflammation and alveolar bone resorption.

Experimentally, inflammatory infiltration of the gingival mucosa intensified in step with the duration of the model: initial infiltration and perivascular cell accumulation on day 30, chronic infiltration with deepening dystrophic changes on day 60, and maximal inflammatory-destructive remodeling with stromal fibrosis on day 90. The concordance between the clinical gradient (higher indices with greater metabolic burden) and the experimental gradient (heavier infiltration with longer model duration) supports a shared pathogenetic mechanism: adipokine-driven inflammation, microcirculatory disturbance, and tissue hypoxia. This clinical-experimental correlation justifies stratifying patients with MetS into dental risk groups and initiating preventive follow-up early.

Conclusion. The intensity of oral mucosa inflammation is directly and significantly related to the severity of metabolic syndrome, a relationship demonstrated consistently by both clinical indices and experimental morphology. Inflammatory indicators of the oral mucosa can therefore serve as accessible markers of metabolic burden and as a basis for dental risk stratification in adolescents with MetS.

References

1. Nibali L., Tatarakis N., Needleman I., Tu Y.K., D'Aiuto F., Rizzo M., Donos N. Association between metabolic syndrome and periodontitis: a systematic review and meta-analysis // Journal of Clinical Endocrinology & Metabolism. 2013;98(3):913–920. https://doi.org/10.1210/jc.2012-3552

2. Gomes-Filho I.S. et al. Moderate and severe periodontitis are positively associated with metabolic syndrome // Clinical Oral Investigations. 2021;25(6):3719–3727. https://doi.org/10.1007/s00784-020-03699-2

3. Daudt L.D., Musskopf M.L., Mendez M. et al. Association between metabolic syndrome and periodontitis: a systematic review and meta-analysis // Brazilian Oral Research. 2018;32:e35. https://doi.org/10.1590/1807-3107bor-2018.vol32.0035

4. Periodontal diseases and the risk of metabolic syndrome: an updated systematic review and meta-analysis // Frontiers in Endocrinology. 2020;11:336. https://doi.org/10.3389/fendo.2020.00336Khoshimov B.L., Beknazarov B.B. Morphological and clinical evaluation of oral mucosa in adolescents with metabolic syndrome // Problems and Solutions of Scientific and Innovative Research. 2025. Vol. 2(5). P. 132–135.

5. Beknazarov B.B., Usmonova Sh.R., Muxamedova N.H. et al. Periodontal disease and its relationship with metabolic syndrome: a detailed review // Innovations in Science and Technologies. 2024. Vol. 1(7). P. 55–78.

6. Saito T., Shimazaki Y. Metabolic disorders related to obesity and periodontal disease // Journal of Clinical Periodontology. 2005. Vol. 32 (Suppl. 6). P. 254–261.

7. Pischon N., Heng N., Bernimoulin J.-P. et al. Obesity, inflammation, and periodontal disease // Journal of Dental Research. 2007. Vol. 86. P. 400–409.

8. Zimmet P., Alberti K.G.M.M., Kaufman F. et al. The metabolic syndrome in children and adolescents // The Lancet. 2007. Vol. 369. P. 2059–2061.