




Results of clinical efficacy of prevention of inflammatory periodontal diseases in children with obesity

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Abstract

AIM. The aim of the study was to improve the effectiveness of preventing inflammatory periodontal diseases in obese children by developing and implementing a comprehensive preventive program.

MATERIALS AND METHODS. The study included 80 children (40 with varying degrees of obesity and 40 otherwise healthy), aged 6 to 12 years. The first part of the study examined the condition of the periodontal tissues in all subjects. In the second part of the study, a targeted program for the prevention of periodontal diseases was carried out in accordance with the characteristics of the periodontal condition in the examined children.

RESULTS. A targeted program for the prevention of periodontal disease in obese patients has demonstrated high effectiveness, as it has improved clinical indices: the Simplified Green Vermillion Index by 5.4 times, the PI index, PMA (the average value in the subgroup of patients with obesity was 0) and the PBI index (the number of patients with no bleeding in the subgroup of patients with obesity was 100%); improve microcirculation parameters to normal values according to laser Doppler flowmetry (decrease in microcirculation parameter by 7.0 ± 0.05 , increase in the coefficient of variation by 6.5 ± 0.03 and neurogenic tone by 0.5 ± 0.02); to stop inflammatory changes in periodontal tissues according to the immunological study of gingival fluid (decrease in lysozyme by 122.2 ± 14.3 , IgA by 0.25, increase in C3 complement by 0.08), to reduce the incidence of the main periodontopathogenic strains – *Prevotella intermedia* (by 12 times), the incidence of *Porphyromonas gingivalis* and *Treponema denticola* to complete absence.

CONCLUSIONS. The study identified dental characteristics in obese children that indicate proinflammatory and inflammatory changes in periodontal tissues, which is an indication for the prevention of inflammatory periodontal diseases. Thanks to the implementation of the program developed in the study, inflammation in periodontal tissues in children with obesity was suppressed, which was confirmed by the study results and indicates its effectiveness.

Keywords: periodontium, inflammation, prevention, obese children

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Результаты клинической эффективности профилактики воспалительных заболеваний пародонта у детей с ожирением

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Резюме

ЦЕЛЬ. Повысить эффективность профилактики воспалительных заболеваний пародонта у детей с ожирением путем разработки и внедрения комплексной профилактической программы.

МАТЕРИАЛЫ И МЕТОДЫ. В ходе исследования было обследовано 80 детей (40 с различной степенью ожирения и 40 соматически здоровых), в возрасте от 6 до 12 лет. В первой части исследования производилось изучение состояния тканей пародонта у всех обследуемых. Во второй части исследования

была проведена целевая программа профилактики заболеваний тканей пародонта в соответствии с особенностями состояния пародонта у обследованных детей.

РЕЗУЛЬТАТЫ. Целевая программа профилактики заболеваний пародонта для пациентов с ожирением продемонстрировала высокую результативность, так как позволила улучшить показатели клинических индексов: индекса ИГР-У в 5,4 раз, индекса PI, РМА (среднее значение в подгруппе пациентов с ожирением составило 0) и индекса РВІ (количество пациентов с отсутствием кровоточивости в подгруппе пациентов с ожирением составило 100%); улучшить показатели микроциркуляции до нормальных значений по данным лазерной доплеровской флоуметрии (снижение показателя микроциркуляции на $7,0 \pm 0,05$, повышение коэффициента вариации на $6,5 \pm 0,03$ и нейрогенного тонуса на $0,5 \pm 0,02$); купировать воспалительные изменения в тканях пародонта по данным иммунологического исследования десневой жидкости (снижение лизоцима на $122,2 \pm 14,3$, IgA – на 0,25, повышение С3-комплемента на 0,08), снизить встречаемость основных пародонтопатогенных штаммов – *Prevotella intermedia* (в 12 раз), встречаемость *Porphyromonas gingivalis* и *Treponema denticola* до полного отсутствия.

ВЫВОДЫ. В исследовании установлены особенности стоматологического статуса детей с ожирением, указывающие на провоспалительные и воспалительные изменения в тканях пародонта, что является показанием для проведения профилактики воспалительных заболеваний пародонта. Благодаря внедрению разработанной в исследовании программы было купировано воспаление в тканях пародонта у детей с ожирением, что было подтверждено результатами исследования и указывает на её эффективность.

Ключевые слова: пародонт, воспаление, профилактика, дети с ожирением

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INTRODUCTION

At present, the impact of excess body weight, obesity, and metabolic syndrome on periodontal tissue status has been demonstrated by numerous researchers in both experimental and clinical settings [1–4]. A substantial theoretical framework exists describing the pathogenetic mechanisms affecting the oral mucosa, periodontal tissues, and even the hard tissues of the teeth. The quality of life of patients with these conditions is significantly reduced due to the severity and multifactorial nature of these pathologies [5–10].

Therefore, the prevention, prediction, and early prophylaxis of dental diseases are of particular relevance. This patient cohort is predisposed to the development of inflammatory periodontal diseases and requires a selective preventive approach [11–16].

A considerable number of studies confirm the increased need for preventive periodontal care in children with obesity; however, there is a lack of a comprehensive approach to the examination and management of oral diseases in these patients. Furthermore, interdisciplinary collaboration remains insufficient, and standardized dental management algorithms for this population have not yet been established [5; 17–21].

These considerations provided the rationale for investigating this issue and enabled the formulation of the study objective.

AIM

The aim of the study is to improve the effectiveness of preventing inflammatory periodontal diseases in children with obesity through the development and implementation of a comprehensive preventive program.

MATERIALS AND METHODS

A single-center, non-randomized retrospective study was conducted based on a comprehensive examination of 80 children aged 6–12 years. In the first phase of the study, the periodontal status of children with and without somatic pathology was assessed, and the severity of periodontal inflammation was compared depending on the degree of adipose tissue accumulation. This approach allowed for the identification of indications for targeted prevention.

In the second phase, a targeted preventive program for periodontal diseases was developed in accordance with the specific characteristics of periodontal tissue status observed in the examined children.

Accordingly, the following groups were defined:

- **Group 1** ($n = 40$): children with obesity (E66 according to ICD-10);
- **Group 2** ($n = 40$): children without somatic diseases.

To identify correlations between the degree of adipose tissue accumulation and the severity of inflammatory periodontal diseases, subgrouping was performed during the first phase based on the degree of obesity:

- **Subgroup 1:** children with grade I obesity ($n = 10$);
- **Subgroup 2:** children with grade II obesity ($n = 10$);
- **Subgroup 3:** children with grade III obesity ($n = 10$);
- **Subgroup 4:** children with grade IV obesity ($n = 10$);
- **Subgroup 5:** children without somatic pathology ($n = 10$).

During the second phase, a targeted preventive program for periodontal diseases in children with obesity was developed and its effectiveness was evaluated. The following subgroups were formed:

– **Subgroups 1 and 3:** patients who received the preventive program developed in the study;

– **Subgroups 2 and 4:** patients who received a standard preventive program for periodontal diseases.

The preventive program developed in this study included the use of toothpaste containing bromelain and xylitol, application of a bacteriophage-based gel, and the use of an oral irrigator.

Clinical methods included assessment of the DMF index, Oral Hygiene Index-Simplified (OHI-S), and periodontal indices (PMA, PBI, PI). In the first phase, pathomorphological features of inflammation were evaluated using the micronucleus test of gingival epithelium. Laser Doppler flowmetry was employed as a functional diagnostic method in both phases of the study. Laboratory methods included the micronucleus test of gingival epithelium (in the first phase), as well as immunological and microbiological analyses (in both phases).

Statistical analysis was performed using the STATISTICA 6.0 software package (StatSoft Inc., USA) for Windows, with the application of relevant analytical modules as required. Standard descriptive statistics (mode, median, mean, standard deviation, etc.) were calculated. Parametric tests were applied for normally distributed data, whereas nonparametric methods were used otherwise. The statistical analysis included Student's *t*-test, Shapiro–Wilk test, Mann–Whitney U test, Wilcoxon test, and Kolmogorov–Smirnov test.

Comparisons were performed between the control and main groups. In the first phase, comparisons were conducted between two groups across all methods and among four subgroups based on clinical parameters. In the second phase, comparisons were made among four subgroups depending on the presence or absence of somatic pathology and the type of preventive intervention. Cluster analysis was applied to evaluate the effectiveness of the preventive periodontal programs.

RESULTS

Patients in the pediatric population require a selective preventive approach, as they demonstrate more pronounced inflammatory changes in periodontal tissues compared to systemically healthy children. This is confirmed by clinical index data: the mean OHI-S value in children with obesity is 1.9 times higher; the PI index is 13 times higher; the proportion of children without gingival bleeding is reduced by 2.5 times (according to the PBI index); and the mean PMA index is twofold higher. The microbiological prevalence of key periodontopathogenic bacteria is also increased in children with obesity: *Porphyromonas gingivalis* by 19.8 times, *Treponema denticola* by 5.2 times, and *Prevotella intermedia* by 4.2 times.

Laser Doppler flowmetry revealed increased periodontal blood perfusion and signs of vascular stasis associated with inflammation. This was evidenced by a 1.4-fold increase in the microcirculation index in obese patients, a 1.4-fold decrease in erythrocyte flow oscillation, a 1.6-fold increase in the coefficient of variation, and a 1.2-fold reduction in neurogenic vascular tone.

Immunological analysis of gingival crevicular fluid demonstrated cellular markers of periodontal inflammation in children with obesity, including a 1.3-fold increase in lysozyme levels, a 1.7-fold increase in IgA, and a 1.3-fold increase in IgG, along with a 1.2-fold decrease in complement component levels compared to systemically healthy children.

A direct positive correlation was established between the degree of adipose tissue accumulation in children and the severity of periodontal inflammation, as evidenced by progressive increases in OHI-S, PI, PBI, and PMA indices with increasing obesity severity. Specifically, mean OHI-S and PI values in the subgroup with morbid obesity (Subgroup 4) exceeded those in the initial obesity subgroup (Subgroup 1) and in healthy controls by 1.7 times; mean PBI and PMA values in Subgroup 4 differed from Subgroup 1 by 1.5 times.

Based on the micronucleus test of gingival epithelium, children with obesity demonstrated an increased frequency of cytogenetic abnormalities, including micronuclei (5-fold increase), karyolysis (8.3-fold increase), “tongue-shaped” protrusions (12.7-fold increase), and “broken egg” formations (27-fold increase). These findings indicate genomic instability in gingival epithelial cells and are consistent with inflammatory processes in periodontal tissues.

The preventive protocol developed in the study, applied in Subgroups 1 and 3, included the use of a toothpaste containing an enzymatic agent for chemical plaque degradation (bromelain was proposed as the active substance), mechanical oral cleansing via an irrigator, and the application of a bacteriophage-containing gel targeting the oral microbiota. In Subgroups 2 and 4, no modification of oral hygiene measures was implemented, and oral care was performed using conventional hygiene products.

Follow-up examinations were scheduled at 2 weeks, 4 months, 8 months, and 12 months from baseline. Clinical indices (OHI-S, Russell's PI, PBI, and PMA), as well as functional, immunological, and microbiological parameters, were reassessed at each time point.

The dynamics of the OHI-S index demonstrated a statistically significant reduction in mean values after 2 weeks across all study subgroups, indicating the effectiveness of the preventive measures performed in the dental clinic. In Subgroup 1, a reduction of 1.5 ± 1.17 was observed compared to baseline; in Subgroup 2, a reduction of 1.0 ± 1.15 ; in Subgroup 3, a reduction of 1.0 ± 1.12 ; and in Subgroup 4, a reduction of 0.7 ± 1.12 .

Further decreases in the mean index value were observed after 4 months from the start of the study, with subsequent stabilization at a “good” hygiene level according to the interpretation scale in Subgroup 1. In Subgroup 3, no statistically significant changes were detected after 2 weeks, indicating a stable “good oral hygiene” status throughout the observation period. In contrast, Subgroup 2 demonstrated a negative trend starting from 4 months, while Subgroup 4 showed deterioration from 8 months until the end of follow-up.

Overall, the most pronounced and sustained reduction in the OHI-S index was observed in Subgroups 1

and 3, where the final values corresponded to a “good” level of oral hygiene that did not require further correction, confirming the effectiveness of the implemented preventive measures.

Following 2 weeks from baseline, a reduction of the PI index of varying magnitude was observed. The most significant decrease occurred in Subgroup 1 (0.6 ± 1.12) and Subgroup 2 (0.5 ± 1.11). In all groups, index values remained within the range corresponding to the initial stage of periodontal disease. In Subgroups 3 and 4, the mean index decreased to zero, indicating the absence of inflammatory changes in periodontal tissues.

Over time, a continued positive trend in index reduction was observed in Subgroup 1, while Subgroup 3 demonstrated a stable outcome consistent with the absence of periodontal inflammation. In Subgroups 2 and 4, an increase in the mean index was recorded at both 8 and 12 months ($p \leq 0.05$). At the final assessment, the best outcomes – indicating no periodontal inflammation (index value = 0) – were observed in Subgroups 1 and 3, confirming the effectiveness of the proposed preventive program.

Gingival bleeding, as one of the most diagnostically significant preclinical markers of inflammation, was objectively assessed using the PBI index. After 2 weeks, a marked positive dynamic was observed across all subgroups. The proportion of patients without gingival bleeding increased by $42.1 \pm 0.02\%$ in Subgroup 1, $32.2 \pm 0.03\%$ in Subgroup 2, $19.3 \pm 0.01\%$ in Subgroup 3, and $18.3 \pm 0.02\%$ in Subgroup 4.

At 4, 8, and 12 months, sustained improvement was maintained only in Subgroups 1 and 3, whereas Subgroups 2 and 4 demonstrated a negative trend, with a return to baseline levels by the end of the study period. The most pronounced increase in the proportion of patients without gingival bleeding was observed in Subgroups 1 and 3, reaching $100 \pm 0.01\%$ by the end of follow-up. In contrast, $57.3 \pm 0.02\%$ and $41.7 \pm 0.03\%$ of patients in Subgroups 2 and 4, respectively, continued to exhibit varying degrees of gingival bleeding and required ongoing preventive interventions ($p \leq 0.01$).

Analysis of the PMA index revealed that after 2 weeks, a substantial reduction in mean values was observed across all subgroups: 32.0 ± 1.11 , 27.6 ± 1.13 , 16.8 ± 1.12 , and 11.4 ± 1.11 in Subgroups 1–4, respectively, corresponding to a mild degree of gingival inflammation in all examined groups.

At 4, 8, and 12 months, a sustained positive trend characterized by continued reduction in the PMA index was observed only in Subgroups 1 and 3, with total reductions of 57.3 ± 1.12 and 29.1 ± 0.11 , respectively ($p \leq 0.01$). In Subgroups 2 and 4, after the initial improvement at 2 weeks, a gradual return to baseline values was observed, with no statistically significant differences between initial and final assessments.

DISCUSSION

A substantial body of contemporary research has demonstrated a significantly increased prevalence of periodontal diseases in patients with obesity compared to systemically healthy individuals, by approximately

threefold. In addition, among patients presenting with moderate to severe periodontitis, metabolic syndrome occurs 1.9 times more frequently. A direct positive correlation has also been described between the clinical severity of metabolic syndrome and the severity of periodontal disease.

The primary pathogenetic mechanism underlying this association, widely reported in the current scientific literature, is oxidative stress. Adipokine secretion by adipose tissue cells triggers a cascade of inflammatory reactions, which at the molecular level is manifested by suppression of the antioxidant defense system, increased formation of lipid peroxidation products, and disruption of cellular membrane structures. At the tissue level, this process presents as inflammation, while at the organ level it contributes to the development of various forms of periodontal disease within the oral cavity. In turn, the active systemic circulation of lipid peroxidation products exacerbates insulin resistance associated with metabolic syndrome, thereby perpetuating a vicious cycle of metabolic dysfunction.

In the present study, a direct positive correlation was established between the degree of adipose tissue accumulation in children and the severity of periodontal inflammation, as confirmed by progressive increases in mean values of clinical indices (OHI-S, PI, PBI, PMA) with increasing degrees of obesity.

Based on the micronucleus assay of gingival epithelial cells, signs of genomic instability were identified in children with obesity, including an increased frequency of micronucleated cells and a higher accumulation index, indicating cellular genetic instability. It was also established that the frequency of micronucleated epithelial cells in children with obesity exceeded that of healthy controls by 1.42%, which may serve as a diagnostic marker of periodontal inflammation. These findings collectively demonstrate genomic instability in gingival epithelial cells and suggest that the micronucleus test may be used as a diagnostic and prognostic tool for periodontal disease in pediatric patients with obesity.

The results obtained in this study indicate that long-standing chronic inflammation in periodontal tissues leads to alterations in the local immune system of the oral cavity, accompanied by quantitative changes in the investigated parameters.

Lysozyme, a key component of non-specific oral immunity, is synthesized by blood cells including neutrophils, monocytes, and macrophages. Therefore, its concentration in gingival crevicular fluid may serve as an objective indicator of inflammatory processes in periodontal tissues. The results of this study demonstrated increased lysozyme levels in gingival fluid in obese patients compared to systemically healthy individuals.

The complement system plays a significant role in humoral immunity as a complex of protective proteins responsible for the recognition and elimination of foreign infectious and viral agents. Approximately 72% of the total complement proteins are represented by the C3 component, which is involved in the main pathways of complement activation, chemotaxis, phagocytosis, and cytolysis, thereby ensuring non-specific resistance

to infection. The quantitative level of the C3 component is a diagnostically relevant parameter for assessing complement system function and the presence of inflammatory or autoimmune conditions.

In this study, a 1.2-fold reduction ($p \leq 0.05$) in C3 component levels was observed in children with obesity compared to healthy controls, confirming the involvement of the complement system in the immune response. Additionally, elevated levels of IgA (1.7-fold increase) and IgG (1.3-fold increase) were recorded in obese patients compared to healthy individuals ($p \leq 0.05$), indicating an early stage of pathological changes in periodontal tissues in pediatric patients with obesity.

The aim of the microbiological investigation was to assess the qualitative composition of periodontopathogenic flora belonging to the so-called “red complex”, which represents the highest risk for periodontal tissues, and to compare its prevalence between children with obesity and systemically healthy controls. The presence of key periodontopathogenic bacteria, including *Porphyromonas gingivalis*, *Treponema denticola*, and *Prevotella intermedia*, was evaluated.

The study demonstrated that the prevalence of “red complex” bacteria – *Porphyromonas gingivalis* and *Treponema denticola*, both of which are associated with intensive periodontal tissue destruction – was significantly higher in the obesity group compared to healthy children. *Prevotella intermedia*, recognized as a predictor of inflammatory periodontal diseases such as gingivitis and periodontitis, was detected in $63.3 \pm 0.02\%$ of children with obesity and in $15.0 \pm 0.01\%$ of healthy children ($p \leq 0.05$). These findings indicate a high risk of development and progression of periodontal disease in pediatric patients with obesity.

Laser Doppler flowmetry revealed increased periodontal blood perfusion and venous stasis associated with inflammation, as evidenced by a 1.4-fold increase in the mean perfusion (PM) in obese patients compared to healthy children, a 1.4-fold decrease in σ , a 1.6-fold increase in the coefficient of variation (Kv), and a 1.2-fold reduction in neurogenic vascular tone. The use of laser Doppler flowmetry enabled the assessment of periodontal microcirculation in both obese and systemically healthy pediatric patients, allowing comparative evaluation of microvascular dysfunction and identification of the need for preventive interventions.

All the above findings demonstrate a direct relationship between obesity and inflammatory processes in periodontal tissues.

Thus, the study identified specific characteristics of the dental status in children with obesity, indicating both pro-inflammatory and inflammatory changes in periodontal tissues, which justifies the need for preven-

tive periodontal interventions. To address this requirement, a selective preventive program was developed and implemented, taking into account both the general somatic and local dental status of the patients.

The targeted preventive program for patients with obesity demonstrated high effectiveness, as evidenced by: improvement in clinical indices, including a 5.4-fold reduction in the mean OHI-S; complete resolution of periodontal inflammation according to PI and PMA indices (mean values in the obesity subgroup reached 0); normalization of bleeding status according to the PBI index (100% of patients in the obesity subgroup exhibited absence of gingival bleeding); improvement of microcirculatory parameters to near-normal values according to laser Doppler flowmetry (PM decreased by 7.0 ± 0.05 , Kv increased by 6.5 ± 0.03 , and neurogenic tone by 0.5 ± 0.02); and reduction of inflammatory changes in gingival crevicular fluid, including a decrease in lysozyme levels by 122.2 ± 14.3 , a reduction in IgA by 0.25, and an increase in C3 complement by 0.08.

Furthermore, a significant reduction in the prevalence of key periodontopathogenic microorganisms was observed, including a 12-fold decrease in *Prevotella intermedia*, and complete elimination of *Porphyromonas gingivalis* and *Treponema denticola*.

The implementation of the developed preventive program resulted in the elimination of periodontal inflammation in children with obesity, as confirmed by the study findings, thereby demonstrating its high clinical effectiveness.

CONCLUSION

1. Children with obesity require a selective, risk-adapted approach from dental practitioners, as they demonstrate a higher predisposition to the development of inflammatory periodontal diseases.

2. In the management of pediatric patients with obesity, a comprehensive approach to the diagnosis, prevention, and treatment of periodontal diseases is recommended. This approach should include consideration of the general somatic status (degree and phenotype of obesity, presence of complications, etc.), local oral health status (presence and severity of periodontal pathology, oral hygiene level, presence of den-toalveolar anomalies, etc.), and psychological status (temperament, treatment adherence, motivation for preventive care, etc.). In addition, active interdisciplinary collaboration and patient referral to pediatricians and endocrinologists should be ensured.

3. The targeted preventive periodontal program developed for children with obesity may also be indicated for pediatric patients with related conditions, including overweight, metabolic syndrome, and type 2 diabetes mellitus.

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