Asian Journal of Periodontics and Orthodontics
2026, Volume 6, Issue 1, Page No: 1-12
Copyright CC BY-NC-SA 4.0
Available online at: www.tsdp.net

ISSN: 3062-3499

Cross-Sectional Study

Clinical and Laboratory Characteristics of Periodontal Disease in Adolescents with Type 1 Diabetes

Georgiy Khetagovich Kibizov[1], Yana Makharbekovna Bedoeva[1], Angelina Zoltanovna Kazieva[1]*, Lana Rafikovna Makieva[1], Irina Aleksandrovna Tokaeva[1], Diana Olegovna Gudieva[1], Iman Alamovna Muzaeva[1], Alexandr Elkanovich Kharebov[1], Anastasia Igorevna Shmailova[2], Maryam Muradovna Alangerieva[3]

[1]Faculty of Pediatrics, North Ossetian State Medical Academy, Vladikavkaz, Republic of North Ossetia-Alania, Russia.
[2]Faculty of Dentistry, Ryazan State Medical University named after Academician I.P. Pavlov, Ryazan, Russia.
[3]Faculty of Pediatrics, Chechen State University named after A.A.Kadyrov, Grozny, Republic of Chechnya, Russia.

***E-mail** ✉ kazieva.a2002@gmail.com

Received: 17 November 2025; **Revised:** 26 February 2026; **Accepted:** 27 February 2026

ABSTRACT

This study aimed to comprehensively assess the impact of type 1 diabetes mellitus (T1DM) on the periodontal status of adolescents. A cross-sectional comparative study involved 180 adolescents aged 12–18 years, divided into groups: patients with compensated T1DM (n=80), conditionally healthy individuals (n=80), and adolescents with periodontitis associated with poor oral hygiene (n=20). The methodology included clinical assessment of periodontal indices (BOP, PPD, CAL), quantitative determination of key periodontopathogenic bacteria (*Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*) using real-time PCR, and analysis of biochemical markers (interleukin-1β, matrix metalloproteinase-8, osteoprotegerin) in oral fluid. The results revealed that adolescents with T1DM, compared to their healthy peers, had a 2.5 times higher bleeding index (BOP: 25.0% vs. 10.5%, $p<0.001$), an elevated concentration of IL-1β in saliva (65.8 pg/mL vs. 25.5 pg/mL, $p<0.001$), and an 11.6-fold increase in *P. gingivalis* levels (5.8×10^3 vs. 0.5×10^3 copies/mL, $p<0.001$). At the same time, no significant loss of clinical attachment was observed, indicating a predominance of severe gingivitis over destructive periodontitis. A direct correlation was established between the level of glycated hemoglobin (HbA1c) and inflammatory parameters: with BOP ($\rho=0.47$) and with IL-1β ($\rho=0.52$). The obtained data indicate that T1DM in adolescents is a significant risk factor for the development of pronounced periodontal inflammation. Its pathogenesis is primarily associated with a systemic pro-inflammatory status induced by hyperglycemia, rather than with an aggressive microbial factor. The results justify the necessity of mandatory inclusion of periodontal screening and preventive dental interventions into the standards of interdisciplinary management for adolescents with T1DM.

Keywords: Type 1 diabetes mellitus, Adolescents, Periodontitis, Gingivitis, Interleukin-1β, *Porphyromonas gingivalis*

How to Cite This Article: Kibizov GK, Bedoeva YM, Kazieva AZ, Makieva LR, Tokaeva IA, Gudieva DO, et al. Clinical and Laboratory Characteristics of Periodontal Disease in Adolescents with Type 1 Diabetes. Asian J Periodont Orthodont. 2026;6(1):1-12. https://doi.org/10.51847/EgocWZMp5C

Introduction

Type 1 diabetes mellitus (T1DM) is a chronic autoimmune disease characterized by an absolute insulin deficiency [1]. Unlike type 2 diabetes, which manifests predominantly in adulthood, T1DM is traditionally considered a disease of the young, with a peak incidence during childhood and adolescence [2, 3]. In recent decades, the global medical community has encountered an unprecedented phenomenon: a

© 2026 Asian Journal of Periodontics and Orthodontics

steady increase in the incidence of T1DM among children and adolescents, which has taken on the character of a global epidemic [4-6]. This rise is observed in almost all countries worldwide; however, its dynamics and absolute rates demonstrate pronounced geographical and temporal heterogeneity, pointing to a complex interaction between genetic predisposition and environmental factors [7].

Historical data clearly illustrate the scale and features of this epidemic. As seen from data aggregated across key world regions, the overall trend has been upward over the past 70 years **(Figure 1)**. Western European nations have generally had the greatest rates [8]. However, the most notable relative growth was observed during the end of the 20th and start of the 21st century in areas with initially low prevalence, like China, India, and Eastern Europe [9]. For instance, in the Russian Federation, the incidence increased from approximately 1.8 cases per 100, 000 children in the 1970s to a projected 22.0 by 2025. In China, the rate increased tenfold over the same period [10]. This growing cohort of patients remains in a state of chronic hyperglycemia for life, which serves as a trigger for the development of microvascular and macrovascular complications [11].

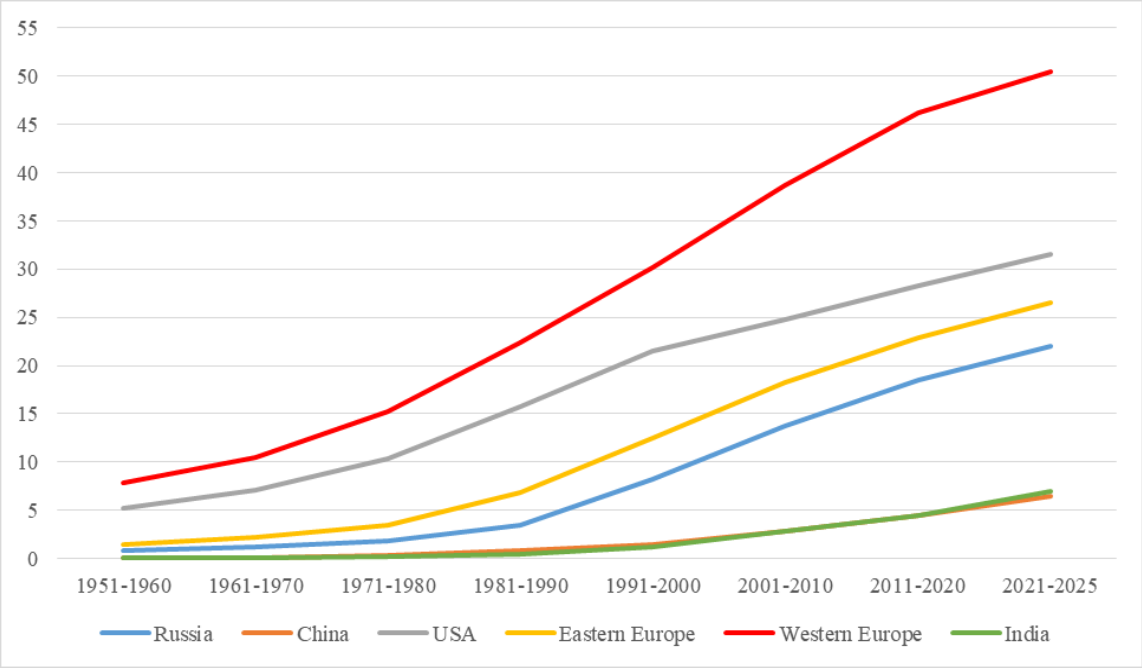


Figure 1. Dynamics of type 1 diabetes mellitus incidence among children aged 0-14 years by world region, 1950s–2020s (new cases per 100, 000 children per year).

One of the significant yet often underestimated complications of T1DM in clinical pediatric practice is periodontal disease [12, 13]. The pathophysiological link between hyperglycemia and periodontal tissue damage is complex [14]. Chronically elevated glucose levels promote non-enzymatic protein glycation with the formation of advanced glycation end products (AGEs) [15]. Their accumulation in periodontal tissues and interaction with specific receptors (RAGE) induce a sustained pro-inflammatory response characterized by hyperproduction of cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β) [16]. Concurrently, neutrophil function, the key first-line cells of antimicrobial defense in the gingival sulcus, is impaired, reducing resistance to bacterial biofilm and promoting dysbiosis [17-19]. Thus, a vicious cycle is formed: systemic inflammation and immune dysfunction associated with diabetes increase the susceptibility of the periodontium to destruction, while chronic local inflammation, in turn, can exacerbate insulin resistance and hinder glycemic control [20].

Despite convincing evidence of a close relationship between diabetes and periodontitis in adults [21-23], the clinical picture and specific features of periodontal damage in adolescents with T1DM remain insufficiently studied. There is a lack of data on whether the systemic impact of diabetes manifests already in adolescence in the form of aggressive gingivitis or early signs of periodontitis, and what the specific features of the periodontal pocket microbiome and the profile of biochemical inflammatory markers in the oral fluid are in this category of patients [24, 25].

This study aimed to conduct a comprehensive comparative assessment of periodontal, microbiological, and biochemical status in adolescents with type 1 diabetes mellitus, their healthy peers, and adolescents with periodontitis due solely to local factors. The objectives of the study included: conducting a comparative analysis of clinical periodontal indices; performing quantitative assessment of key periodontopathogenic bacteria in the contents of periodontal pockets; determining the levels of inflammation markers and bone metabolism markers in oral fluid; establishing correlation relationships between indicators of glycemic control (glycated hemoglobin level, HbA1c) and parameters of inflammation in periodontal tissues. The obtained results are intended to deepen the understanding of early dental manifestations of T1DM and to substantiate the need for developing targeted programs for periodontal monitoring and preventive interventions for this growing and vulnerable patient group.

Materials and Methods

Study design

This cross-sectional comparative study was conducted at the clinical base of the Faculty of Pediatrics of the North Ossetian State Medical Academy (Vladikavkaz, Russia) between March and October 2025. The study was performed in accordance with the ethical principles of the Declaration of Helsinki and local regulatory acts [26]. Written informed consent was obtained from all participants aged 15 years and older, and from participants younger than 15 years; consent was obtained from their legal representatives (parents or guardians), considering the minor's opinion.

Study participants

The study included 180 adolescents aged 12 to 18 years (mean age 15.2 ± 1.8 years), permanently residing in the Republic of North Ossetia-Alania. All participants were divided into three clinically defined groups based on medical history and dental status.

The first group (Group 1, n=80) consisted of conditionally healthy adolescents without a diagnosis of any type of diabetes mellitus or other significant systemic diseases affecting periodontal status (blood disorders, immunodeficiency states, severe systemic connective tissue diseases). The periodontal condition of participants in this group was assessed as clinically healthy or characterized by mild catarrhal gingivitis.

The second group (Group 2, n=80) was formed from patients with a verified diagnosis of "type 1 diabetes mellitus" of at least one year's duration, on constant insulin therapy, and monitored at the Republican Endocrinology Center. The inclusion criterion for this group was a glycated hemoglobin (HbA1c) level of less than 8.5% to minimize the influence of carbohydrate metabolism decompensation on the study results [27]. Patients with the presence of pronounced late complications of diabetes (proliferative retinopathy, nephropathy at the CKD stage) were not included.

The third group (Group 3, n=20) served as a control group with induced local inflammation. It included adolescents without a diagnosis of diabetes mellitus or significant concomitant somatic pathology, but with a clinical picture of generalized mild or moderate periodontitis associated with poor oral hygiene and the presence of multiple carious lesions [28]. The purpose of including this group was to compare microbiological and clinical parameters in inflammation induced primarily by local factors with inflammation against the background of systemic pathology (T1DM).

General exclusion criteria for all groups were: ongoing orthodontic treatment with fixed appliances at the time of the study, intake of antibiotics or anti-inflammatory drugs within the last 3 months, and refusal to participate in the study.

Clinical dental examination

A complete dental examination was performed on each participant, including an assessment of periodontal tissue condition using standard periodontal indices [29]. The examination was performed by one trained and calibrated examiner (kappa coefficient >0.85) using a sterile periodontal probe PCPUNC 15 (Hu-Friedy, USA).

The following parameters were recorded: Silness-Löe Plaque Index (PI) to assess the amount of dental plaque; Bleeding on Probing (BOP) index, expressed as a percentage of the total number of sites examined; Papillary-Marginal-Alveolar (PMA) index in Parma modification; probing pocket depth (PPD) and clinical attachment level (CAL) at six points around each tooth. The Community Periodontal Index (CPI) was used for screening assessment [30-32]. All measurements were recorded in an individual periodontal chart [33-36].

Collection of biological samples and laboratory methods

Two types of biological samples were collected from all participants. The first sample was oral fluid (mixed saliva) for the assessment of general inflammation markers. Collection was performed in the morning on an empty stomach by spitting into a sterile container for 5 minutes. The sample was immediately centrifuged to separate the cellular pellet, and the supernatant was frozen at -70°C for subsequent analysis.

The second sample was a microbiological scraping from the periodontal pocket. In participants of Groups 1 and 2, samples were taken from the gingival sulcus/shallow pocket (PPD ≤ 3 mm) in the area of the first molars. In participants of Group 2 (in the presence of pockets ≥ 4 mm) and all participants of Group 3, material was collected from the deepest periodontal pocket in each segment. Collection was performed using a sterile paper point No. 30, which, after being placed in the pocket area for 20 seconds, was transferred to a sterile Eppendorf-type tube with transport medium.

Total DNA extraction from microbiological scrapings was performed using the "DNA-sorb-B" kit (AmpliSens, Russia) according to the manufacturer's instructions. Quantitative analysis of key periodontopathogenic bacteria content was performed by real-time polymerase chain reaction (RT-PCR) on a "Rotor-Gene Q" detection amplifier (Qiagen, Germany). Absolute concentrations (copies/mL) of the following microorganisms were determined: *Porphyromonas gingivalis* (Pg), *Tannerella forsythia* (Tf), *Treponema denticola* (Td), and *Aggregatibacter actinomycetemcomitans* (Aa). For amplification, commercial reagent kits "AmpliSens® Parodont-screen-FL" (FBUN Central Research Institute of Epidemiology, Rospotrebnadzor, Russia) with hybridization-fluorescence detection were used. Calibration curves were constructed using standard samples of known concentration included in the kits.

In oral fluid samples, concentrations of biomarkers of systemic inflammation and connective tissue destruction, interleukin-1 beta (IL-1β), matrix metalloproteinase-8 (MMP-8), and osteoprotegerin (OPG), were determined by enzyme-linked immunosorbent assay (ELISA) using commercial kits ("IL-1β-ELISA-BEST", "MMP-8-ELISA-BEST", "Osteoprotegerin-ELISA-BEST", Vector-Best, Russia).

Statistical analysis

Statistical data processing was performed using the IBM SPSS Statistics 26.0 software package (IBM Corp., USA). Description of quantitative indicators is presented as median and interquartile range (Me [Q25; Q75]) due to deviation of the distribution of most parameters from normality, tested by the Shapiro-Wilk test. For comparing indicators among the three independent groups, the nonparametric Kruskal-Wallis test was used with subsequent pairwise comparison by Dunn's method with Bonferroni correction. Categorical variables were analyzed using the chi-square (χ^2) test. To assess relationships between clinical, microbiological, and biochemical parameters, Spearman's rank correlation coefficient (ρ) was applied. The level of statistical significance was set at $p < 0.05$ [37-42].

Results and Discussion

Clinical and laboratory examination of 180 adolescents revealed significant differences in periodontal status and associated biochemical parameters among the formed groups.

Clinical and demographic characteristics of the groups

Baseline data of the study participants are presented in **Table 1**. The groups were comparable in age and sex ($p>0.05$). As expected, the median glycated hemoglobin (HbA1c) level was statistically significantly higher in the group of adolescents with type 1 diabetes mellitus (T1DM), while in groups 1 and 3 this indicator was within reference limits. The duration of T1DM in group 2 averaged 5.8 years. The group of conditionally healthy teenagers had the lowest Plaque Index (PI) values, which indicate high hygiene. Hygiene levels in groups 2 and 3 were significantly worse, with group 3 (periodontitis associated with poor hygiene) showing the highest PI values.

Table 1. Clinical and demographic characteristics of the examined adolescents (Me [Q25; Q75])

Parameter	Group 1 (healthy, n=80)	Group 2 (T1DM, n=80)	Group 3 (periodontitis, n=20)	p-value
Age, years	15.0 [13.0; 16.0]	15.0 [14.0; 17.0]	16.0 [14.0; 17.0]	0.128*
Male, n (%)	42 (52.5%)	38 (47.5%)	11 (55.0%)	0.741**
HbA1c, %	5.1 [4.9; 5.3]	7.3 [6.8; 7.9]	5.2 [5.0; 5.4]	<0.001*
Duration of T1DM, years	–	5.8 [3.2; 8.1]	–	–
Plaque Index (PI), scores	0.8 [0.5; 1.1]	1.5 [1.2; 1.9]	2.3 [1.9; 2.6]	<0.001*

Notes: Me – median, [Q25; Q75] – interquartile range. * – Kruskal-Wallis test; ** – χ^2 test. Significant differences ($p<0.05$) between group 2 and others are in bold.

Condition of periodontal tissues

Results of the periodontal examination are presented in **Table 2**. Despite a comparable hygiene level to the healthy group, adolescents with T1DM demonstrated significantly more pronounced signs of inflammation. The Bleeding on Probing (BOP) index in this group

was 2.5 times higher than in group 1 (p<0.001) and was not inferior to the same indicator in the group with localized periodontitis. PMA index values were also significantly higher in groups 2 and 3 compared to the control. At the same time, probing pocket depth (PPD) and clinical attachment loss (CAL) in adolescents with diabetes were significantly lower than in group 3 but exceeded those in healthy peers. This indicates a predominance of the inflammatory component (gingivitis) over the destructive one in T1DM patients against a background of systemic predisposition.

Table 2. Periodontal status indicators in the examined adolescents (Me [Q25; Q75]).

Periodontal Index	Group 1 (healthy, n=80)	Group 2 (T1DM, n=80)	Group 3 (periodontitis, n=20)	p-value*	Pairwise Comparisons (p<0.05)
BOP, %	10.5 [7.0; 15.0]	25.0 [18.0; 35.0]	28.0 [22.0; 40.0]	<0.001	Gr.1 < Gr.2 = Gr.3
PMA Index, %	15.0 [10.0; 22.5]	35.0 [25.0; 48.0]	55.0 [45.0; 65.0]	<0.001	Gr.1 < Gr.2 < Gr.3
PPD, mm	2.0 [2.0; 2.5]	2.5 [2.0; 3.0]	3.5 [3.0; 4.0]	<0.001	Gr.1 < Gr.2 < Gr.3
CAL, mm	0.0 [0.0; 0.0]	0.0 [0.0; 1.0]	2.0 [1.0; 3.0]	<0.001	Gr.1 = Gr.2 < Gr.3

Notes: * – Kruskal-Wallis test. Significant differences from Group 1 are in bold.

Periodontal microbiological profile

Quantitative analysis of key periodontopathogens by RT-PCR revealed a differentiated picture of bacterial colonization **(Table 3)**. The concentration of "red complex" bacteria (*P. gingivalis, T. forsythia, T. denticola*) was minimal in the healthy group. Adolescents with T1DM showed a statistically significant increase in the quantity of all three pathogens compared to group 1, although their median levels remained below the threshold of high pathogenicity. In contrast, the group with localized periodontitis (Group 3) recorded maximum concentrations of Pg and Tf, which is characteristic of a chronic inflammatory-destructive process. *A. actinomycetemcomitans* (Aa) was detected in low titers, with no significant intergroup differences, consistent with its role in aggressive rather than chronic forms of periodontitis.

Table 3. Concentration of periodontopathogenic bacteria in periodontal pockets (RT-PCR, copies/mL × 10^3).

Microorganism	Group 1 (healthy, n=80)	Group 2 (T1DM, n=80)	Group 3 (periodontitis, n=20)	p-value*
***P. gingivalis* (Pg)**	0.5 [0.1; 2.1]	5.8 [2.0; 15.0]	45.2 [21.0; 102.5]	<0.001
***T. forsythia* (Tf)**	1.2 [0.3; 3.8]	8.5 [3.2; 20.1]	38.7 [15.4; 88.0]	<0.001
***T. denticola* (Td)**	0.8 [0.2; 2.5]	4.2 [1.5; 10.8]	15.3 [7.1; 30.5]	<0.001
***A. actinomycetemcomitans* (Aa)**	0.1 [0.0; 0.5]	0.3 [0.0; 1.2]	0.4 [0.0; 2.0]	0.089

Notes: Data presented as Me [Q25; Q75]. * – Kruskal-Wallis test. Significant differences from Group 1 are in bold.

Biochemical markers in oral fluid. Results of ELISA analysis of mixed saliva samples showed activation of inflammatory and destructive processes even in the absence of deep periodontal pockets **(Table 4)**. The level of pro-inflammatory cytokine IL-1β was significantly increased in groups 2 and 3 compared to the control, with the highest values noted in adolescents with T1DM. The concentration of matrix metalloproteinase-8 (MMP-8), a marker of collagen degradation, was maximal in the periodontitis group, correlating with clinical attachment loss. However, in diabetic patients, MMP-8 levels were also elevated relative to normal. An interesting finding was the dynamics of osteoprotegerin (OPG): its concentration was highest in the healthy group and significantly decreased both in T1DM and, especially, in periodontitis, which may indicate depletion of protective anti-resorptive mechanisms.

Table 4. Concentration of biochemical markers in oral fluid (ELISA) (Me [Q25; Q75]).

Biomarker	Group 1 (healthy, n=80)	Group 2 (T1DM, n=80)	Group 3 (periodontitis, n=20)	p-value*
IL-1β, pg/mL	25.5 [18.0; 35.2]	65.8 [45.1; 90.0]	55.3 [40.0; 75.0]	<0.001
MMP-8, ng/mL	45.0 [30.0; 62.0]	85.0 [60.0; 120.0]	180.0 [135.0; 250.0]	<0.001

Osteoprotegerin (OPG), pmol/L	8.5 [6.8; 10.5]	6.0 [4.5; 7.8]	4.2 [3.0; 5.5]	<0.001

Notes: * – Kruskal-Wallis test. Significant differences from Group 1 are in bold.

Correlation analysis

To identify relationships between glycemic control, periodontal inflammation, and laboratory parameters in the group of adolescents with T1DM, Spearman correlation analysis was performed **(Table 5)**. The HbA1c level demonstrated a significant direct correlation of moderate strength with the BOP index and IL-1β concentration in saliva, as well as a weak positive association with MMP-8 level. This confirms that worsening metabolic control is associated with increased local inflammation and activation of destructive processes in the periodontium. Interestingly, diabetes duration correlated primarily with microbiological indicators (Tf, Td) but not with clinical indices, which may indicate a gradual change in the microbiome independent of current hygiene.

Table 5. Correlation relationships (Spearman's ρ) between key parameters in the group of adolescents with T1DM (n=80).

Parameter 1	Parameter 2	Correlation Coefficient (ρ)	p-value
HbA1c	BOP, %	0.47	<0.001
HbA1c	IL-1β in saliva	0.52	<0.001
HbA1c	MMP-8 in saliva	0.28	0.012
T1DM duration	*T. forsythia* concentration	0.31	0.005
T1DM duration	*T. denticola* concentration	0.26	0.021
PI (hygiene)	*P. gingivalis* concentration	0.58	<0.001
BOP, %	IL-1β in saliva	0.61	<0.001

Thus, the obtained results demonstrate that type 1 diabetes mellitus in adolescents is a significant risk factor for the development of pronounced gingivitis, characterized by dysbiosis of the periodontal microbiome with an increase in the pool of pathogenic bacteria, and accompanied by a systemic inflammatory response registered in oral fluid.

This study provides convincing evidence that type 1 diabetes mellitus in adolescents acts as an independent and powerful risk factor for the development of inflammatory periodontal pathology [43, 44]. The obtained data not only confirm this association but also reveal key pathogenetic features that distinguish the periodontal status of adolescents with T1DM from both clinically healthy peers and patients with classical periodontitis induced solely by local factors.

The clinical picture in adolescents with T1DM was characterized by a pronounced inflammatory response with a relatively preserved structure of periodontal attachment. This is confirmed by a significant, 2.5-fold, increase in the bleeding index (BOP: 25.0% vs. 10.5% in the control) and an increase in the PMA index (35.0% vs. 15.0%) in the absence of statistically significant attachment loss (CAL: 0.0 mm), which was characteristic of the periodontitis group (2.0 mm). This pattern corresponds more to severe, generalized gingivitis rather than to established periodontitis [45-47]. This is an extremely important observation, as it indicates that in adolescence, diabetes primarily realizes its pathogenic potential by enhancing the vascular-inflammatory response, rather than by activating destructive processes [48]. Nevertheless, the revealed increase in pocket depth to 2.5 mm against the background of hyperglycemia signals the initial stage of pathological pocket formation, requiring active preventive measures [49].

Data from microbiological analysis and salivary biomarkers shed light on the mechanisms underlying the observed clinical changes. The established increase in the concentration of "red complex" bacteria, in particular P. gingivalis (5.8 $\times 10^3$ copies/mL vs. 0.5 $\times 10^3$ in the control), indicates the development of dysbiosis in the periodontal microbiome [50, 51]. However, this bacterial load was an order of magnitude lower than in the group with manifest periodontitis (45.2 $\times 10^3$ copies/mL), where tissue destruction is the leading factor. This suggests that in adolescents with T1DM, the microbiological shift is more a consequence and a sustaining factor of systemic inflammation, rather than its primary cause [52, 53]. Indirect confirmation of this hypothesis is provided by the identified direct correlation between HbA1c level and the concentration of the pro-inflammatory cytokine IL-1β in saliva ($\rho=0.52$, $p<0.001$) [54]. This cytokine, being a key mediator of inflammation in periodontitis, was elevated in adolescents with diabetes even more strongly (65.8 pg/mL) than in the localized periodontitis group (55.3 pg/mL). Thus, chronic hyperglycemia creates a kind of "inflammatory microenvironment" in periodontal tissues, which, in

turn, modulates the local immune response and the composition of the microflora [55-57].

For a visual representation of the complex interaction of systemic and local factors, a cause-and-effect diagram was compiled, presented in **Figure 2**.

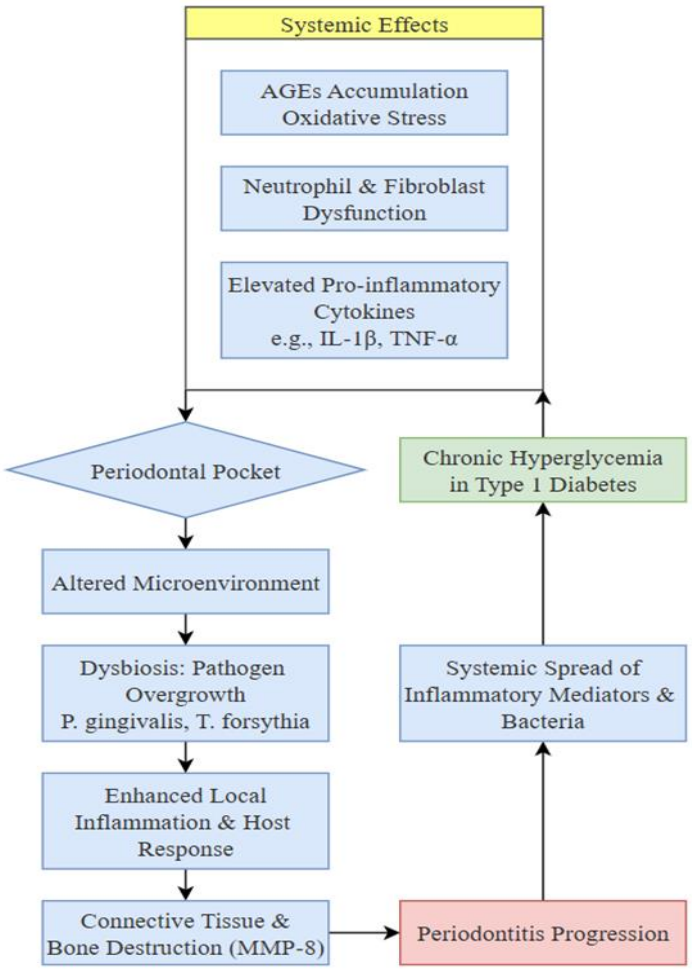


Figure 2. Proposed Model of the Vicious Cycle Linking Type 1 Diabetes and Periodontal Disease Pathogenesis.

The obtained data align with modern concepts of the bidirectional link between diabetes and periodontitis [58-63]. Numerous studies conducted on adult populations have repeatedly demonstrated that patients with diabetes, especially those with poor glycemic control, have a 2-3 times higher risk of developing periodontitis, greater severity of its course, and faster tooth loss [64-67]. For instance, a number of large cohort studies have shown that a 1% increase in HbA1c level increases the risk of periodontitis progression by approximately 20-30% [68-70]. Our results, although obtained from a younger cohort with less pronounced tissue destruction, fully align with this trend: the correlation between HbA1c and gingival bleeding (ρ=0.47) clearly indicates that worsening metabolic control directly exacerbates inflammation in the periodontium already in adolescence [71-75].

Of particular interest are the data on osteoprotegerin (OPG). The decrease in its concentration in the saliva of adolescents with T1DM (6.0 pmol/L vs. 8.5 pmol/L in the control) and, especially, in the periodontitis group (4.2 pmol/L) indicates possible depletion of protective anti-resorptive mechanisms. In recent studies, OPG deficiency has been considered one of the key mechanisms linking diabetes and accelerated bone loss in periodontitis [76-78]. Our data suggest that this mechanism begins to form already at early, pre-destructive stages [79-82].

The limitations of the study should be noted. Its cross-sectional design does not allow for establishing the temporal sequence of events or drawing definitive conclusions about cause-and-effect relationships. Despite adjusting for hygiene level, the influence of behavioral factors cannot be completely ruled out. Nevertheless, the comprehensive approach, including simultaneous assessment of clinical, microbiological, and biochemical markers, provides a holistic understanding of the pathological process.

Thus, the results of this work confirm the hypothesis that type 1 diabetes mellitus in adolescents is a significant risk factor for the development of periodontal diseases. The pathogenesis is associated not so much with an aggressive microbial factor, but with the formation of a background of chronic systemic inflammation due to hyperglycemia, which sharply increases the vulnerability of periodontal tissues even to ordinary bacterial load. The identified correlations between HbA1c and inflammation markers make glycemic control a central element in the prevention of dental complications in this category of patients. The obtained data justify the necessity of including mandatory periodontal screening and preventive interventions in the management standards for adolescents with T1DM.

Conclusion

This study convincingly demonstrates that type 1 diabetes mellitus in adolescents acts as a powerful independent risk factor for the development of pronounced inflammation of periodontal tissues, forming a clinical-pathogenetic profile distinct from both the norm and classical periodontitis. The key finding of the work is the observation that adolescents with T1DM exhibit an intense inflammatory reaction with relative preservation of supporting structures. This is reflected in a statistically significant increase in the gingival bleeding index to 25.0%, which is 2.5 times higher than the indicators of the healthy group (10.5%), against the background of the absence of significant clinical attachment loss (CAL: 0.0 mm vs. 2.0 mm in the periodontitis group). Thus, in adolescence, the pathogenic influence of diabetes is realized primarily through mechanisms of enhanced vascular-inflammatory response, which clinically corresponds to severe generalized gingivitis.

Microbiological and biochemical analyses allowed the elucidation of the mechanisms underlying the observed changes. The identified dysbiosis of the periodontal microbiome, characterized by an increase in the concentration of *Porphyromonas gingivalis* to 5.8×10^3 copies/mL (which is 11.6 times higher than in the control), is a significant but not a leading factor. The critical role is played by the systemic pro-inflammatory state induced by chronic hyperglycemia. This is confirmed by a significant increase in the level of interleukin-1β in oral fluid to 65.8 pg/mL and the presence of a strong direct correlation between glycated hemoglobin level and this marker ($\rho=0.52$, $p<0.001$). The simultaneous decrease in osteoprotegerin concentration to 6.0 pmol/L signals potential depletion of local protective anti-resorptive mechanisms, creating preconditions for future bone tissue destruction. The established relationship between the quality of glycemic control and periodontal status, where the correlation coefficient between HbA1c and the bleeding index was $\rho=0.47$ ($p<0.001$), has fundamental clinical significance.

Consequently, adolescents with type 1 diabetes mellitus represent a special high-risk group for periodontal disease, requiring the development and implementation of a specialized interdisciplinary approach to their management. The foundation of this approach should be mandatory regular periodontal screening integrated into the system of dispensary observation by an endocrinologist. Preventive measures, including motivation for ideal oral hygiene and professional cleanings, should be considered an integral part of the diabetes control strategy. Timely management of gingivitis in adolescence is an effective measure not only for preventing future tooth loss but also for improving overall metabolic status by breaking the pathogenic chain linking chronic oral and systemic inflammation. The obtained data justify the need to revise clinical protocols, highlighting dental health as one of the key parameters of quality of life and long-term prognosis in patients with the onset of T1DM in childhood.

Acknowledgments: The authors are deeply thankful to all the children and their parents who participated in this research for their time, cooperation, and trust.

Conflict of Interest: None

Financial Support: None

Ethics Statement: The study was performed in accordance with the ethical principles of the Declaration of Helsinki and local regulatory acts. Written informed consent was obtained from all participants aged 15 years and older, and from participants younger than 15 years; consent was obtained from their legal representatives (parents or guardians), considering the minor's opinion.

References

1. Aguiar GB, Machado MED, Silva LFD, Aguiar RCB, Christoffel MM. Children with type 1 diabetes mellitus: the experience of disease. Rev Esc Enferm USP. 2021;55:e03725. doi:10.1590/S1980-220X2020011803725
2. Grabia M, Markiewicz-Żukowska R, Socha K. Prevalence of metabolic syndrome in children and adolescents with type 1 diabetes mellitus and

possibilities of prevention and treatment: a systematic review. Nutrients. 2021;13(6):1782. doi:10.3390/nu13061782
3. Maguolo A, Mazzuca G, Smart CE, Maffeis C. Postprandial glucose metabolism in children and adolescents with type 1 diabetes mellitus: potential targets for improvement. Eur J Clin Nutr. 2024;78(2):79-86. doi:10.1038/s41430-023-01359-8
4. Monaghan M, Bryant BL, Inverso H, Moore HR, Streisand R. Young children with type 1 diabetes: recent advances in behavioral research. Curr Diab Rep. 2022;22(6):247-56. doi:10.1007/s11892-022-01465-0
5. Bell KJ, Lain SJ. The changing epidemiology of type 1 diabetes: a global perspective. Diabetes Obes Metab. 2025;27 Suppl 6:3-14. doi:10.1111/dom.16501
6. Almomani BA, Elayyan RN, Al-Shatnawi SF. Type 1 diabetes mellitus in children: patient-reported outcomes. PLoS One. 2025;20(5):e0322882. doi:10.1371/journal.pone.0322882
7. Wagenknecht LE, Lawrence JM, Isom S, Jensen ET, Dabelea D, Liese AD, et al. Trends in incidence of youth-onset type 1 and type 2 diabetes in the USA, 2002-18. Lancet Diabetes Endocrinol. 2023;11(4):242-50. doi:10.1016/S2213-8587(23)00025-6
8. Lawrence JM, Divers J, Isom S, Saydah S, Imperatore G, Pihoker C, et al. Trends in prevalence of type 1 and type 2 diabetes in children and adolescents in the US, 2001-2017. JAMA. 2021;326(8):717-27. doi:10.1001/jama.2021.11165
9. Kandemir N, Vuralli D, Ozon A, Gonc N, Ardicli D, Jalilova L, et al. Epidemiology of type 1 diabetes mellitus in children and adolescents: a 50-year experience. J Diabetes. 2024;16(5):e13562. doi:10.1111/1753-0407.13562
10. Petryaykina EE, Laptev DN, Vorontsova IG, Demidov NA, Ryapolova YA. Diabetes mellitus type 1 in children and adolescents in Moscow. Probl Endokrinol (Mosk). 2021;67(6):113-23. doi:10.14341/probl12795
11. Chen Q, Xu N, Chen Y, Yan M, Tian F, Yang W, et al. Incidence of newly diagnosed type 1 diabetes mellitus in children and adolescents in Henan province. J Clin Res Pediatr Endocrinol. 2022;14(3):287-92. doi:10.4274/jcrpe.galenos.2022.2022-12-4
12. Vlachou S, Loumé A, Giannopoulou C, Papathanasiou E, Zekeridou A. Periodontal disease and type 1 diabetes mellitus: a review. Int J Mol Sci. 2024;25(13):7299. doi:10.3390/ijms25137299
13. Reddy M, Gopalkrishna P. Type 1 diabetes and periodontal disease: a literature review. Can J Dent Hyg. 2022;56(1):22-30.
14. Zhang B, Yang Y, Yi J, Zhao Z, Ye R. Hyperglycemia modulates macrophage polarization in periodontitis. J Periodontal Res. 2021;56(5):991-1005. doi:10.1111/jre.12912
15. Sadovoy VV, Selimov M, Shchedrina T, Nagdalian AA. Nutritional supplement for the control of diabetes. J Excip Food Chem. 2017;8352017:1843.
16. Shcherba V, Krynytska I, Marushchak M, Korda M. Thyroid dysfunction and inflammatory mediators in periodontitis. Endocr Regul. 2021;55(3):131-41. doi:10.2478/enr-2021-0014
17. Bonner M. Evaluation of periodontitis parameters and plaque examination. Front Dent Med. 2024;5:1451698. doi:10.3389/fdmed.2024.1451698
18. Timmerman MF, Van der Weijden GA, Abbas F, Arief EM, Armand S, Winkel EG, et al. Untreated periodontal disease in Indonesian adolescents. J Clin Periodontol. 2000;27(12):932-42. doi:10.1034/j.1600-051x.2000.027012932.x
19. Chapple ILC, Mealey BL, Van Dyke TE, Bartold PM, Dommisch H, Eickholz P, et al. Periodontal health and gingival diseases: consensus report. J Periodontol. 2018;89 Suppl 1:S74-84. doi:10.1002/JPER.17-0719
20. Herrera D, Sanz M, Shapira L, Brotons C, Chapple I, Frese T, et al. Periodontal diseases and systemic diseases: consensus report summary. Eur J Gen Pract. 2024;30(1):2320120. doi:10.1080/13814788.2024.2320120
21. Zhao M, Xie Y, Gao W, Li C, Ye Q, Li Y. Diabetes mellitus and susceptibility to periodontitis. Front Endocrinol (Lausanne). 2023;14:1192625. doi:10.3389/fendo.2023.1192625
22. Wang Q, Nie L, Zhao P, Zhou X, Ding Y, Chen Q, et al. Diabetes fuels periodontal lesions via GLUT1. Int J Oral Sci. 2021;13(1):11. doi:10.1038/s41368-021-00116-6
23. Alwithanani N. Periodontal diseases and diabetes mellitus: a systematic review. J Pharm Bioallied Sci. 2023;15 Suppl 1:S54-63. doi:10.4103/jpbs.jpbs_515_22
24. Chakraborty P, Chowdhury R, Bhakta A, Mukhopahyay P, Ghosh S. Microbiology of periodontal disease in adolescents with type 1 diabetes. Diabetes Metab Syndr.

2021;15(6):102333. doi:10.1016/j.dsx.2021.102333
25. Hopealaakso TK, Thomas JT, Pätilä T, Penttala M, Sakellari D, Grigoriadis A, et al. Periodontitis, metabolic syndrome and diabetes using aMMP-8 test. Diagnostics (Basel). 2024;14(24):2878. doi:10.3390/diagnostics14242878
26. World Medical Association. Declaration of Helsinki: ethical principles for medical research. JAMA. 2025;333(1):71-4. doi:10.1001/jama.2024.21972
27. Dunn TC, Xu Y, Bergenstal RM, Ogawa W, Ajjan RA. Personalized glycated hemoglobin in diabetes management. Diabetes Technol Ther. 2023;25 Suppl 3:S65-74. doi:10.1089/dia.2023.0146
28. Pederson Z, Madrahimov A, Samson K, Reinhardt RA. Association of oral hygiene tracking with dental outcomes. Am J Dent. 2021;34(5):257-60.
29. Pesce P, Menini M, Ugo G, Bagnasco F, Dioguardi M, Troiano G. Periodontal indices in smokers and non-smokers. Clin Oral Investig. 2022;26(7):4701-14. doi:10.1007/s00784-022-04531-9
30. Tripodi D, Cosi A, Valloreo R, Fulco D, Tieri M, Alberi Auber L, et al. Salivary parameters and oral health in athletes. J Int Soc Sports Nutr. 2025;22(1):2443018. doi:10.1080/15502783.2024.2443018
31. Hwang SY, Park JE. Urine pH and periodontal index in Korean adults. Healthcare (Basel). 2024;12(7):740. doi:10.3390/healthcare12070740
32. Singh A, Shrestha A, Bhagat T. Pain perception during periodontal probing. BMC Oral Health. 2021;21(1):82. doi:10.1186/s12903-021-01437-y
33. Constantin VD, Silaghi A, Epistatu D, Dumitriu AS, Paunica S, Bălan DG, et al. Diagnostic and therapeutic insights into colorectal carcinoma. Arch Int J Cancer Allied Sci. 2022;2(1):24-8. doi:10.51847/HojLmKBDvP
34. Mojsak D, Dębczyński M, Kuklińska B, Mróz RM. Ewing's sarcoma in a 58-year-old man. Arch Int J Cancer Allied Sci. 2022;2(1):37-41. doi:10.51847/J1EMRn8tE2
35. Lee MJ, Ferreira J. COVID-19 and children: ethical framework. Asian J Ethics Health Med. 2024;4:1-19. doi:10.51847/haLKYCQorD
36. Kajanova J, Badrov A. Medical students' perspectives on trust in AI. Asian J Ethics Health Med. 2024;4:44-57. doi:10.51847/36mpdZ9AZ8
37. Uneno Y, Morita T, Watanabe Y, Okamoto S, Kawashima N, Muto M. Supportive care needs of elderly cancer patients. Int J Soc Psychol Asp Healthc. 2024;4:13-9. doi:10.51847/o4njwxvRSF
38. Delcea C, Gyorgy M, Siserman C, Popa-Nedelcu R. Cognitive schemas and suicidal behavior. Int J Soc Psychol Asp Healthc. 2024;4:42-6. doi:10.51847/EHCf9HzLEP
39. Essah A, Igboemeka C, Hailemeskel B. Gabapentin for pruritus. Ann Pharm Educ Saf Public Health Advocacy. 2024;4:1-6. doi:10.51847/h8xgEJE3NE
40. Frost N, Deckert PM, Nolte CH, Kohl R, Schreiber SJ. Recruiting patients for clinical trials. Ann Pharm Educ Saf Public Health Advocacy. 2024;4:50-62. doi:10.51847/BLHlqwfTFT
41. Rosellini E, Giordano C, Guidi L, Cascone MG. Novel surgical suture material. J Med Sci Interdiscip Res. 2024;4(1):1-7. doi:10.51847/7denx72XdE
42. Umarova MS, Akhyadova ZS, Salamanova TO, Dzhamaldinova ZI, Taysumova ZD, Bekmurzaeva MR, et al. Effects of vibrations on protein metabolism. J Med Sci Interdiscip Res. 2024;4(1):39-44. doi:10.51847/Jk38F1v5XH
43. Ferizi-Shabani L, Mrasori S, Ferizi V, Barku G, Gjocaj M, Krasniqi B, et al. Dental and periodontal status in children with type 1 diabetes mellitus. Georgian Med News. 2024;(352-353):208-12.
44. Carelli M, Maguolo A, Zusi C, Olivieri F, Emiliani F, De Grandi G, et al. Oral microbiota in children and adolescents with type 1 diabetes mellitus. Microorganisms. 2023;11(3):668. doi:10.3390/microorganisms11030668
45. Ribeiro TR, Silva SM, Martins RARC, Santos CF, Silva PGB, Forti ACE, et al. Salivary immunoglobulin levels and periodontal indices in children with type 1 diabetes. Braz Oral Res. 2024;38:e043. doi:10.1590/1807-3107bor-2024.vol38.0043
46. Dămăşaru MS, Păcurar M, Mariş M, Dămăşaru E, Mariş M, Tilinca CM. Implications of type 1 diabetes in dento-maxillary anomalies. Med Pharm Rep. 2025;98(1):135-43. doi:10.15386/mpr-2822
47. Costa R, Ríos-Carrasco B, Monteiro L, López-Jarana P, Carneiro F, Relvas M. Association between type 1 diabetes mellitus and periodontal diseases. J Clin Med. 2023;12(3):1147. doi:10.3390/jcm12031147
48. Janner M, Saner C. Impact of type 1 diabetes mellitus on bone health in children. Horm Res Paediatr. 2022;95(3):205-14. doi:10.1159/000521627
49. Saotome M, Kuraji R, Numabe Y. Hyperglycemia exacerbates periodontal destruction. J Periodontal Res. 2025;60(6):590-603. doi:10.1111/jre.13366

50. Agho ET, Owotade FJ, Kolawole BA, Oyetola EO, Adedeji TA. Salivary inflammatory biomarkers and HbA1c in diabetes. BMC Oral Health. 2021;21(1):101. doi:10.1186/s12903-021-01453-y
51. Alqaderi H, Hegazi F, Al-Mulla F, Chiu CJ, Kantarci A, Al-Ozairi E, et al. Salivary biomarkers as predictors of obesity and hyperglycemia. Front Public Health. 2022;10:800373. doi:10.3389/fpubh.2022.800373
52. Yanko NV, Kaskova LF, Kulai OO, Khmil OV, Novikova SC. Neutrophil activities in adolescents with type 1 diabetes. Wiad Lek. 2022;75(11 Pt 2):2826-30. doi:10.36740/WLek202211217
53. D'Agostino S, Valentini G, Dolci M. Interleukin levels in type 1 diabetes and periodontitis. Children (Basel). 2024;11(2):238. doi:10.3390/children11020238
54. Oruc AH, Babayiğit O. Salivary asprosin and cytokines in diabetic patients with periodontitis. Clin Oral Investig. 2026;30(1):52. doi:10.1007/s00784-026-06744-8
55. Narukawa Y, Sugiyama N, Miura J, Yamashita R, Tominaga S, Izumi Y, et al. Hyperglycemia and periodontal epithelium permeability. J Periodontal Res. 2023;58(4):813-26. doi:10.1111/jre.13140
56. Sohn Y, Jeong HJ, Park J. Hyperinsulinemia and periodontitis in diabetics. J Dent Res. 2025;104:220345251366952. doi:10.1177/00220345251366952
57. Qin H, Li G, Xu X, Zhang C, Zhong W, Xu S, et al. Oral microbiome in periodontitis under diabetes mellitus. J Oral Microbiol. 2022;14(1):2078031. doi:10.1080/20002297.2022.2078031
58. Rojas P, Soto M, Vargas I. Influence of age on breast cancer prognosis. Asian J Curr Res Clin Cancer. 2022;2(2):33-42. doi:10.51847/AsL4oopFGu
59. Al Abadie M, Sharara Z, Ball PA, Morrissey H. Janus kinase inhibition in autoimmune diseases. Ann Pharm Pract Pharmacother. 2023;3:1-8. doi:10.51847/lhABjfuIwh
60. Guzek K, Stelmach A, Rożnowska A, Najbar I, Cichocki Ł, Sadakierska-Chudy A. Genetic variants and aripiprazole adverse effects. Ann Pharm Pract Pharmacother. 2023;3:40-7. doi:10.51847/ZT28xcs95J
61. Sri KB, Fatima MS, Sumakanth M. UV spectroscopic method for baricitinib. Pharm Sci Drug Des. 2022;2:8-13. doi:10.51847/JxHXkcB6tD
62. Adeleke OA. Liquisolid compact with rifampicin and quercetin. Pharm Sci Drug Des. 2022;2:14-25. doi:10.51847/lw1PmMAVuw
63. Simonyan R, Babayan M, Yekmalyan H, Alexanyan A, Simonyan G, Alexanyan S, et al. Superoxide-generating proteins extraction. Spec J Pharmacogn Phytochem Biotechnol. 2023;3:15-20. doi:10.51847/Vj5MeBCcDs
64. Majo I, Manovijay B, Jacob M, Rajathi P, Fenn SM, Ravi S. Residual periodontal ligament in diabetics. J Indian Soc Periodontol. 2023;27(5):492-5. doi:10.4103/jisp.jisp_522_22
65. Thouvenot K, Turpin T, Taïlé J, Clément K, Meilhac O, Gonthier MP. Insulin resistance and periodontal bacteria. Biomolecules. 2022;12(3):378. doi:10.3390/biom12030378
66. Nie L, Sun Y, Dong H, You M, Cui A, Yue Z, et al. Metabolic memory and periodontal destruction. J Dent Res. 2025;104(13):1495-505. doi:10.1177/00220345251340632
67. Xiang DD, Sun YX, Jiao C, Guo YQ, Fei YX, Ren BQ, et al. Diabetes and periodontitis mechanisms. Stem Cell Res Ther. 2025;16(1):366. doi:10.1186/s13287-025-04441-z
68. Zhao D, Sun Y, Li X, Wang X, Lu L, Li C, et al. Periodontitis and HbA1c in non-diabetics. Healthcare (Basel). 2023;11(19):2649. doi:10.3390/healthcare11192649
69. Chen YF, Zhan Q, Wu CZ, Yuan YH, Chen W, Yu FY, et al. HbA1c and periodontal therapy outcomes. Diabetes Ther. 2021;12(5):1249-78. doi:10.1007/s13300-021-01000-6
70. Banjar A, Alyafi R, AlGhamdi A, Assaggaf M, Almarghlani A, Hassan S, et al. HbA1c and stage of periodontitis. PLoS One. 2023;18(1):e0279755. doi:10.1371/journal.pone.0279755
71. Tsiganock AS, Bgantseva AE, Vostrikova VR, Shevel DS, Saidarova AI, Bekbuzarov IM, et al. Wound healing in diabetes mellitus. Spec J Pharmacogn Phytochem Biotechnol. 2023;3:31-8. doi:10.51847/Y5Fvcyw12s
72. Sanlier N, Yasan N. COVID-19 and vitamin D. Interdiscip Res Med Sci Spec. 2024;4(1):23-32. doi:10.51847/skW1PmtWeB
73. Ribeiro A, Martins S, Fonseca T. Medicines policy implementation. Interdiscip Res Med Sci Spec. 2024;4(1):42-56. doi:10.51847/0eVBxAI8y0
74. Balaji A, Jei JB, Murugesan K, Muthukumar B. Distal extension edentulous rehabilitation. J Curr Res Oral Surg. 2022;2:16-9. doi:10.51847/OhXCPOyjBp
75. Alhussain BS, Alamri FS, Alshehri FA, Aloraini AA, Alghamdi SM, Alfuhaid NA, et al.

CAD/CAM ceramic endocrowns. J Curr Res Oral Surg. 2022;2:20-6.

76. García-Parra MI, Jiménez-Coello M, Carrillo-Ávila BA, Hernández-Cortázar I, Chávez-Cortez G, Martínez-Aguilar VM. Osteoprotegerin plasma levels in periodontitis. P R Health Sci J. 2022;41(1):5-8.
77. Asif S, Ahmad B, Hamza SA, Taib H, Kassim NK, Zainuddin SLA. Salivary RANKL and OPG in periodontitis. Eur J Dent. 2022;16(1):173-8. doi:10.1055/s-0041-1731930
78. Abduljalil SMA, Hashim NT, Rahman MM, Chaitanya NC, Mukhtar MM, Gismalla BG. RANKL and OPG in smokers with periodontitis. J Contemp Dent Pract. 2023;24(10):771-8. doi:10.5005/jp-journals-10024-3580
79. Oran IB, Azer OA. Turkey's role in international development. Ann Organ Cult Leadersh Extern Engagem J. 2023;4:1-8. doi:10.51847/oNOPb4T9g1
80. Ncube M, Sibanda M, Matenda FR. AI and pandemic in BRICS nations. Ann Organ Cult Leadersh Extern Engagem J. 2023;4:17-24. doi:10.51847/lrMvYTE3OF
81. Lee S, Kim J, Byun G. Political skill and leadership outcomes. Ann Organ Cult Leadersh Extern Engagem J. 2023;4:45-53. doi:10.51847/vAKE892Paf
82. Razhaeva MU, Khuchieva LA, Musaev SA, Rustamov AK, Bicherkaeva KS, Usmanova KS. Environmental impact of HCH isomer. Asian J Curr Res Clin Cancer. 2022;2(2):1-5. doi:10.51847/Rtj57FuF6z