CLINICAL COURSE OF CHRONIC GENERALIZED PERIODONTITIS IN OBESE PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Type 2 diabetes mellitus (T2D) and obesity enhance systemic inflammation, microcirculation and immune disorders, which can make the course of chronic generalized periodontitis more severe. Comparative assessment of clinical and radiological manifestations of the disease in individuals with these conditions is important for interdisciplinary management of patients. The study aimed to conduct comparative assessment of clinical and radiological manifestations of chronic generalized periodontitis (CGP) in patients with T2D, obesity and having no somatic disorders. A total of 90 patients with moderate CGP aged 35–60 were assessed, who were stratified into three groups (30 individuals per group) matched for gender and age: without any somatic disorders, with T2D and obesity. OHI-S, SBI, periodontal pocket depth (PPD), clinical attachment loss (CAL), and radiological signs of bone resorption were assessed; the analysis of correlations with HbA1c, BMI, and lipid profile was conducted. OHI-S was 1.8 ± 0.3 in group I, 2.3 ± 0.4 in group II, and 2.2 ± 0.5 in group III; SBI was 42 ± 9%, 61 ± 11%, and 56 ± 10%, respectively ($p < 0.05$ for groups II and III compared to group I). PPD and CAL were higher in obese patients with T2D, than in patients having no somatic disorders ($p < 0.05$), while the differences between groups II and III were non-significant (for PPD $p = 0.09$). HbA1c levels were correlated to PPD ($r = 0.42$), CAL ($r = 0.39$), and SBI ($r = 0.36$); BMI was correlated to PPD ($r = 0.33$) and SBI ($r = 0.35$) ($p < 0.05$). Thus, T2D and obesity are associated with the more adverse clinical and radiological manifestations of CGP; it is necessary to consider poorer oral hygiene in patients with comorbidities when interpreting intergroup differences.

Keywords: chronic generalized periodontitis, type 2 diabetes mellitus, obesity, comorbidity, systemic inflammation, metabolic syndrome, HbA1c, alveolar bone loss

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Compliance with ethical standards: the study was approved by the Ethics Committee of the North Ossetian State Medical Academy (protocol No. 6 dated 18 March 2025). All subjects submitted the informed consent for participation in the study and personal data processing.

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КЛИНИЧЕСКОЕ ТЕЧЕНИЕ ХРОНИЧЕСКОГО ГЕНЕРАЛИЗОВАННОГО ПАРОДОНТИТА У ПАЦИЕНТОВ С САХАРНЫМ ДИАБЕТОМ 2-ГО ТИПА И ОЖИРЕНИЕМ

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Сахарный диабет 2-го типа (СД2) и ожирение усиливают системное воспаление, нарушения микроциркуляции и иммунного ответа, что может утяжелять течение хронического генерализованного пародонтита. Сравнительная оценка клинико-рентгенологических проявлений заболевания при этих состояниях важна для междисциплинарного ведения пациентов. Целью исследования было провести сравнительную оценку клинико-рентгенологических проявлений хронического генерализованного пародонтита (ХГП) у пациентов с СД2, ожирением и без соматической патологии. Обследовали 90 пациентов 35–60 лет с ХГП средней степени тяжести, разделенных на три сопоставимые по полу и возрасту группы по 30 человек: без соматической патологии, с СД2 и с ожирением. Оценивали OHI-S, SBI, глубину пародонтальных карманов (ГПК), потерю клинического прикрепления (КУП) и рентгенологические признаки резорбции кости; выполняли корреляционный анализ с HbA1c, ИМТ и липидным профилем. Индекс OHI-S составил 1,8 ± 0,3 в I группе, 2,3 ± 0,4 во II и 2,2 ± 0,5 в III; SBI — 42 ± 9%, 61 ± 11% и 56 ± 10% соответственно ($p < 0,05$ для II и III групп по сравнению с I). ГПК и КУП были выше у пациентов с СД2 и ожирением, чем у пациентов без соматической патологии ($p < 0,05$), тогда как различия между II и III группами не достигали статистической значимости (для ГПК $p = 0,09$). Уровень HbA1c коррелировал с ГПК ($r = 0,42$), КУП ($r = 0,39$) и SBI ($r = 0,36$), а ИМТ — с ГПК ($r = 0,33$) и SBI ($r = 0,35$) ($p < 0,05$). Таким образом, СД2 и ожирение ассоциированы с более неблагоприятными клинико-рентгенологическими проявлениями ХГП; интерпретация межгрупповых различий должна учитывать худшее гигиеническое состояние полости рта у коморбидных пациентов.

Ключевые слова: хронический генерализованный пародонтит, сахарный диабет 2-го типа, ожирение, коморбидность, системное воспаление, метаболический синдром, HbA1c, резорбция альвеолярной кости

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Chronic generalized periodontitis (CGP) remains one of the most common inflammatory disorders of the oral cavity. In is more and more often considered as a component of the systemic inflammatory continuum. According to the GBD 2021 analysis, the number of cases of severe periodontitis in the world increased by 91.54% in 1990–2021, which emphasizes stable medical and social significance of the disorder [1]. In this context, the growing prevalence of metabolic disorders, primarily type 2 diabetes mellitus (T2D) and obesity, reinforces the urgency of multidisciplinary analysis of periodontal tissue damage [2, 3].

T2D is associated with chronic hyperglycemia, accumulation of advanced glycation end products, impaired microcirculation, and immune dysfunction. These mechanisms make periodontal tissues more susceptible to inflammation, accelerate the clinical attachment loss, and enhance the alveolar bone resorption [4–6]. According to the data of the systematic review and meta-regression analysis of prospective studies, diabetes is associated with higher risk of periodontitis [7], and a modern review reports the increase in the risk of the disease occurrence or progression by 86% (RR 1.86; 95% CI 1.3–2.8) [5].

Obesity is considered as an independent risk factor of inflammatory periodontal disorders. Adipokines and pro-inflammatory cytokines of the adipose tissue support chronic subclinical inflammation, alter tissue reactivity; these also can make the course of periodontitis more severe [8–13]. In one of the studies, among 314 obese young adult patients, gingivitis was found in 63.7% of cases, and stage III periodontitis in 22.6% of cases; the increase in body mass index was positively correlated to the increase in PD, CAL, RD, and PI ($p < 0.05$) [11]. In the published systematic review and meta-analysis, obesity was associated with the increased risk of periodontitis (OR 1.31; 95% CI 1.22–1.41) [14].

Systemic inflammation, endothelial dysfunction, and metabolic disorders provide the common pathogenetic background for periodontitis, T2D, obesity, and cardiovascular disorders [15]. At the same time, direct comparisons of the CGP clinical and radiological phenotype in patient with T2D and obese patients within the same diagnostic protocol are limited. Such comparative assessment is important for the clinical routing of patients and clarification of the factors associated with the more severe disease course. The study aimed to conduct comparative assessment of clinical and radiological manifestations of CGP in patients with T2D, obesity and those having no somatic disorders.

METHODS

A total of 90 patients (42 males and 48 females) aged 35–60 (average age 47.3 ± 6.1 years) with the diagnosis of moderate chronic generalized periodontitis, exacerbation or unstable remission, were included in the comparative cross-sectional clinical trial. All patients were divided into three groups, 30 individuals per group: group I — patients with CGP without any defined somatic disorder; group II — patients with CGP against the background of T2D; group III — patients with CGP against the background of obesity. The groups formed were matched for gender and age.

Inclusion criteria: age 35–60 years, at least 20 teeth, and the confirmed diagnosis of moderate CGP. Patients with acute inflammatory disorders of the oral cavity, severe decompensation of concomitant conditions, and the combination of T2D and obesity were not included in the study. The groups were allocated based on the somatic status and the data on the presence of T2D or obesity from medical records. The smoking status and other behavioral factors were not used as the criteria for stratification of the groups, which was taken into account as a potential limitation of the study when interpreting the results.

Clinical assessment included estimation of the simplified oral hygiene index (OHI-S), sulcus bleeding index (SBI), periodontal pocket depth (PPD), clinical attachment loss (CAL). Measurement was performed using a standard periodontal probe at six points near each tooth. CAL was calculated as the sum of the periodontal pocket depth and the value of the gingival recession/displacement of the gingival margin relative to the cement-enamel junction. Radiological assessment of the alveolar bone condition was performed based on the digital orthopantomography data and targeted intraoral radiographs obtained using the standard protocol in the clinic X-ray room. In patients of group II, the T2D duration, glucose-lowering therapy pattern, and HbA1c levels were also assessed; in patients of group III, body mass index (BMI) and the lipid profile indicators were evaluated.

Statistical data processing was conducted using the standard software package. The data are presented as M ± SD. The distribution was tested for normality using the Shapiro–Wilk test. One-way analysis of variance (ANOVA) with subsequent post-hoc Tukey's test was used to compare quantitative indicators between three groups. Student's *t*-test was used for pairwise comparison, and Pearson's correlation coefficient was used to assess the relationship between traits. The 95% confidence intervals (CI) of the differences of mean values were also calculated for some key intergroup differences. The differences were considered significant at $p < 0.05$.

RESULTS

There were no significant differences in the average age of patients in the groups (I: 46.8 ± 6.0; II: 47.5 ± 5.9; III: 47.7 ± 6.5 years; $p > 0.05$). The share of males was 43.3% in group I, 46.7% in group II, and 46.7% in group III ($p > 0.05$), which suggested that the groups were comparable based on major demographic characteristics.

In group II, the average T2D duration was 9.2 ± 3.8 years; the combination glucose-lowering therapy was reported in 63.3% of cases, and insulin therapy was reported in 36.7% of cases. The average HbA1c level was 8.1 ± 1.0%, which corresponded to insufficient glycemic control in a large number of patients. In group III, BMI was 33.8 ± 3.4 kg/m^2; class II obesity was diagnosed in 46.7% of patients, and class III obesity was reported in 23.3%. Hypercholesterolemia and atherogenic dyslipidemia (increased levels of triglycerides and LDL cholesterol, decreased HDL cholesterol levels) were typical for this group.

The gum hygiene and inflammation indicators were worse in patients with somatic comorbidities. OHI-S was higher in groups II and III (2.3 ± 0.4 and 2.2 ± 0.5 points, respectively) compared to group I (1.8 ± 0.3; $p < 0.05$). The difference between the mean OHI-S values was 0.50 points (95% CI 0.32–0.68) for II–I and 0.40 points (95% CI 0.19–0.61) for III–I. SBI was 42 ± 9% in group I, 61 ± 11% in group II, and 56 ± 10% in group III; the differences II–I and III–I were significant ($p < 0.05$). The difference between the mean SBI values was 19 percentage points (95% CI 13.8–24.2) between groups II and I and 14 percentage points (95% CI 9.1–18.9) between groups III and I. Diffuse gingival bleeding and episodes of spontaneous bleeding with minimal trauma were more often found in patients with T2D, while in obese patients the localized areas of hyperemia and hypertrophy of the interdental papillae with the pronounced bleeding upon probing prevailed.

The periodontal pocket depth and clinical attachment loss values were higher in groups II and III compared to group I. The differences between groups II and I, as well as between groups III and I were significant ($p < 0.05$). There were no significant differences in PPD between groups II and III ($p = 0.09$), and the differences in CAL were also non-significant ($p > 0.05$).

The X-ray examination more often revealed infrabony pockets and uneven resorption of the interalveolar septa, while the combination of the horizontal and focal vertical resorption, especially in the area of multi-rooted teeth, prevailed in obese patients. In group II, a moderate positive correlation between HbA1c levels and the periodontal pocket depth ($r = 0.42$; $p < 0.05$), clinical attachment loss ($r = 0.39$; $p < 0.05$), and SBI ($r = 0.36$; $p < 0.05$) was revealed. In group III, there was a moderate correlation of BMI with PPD ($r = 0.33$; $p < 0.05$) and SBI ($r = 0.35$; $p < 0.05$), and triglyceride levels were correlated to the bone resorption severity ($r = 0.31$; $p < 0.05$).

Thus, in this cross-sectional study T2D and obesity were associated with the less favorable clinical and radiological characteristics of CGP; some intergroup differences could be due to poorer oral hygiene in patients with comorbidities.

DISCUSSION

The data obtained confirm that metabolic disorders considerably modify the CGP course. The less favorable indicators in patients with T2D are consistent with the modern reviews and meta-analyses emphasizing the role of chronic hyperglycemia, advanced glycation end products, oxidative stress, and microcirculation disorders in the progression of periodontitis; in particular, the risk of periodontitis development or progression in patients with diabetes was estimated to be 86% higher (RR 1.86; 95% CI 1.3–2.8) [5].

The identified correlation of HbA1c with the pocket depth, attachment loss, and bleeding suggests the clinical significance of carbohydrate metabolism compensation. This is in line with the concept of bidirectional relation between diabetes and periodontitis, with which inflammation in periodontal tissues maintaining the systemic pro-inflammatory background can make it difficult to achieve glycemic targets [2–6].

In obese patients, a significant worsening of the CGP course compared to the group without somatic disorders was also observed. This is consistent with both distinct case reports and modern reviews. Thus, among obese young adult patients gingivitis was reported in 63.7% of cases, and stage III periodontitis in 22.6% of cases; the increase in BMI was accompanied by the increase in PD, CAL, RD, and PI ($p < 0.05$) [11]. According to the results of the published systematic review and meta-analysis [14], obesity is associated with the increased risk of periodontitis (OR 1.31; 95% CI 1.22–1.41). The association of BMI and triglycerides with periodontal indicators revealed in our study further confirms the involvement of the adipose tissue hormonal and metabolic disorders in the periodontal destruction progression.

When directly comparing the groups with T2D and obesity, the differences in PPD and CAL were non-significant. That is why we cannot declare a definitely more severe overall CGP course in individuals with T2D compared to obese ones based on our data. At the same time, a diffuse bleeding pattern, episodes of spontaneous hemorrhage with minimal trauma, more frequent vertical bone defects, and a correlation of periodontal indicators with HbA1c were more typical for patients with T2D, which suggests differences in the periodontal tissue lesion clinical phenotype. This is likely to reflect differences in predominant pathogenetic mechanisms. However, systemic inflammation linking periodontitis to metabolic syndrome and cardiovascular risk remains a common link for both disorders [15].

The relatively small sample size, cross-sectional design, and the lack of a separate group of patients with the combination of T2D and obesity should be considered the study limitations, which make it impossible to assess a possible synergistic effect of these conditions. The number of patients with a combination of disorders not included in the sample was not separately recorded within the protocol. Furthermore, the groups were not stratified based on the smoking status, and higher OHI-S values in groups II and III indicate a possible confounding influence of behavioral factors and the quality of personal hygiene. This limits the interpretation of the identified intergroup differences as an independent effect of metabolic disorder alone. Prospective studies with the inclusion of local and systemic inflammation biomarkers, a separate group of patients with a combination of T2D and obesity, and a multifactorial adjustment for behavioral risk factors are promising.

CONCLUSIONS

The cross-sectional comparative study revealed poorer oral hygiene and more severe gum inflammation in patients with T2D and obesity, than in patients without any somatic disorder: OHI-S was 2.3 ± 0.4 and 2.2 ± 0.5 vs. 1.8 ± 0.3 points, SBI was 61 ± 11% and 56 ± 10% vs.42 ± 9%, respectively ($p < 0.05$). PPD and CAL were higher in the groups with T2D and obesity, than in the group without somatic disorders ($p < 0.05$), but there were no significant differences in PPD between T2D and obesity ($p = 0.09$), and the differences in CAL were also non-significant. Correlations of HbA1c with PPD ($r = 0.42$), CAL ($r = 0.39$), and SBI ($r = 0.36$) were typical for the T2D group, which suggests the relationship between the degree of carbohydrate metabolism compensation and severity of periodontal changes. The findings should be interpreted considering higher levels of dental plaque in the groups with comorbidities and the lack of stratification by smoking; at the same time, patients with CGP and metabolic disorders need multidisciplinary management involving dentist, endocrinologist, and general practitioner.

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