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**Review Article**

# Effect of periodontal disease on children with diabetes and their quality of life

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## ABSTRACT

Diabetes mellitus (DM) is a life-altering and comparatively prevalent chronic condition affecting children. The root cause of the condition is linked to the pancreatic islets of Langerhans, which produce the hormone insulin and are destroyed by an autoimmune inflammation. The illness then shows up clinically as a result. In the context of the combination of hereditary and external variables, the condition emerges in people with polygenic genetic susceptibility. Concerning a potential connection involving DM and periodontitis, many investigations have been released. Conflicting results, though, have been recorded. Certain authors believe that individuals with DM have significantly higher levels of gingival inflammation and marginal periodontitis than non-DM controls, despite the fact that this association has not been discovered in another research. Dental plaque microbes are the primary cause of periodontal pathologies, in which the tissues around the teeth become infected. Periodontitis manifests in DM patients earlier than it does in the general demographic of healthy individuals. Periodontal deterioration typically shows up in children with DM throughout puberty, while it can happen sooner. Also, in juvenile subjects with type 1 DM, the development of periodontal infection has been linked to poorly managed DM. Elevated access to glucose in both the fluid around the gingival crevice and saliva promotes the growth of caries and periodontal pathology causing microbes and worsens oral inflammation. Owing to inflammation, periodontal disease affects the cellular and humoral immune systems. The generation of insulin and metabolic control are impacted by these modifications in immune responses. Thus, periodontal disease can hinder glycemic control, and impaired metabolic control can further encourage periodontitis; a loop may be generated that worsens both problems. Hence, it is vital to prevent and manage oral inflammatory conditions in order to effectively prevent and control DM complications.

**Keywords:** Juvenile-onset diabetes mellitus, Periodontitis, Periodontal disease, Metabolic syndrome, Quality of life

## INTRODUCTION

Diabetes mellitus (DM) is a life-altering and comparatively prevalent chronic condition affecting children. The root cause of the condition is linked to the pancreatic islets of Langerhans, which produce the hormone insulin and are destroyed by an autoimmune inflammation. The illness then shows up clinically as a result. In the context of the combination of hereditary and external variables, the condition emerges in people with polygenic genetic susceptibility.[1] According to estimates, hereditary and nongenetic variables contribute to the condition's development in almost equal measures. Enteroviral infections (coxsackievirus type B), some dietary factors (such as early introduction to bovine proteins, large intake of dairy items in early life, how long the child was breastfed, the influence of nitrites and nitrates and vitamin D deficiency), and certain variables during pregnancy, and after birth are important ecological predisposing variables.[2-5] A rise in the percentage of risk genetic variants for type 1 DM in the demography is not plausible, so ecological variables, or shifts in how people are exposed to some nongenetic elements, are most likely to account for the sharp rise in the number of type 1 DM cases in the past years. There are no reliable preventative interventions for DM because its pathophysiology is still not completely understood.[6] Only 5-10% of cases are siblings of children or offspring of parents with DM, indicating that the condition is primarily not hereditary.[1] A development of type 1 DM simultaneously was seen in 23-53% of identical twins.[6] When the renal threshold for glucose elimination is surpassed in children, increased urination, and excessive thirst result. This is accompanied by a slow metabolic deterioration and the characteristic manifestations of diabetes. Glycosuria, ketonuria, and hyperglycemia are all observed in the disease. The concentration of glycated hemoglobin (HbA1c), which indicates the blood glucose changes over the previous six weeks, are tracked in order to assess the metabolic control of the disease and therapeutic response. The present standard of care for type 1 DM centers on an enhanced insulin regimen that mimics the release of insulin normally, timely patient self-assessing of certain metabolism indicators, and counseling of the patients and their families.[1]

Concerning a potential connection involving DM and periodontitis, many investigations have been released. Conflicting results, though, have been recorded. Therefore, it has been said in certain papers that individuals with DM had significantly higher levels of gingival inflammation and marginal periodontitis than non-DM controls, despite the fact that this association has not been discovered. Cohen et al. observed in 1970 that the periodontal health of women with and without diabetes over a three-year period.[7] At the outset, it was discovered that the patients with DM had superior dental hygiene than the non-DM controls, but also higher amount of gingival inflammation and a modest increase in attachment loss. Throughout the monitoring period, there was a tendency for the DM group's gingivitis intensity to worsen. Bernick et al discovered that notwithstanding the two groups' equal dental hygiene levels, children with diabetes had a greater incidence of gingivitis than age-matched counterparts.[8] In 1980, Gislen et al. noted that children with DM who displayed low glycemic control generally had more severe gingival inflammation grades than non-DM patients.[9] In 1977, Ringelberg et al said that children with DM had more severe manifestations of gingival inflammation than non-DM controls.[10] Information on the incidence and extent of gingival inflammation and periodontitis in juvenile patients with insulin-dependent DM were described by Cianciola et al When they looked at 263 individuals with DM and 208 non-DM controls, they found that lost attachments and deeper periodontal pockets appeared in 9.8% of the individuals with DM and just 1.7% of the individuals without DM among participants between the ages of 11 and 18.[11] Additionally, they observed that among subjects with DM who were 19 years of age or above, the occurrence of periodontitis spiked to 39%. Nevertheless, several investigations that have been released have completely fallen short of proving a connection between DM and periodontitis. Hove and Stallard did not discover any variations in the severity of marginal periodontal inflammation in the 28 subjects with DM and the 16 non-DM controls, nor was there any connection between the length of the metabolic condition and the severity of periodontal tissue injury in the individuals with DM.[12] When comparing the outcomes of treatment in a control sample of people without preexisting health conditions, Bay et al observed no differences in the levels of remission of gingival inflammation following scaling and root planning in juvenile subjects with DM.[13] In a cohort of 10 to 18-year-old insulin-dependent DM subjects, Barnett et al investigated the occurrence of periodontal inflammation but found no imaging evidence of the condition in any of the cases.[14] In conclusion, whereas multiple epidemiological investigations have demonstrated that diabetes circumstances are linked to a greater occurrence and intensity of gingival inflammation and periodontal tissue destruction, particularly in juvenile subjects, a number of investigations have been unable to demonstrate this link.

## METHODOLOGY

This study is based on a comprehensive literature search conducted on 21 November 2022, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the information about effect of periodontal disease on children with diabetes and their quality of life. There were no restrictions on date, language, participant age, or type of publication.

## DISCUSSION

Numerous research conducted in different parts of the world have looked into the connection between DM as a fundamental condition, and various elements of dental health. While some relationships have been proven, others are still up for debate, and the complex pathogenesis of the majority of oral illnesses makes the conclusions of published research frequently contentious. It is now generally acknowledged that dental plaque microbes are the primary cause of periodontal pathologies, in which the tissues around the teeth become infected. Dental plaque that has turned calcified into calculus promotes plaque buildup and causes gingival irritation. Only the gingiva is impacted in the initial phases. In the absence of therapy, the infection persists for a while and eventually affects all supporting structures of the periodontium. The teeth eventually become mobile and are lost as a result of osteoclastic activity.[15] The bulk of research have found that type 1 DM patients had a much greater occurrence of chronic gingival inflammation than the normal community, and that this incidence rises with aging. The average scores of the gingivitis index on a scale of 1 to 3 was 1.54±0.5 in a group of juvenile subjects with DM between five to nine years of age while it was 1.14±0.5 in the comparison group. The average gingivitis index was 1.98±0.6 versus 1.17±0.5 in a sample of juvenile subjects with DM between the ages of ten to 14 years, correspondingly.[16] There were no variations in the plaque index values or the microbial plaque structure among the type 1 subjects with DM and healthy subjects in a Switzerland based clinical trial on investigational gingival inflammation evoked by forgoing dental hygiene for three weeks, but the subjects with DM reacted to plaque discomfort by a sooner evolved and more serious gingivitis, that conformed to a considerably greater amount of certain inflammatory biomarkers in gingival crevice. In a different study, which involved a sizable cohort of Brazilian children with diabetes with an average age of 13±0.5 years, gingival inflammation and periodontal disease were found in 21% and 6% of the participants, correspondingly.[17] The occurrence of gingival inflammation was 27% higher in a sample of 10- to 15-year-old juvenile subjects with DM from Lithuania than in the control group.[18] Comparable to this, reports indicate that juvenile subjects with DM have a greater rate of microbial plaque deposition and a sooner and bulkier calculus development.[19] Adolescence is when people with DM and their non-DM counterparts begin to vary significantly from one another.[16,18] Periodontitis is referred to as the sixth chronic consequence of DM, despite the fact that it is not one of the clinical presentations of any kind of DM. It has been established that those with DM have a roughly three-fold increased chance of developing periodontal inflammation. As a result, DM is thought to be a risk factor for periodontal inflammation.[20] Periodontitis manifests in DM patients earlier than it does in the general demographic of healthy individuals.[21,22] Periodontal deterioration typically shows up in children with DM throughout puberty while it can happen sooner.[19,23,24] Also, in juvenile subjects with type 1 DM, the development of periodontal infection has been linked to uncontrolled diabetes (increased HbA1c levels).[17,20] Research has linked the length of DM to the intensity of periodontal inflammation.[17,21] In contrast hand, it has been established that periodontal inflammation has a deleterious impact on serum glucose concentrations.[20] Even though some initial research did not validate this theory, more current research has proven the notion that treating periodontal inflammation leads to better glycemic control of DM.[25-27] Recently conducted trials have shown that effective periodontal disease treatment can reduce HbA1c levels by 0.4% however this effect was mostly seen in people with type 2 DM.[28] DM-related microangiopathy, altered oral microbiota, problems with collagen physiology, a weakened immune system and decreased susceptibility to infections are likely to be the causes of the onset and development of periodontal inflammation in DM individuals.[23] Hypoglycemia and hyperglycemia have been shown to directly harm periodontal tissues in in vitro studies. Another unfavorable consequence of high blood glucose levels is that it causes immune system to generate inflammatory cytokines.[29] Investigations that discuss the molecular underpinnings of periodontitis in DM have recently appeared.

### *Impaired immune response in periodontitis and DM*

DM-induced high blood sugar concentrations can affect the immune system in a variety of ways. Firstly, it raises the level of glucose in both the fluid around the gingival crevice and saliva. This higher access to glucose in the periodontal tissues and teeth promotes the growth of caries and periodontal pathology causing microbes and worsens oral inflammation. It has been demonstrated that unmanaged DM leads to increased amounts of inflammatory cytokines in their gingival crevicular fluid as compared to non-DM or well-controlled cases, which causes considerable periodontal damage in response to an identical microbial assault.[30-32] DM-induced high blood sugar can also induce microangiopathy. Vascular endothelial cells utilize more glucose than normal, producing additional glycoproteins on their exterior as the basement membrane becomes denser and less elastic. Vascular walls swell and weaken, allowing for easy bleeding and protein leakage. The advancement of periodontal disease is facilitated by these vasculopathic changes in the periodontal tissues, which reduce polymorphonuclear cellular activities like chemotactic response, adhesion, phagocytic function and migration, oxygen use, and antigens removal. Advanced glycation end-product production is also accelerated by high blood sugar. Nonenzyme-linked glycation and oxidation are brought on by excessive aldose sugar access to proteins (like collagenic proteins) or lipids. These glycated metabolites can produce complicated compounds that decrease collagen resorption and raise concentrations of inflammatory cytokines that cause connective tissue breakdown. Both mineralized bone and nonmineralized connective tissue degrade more quickly as

a result of alterations in collagen breakdown.[31,33] The generation of cytokines and matrix metalloproteinases, such as collagenases and other connective tissue-lysing enzymes, is stimulated by the contact of advanced glycosylation end-products with target cells, like macrophages, through cellular polypeptide receptors.[30] Interleukin-1$\beta$, tumor necrosis factor-$\alpha$, and prostaglandin E2 are only a few examples of the inflammatory mediators that display monocytic overexpression in DM.[34,35] In cases with DM, this inflammatory activity is exacerbated, which can hinder healing process and aggravate injury to tissues. The chemotactic characteristics of advanced glycosylation end-products for human monocytes that develop into chronic inflammatory macrophages can thus exacerbate this proinflammatory response.[31] Periodontitis and delayed wound recovery both advance as a result of the breakdown of recently generated collagen in connective tissues and changes in the immunological response. In contrast hand, periodontal disease, a connective tissue pathology, can produce inflammation that leads to insulin resistance. Monocytic and macrophagic production of tumor necrosis factor-$\alpha$ in response to lipopolysaccharides from periopathogenic Gram-negative bacteria. This cytokine has the potential to disrupt lipid metabolism, lessen cellular glucose absorption, and promote insulin resistance. Due to its high blood flow, an inflamed periodontium may operate as a gateway for bacterial byproducts and locally generated inflammatory mediators into the bloodstream.[36] Owing to inflammation, periodontal disease affects the cellular and humoral immune systems. Activities of polymorphonuclear cells and macrophages are impacted; cytokines crucial for humoral response are also generated. The generation of insulin and metabolic control are impacted by these modifications in immune responses.[37,38] Thus, periodontal disease can hinder glycemic control, and impaired metabolic control can further encourage periodontitis; a loop may be generated that worsens both problems. Hence, it is vital to prevent and manage oral inflammatory conditions in order to effectively prevent and control DM complications.[39]

## CONCLUSION

According to epidemiological research, both juvenile and adult-onset diabetes are associated with more severe cases of periodontitis. Periodontal disease appears to start in young diabetes people around puberty and progress as they get older. An examination of the scientific literature shows that recognized systemic problems caused by DM tend to manifest at about the same age. The extent of periodontal disease in individuals with DM has been reported to be closely correlated with microangiopathy, aberrant collagen processing, aberrant PMN activity, and altered sulcular bacterial flora. Studying the activity of neutrophils in periodontitis is made possible by the link between aberrant neutrophil activity and the extent of periodontitis in individuals with DM. Future research on the role of sulcular PMN could provide insight into the intricate processes that underlie periodontal disease.

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