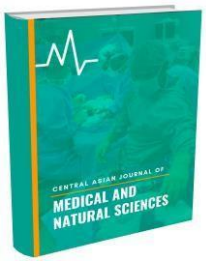

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The Relation of The Diabetes and Periodontal Diseases

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Abstract: Periodontal disease and diabetes mellitus are prevalent chronic conditions with significant public health implications. Emerging research suggests a bidirectional relationship between the two, where diabetes exacerbates periodontal inflammation and vice versa. Clinical and microbiological evidence indicates that individuals with diabetes have increased susceptibility to periodontitis, and periodontal inflammation can impair glycaemic control. Despite the extensive body of literature, inconsistencies in diagnostic criteria, population heterogeneity, and evolving methods of glycaemic assessment complicate direct comparisons and hinder comprehensive understanding. This study aims to evaluate the interrelation between diabetes and periodontal diseases by synthesizing existing clinical, pathological, and microbiological findings to clarify the underlying mechanisms and the potential for therapeutic intervention. The review confirms that hyperglycemia leads to prolonged inflammatory responses, impaired healing, and altered immune function, which contribute to periodontal tissue destruction. Periodontal therapy, particularly non-surgical interventions, has been associated with modest but clinically meaningful improvements in HbA1c levels. Differences in oral microbiota, including elevated levels of P. gingivalis in diabetic individuals, further support a microbiological link. The synthesis emphasizes the reciprocal influence of both diseases, highlighting the role of inflammatory mediators such as TNF-α, IL-1, and MMPs, and identifies the potential of periodontal care as an adjunctive strategy in diabetes management. These findings support integrating periodontal assessment and treatment into comprehensive diabetes care, encouraging interdisciplinary collaboration to mitigate the impact of both conditions and improve patient outcomes.

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1. Introduction

The only dental disorder that has a greater impact on tooth lifetime in the dentition than periodontitis is dental caries. This disorder affects around 5–10% of people, according to [1]. Gingivitis is the more common of the two forms of periodontal disorders, along with periodontitis. Inflammation and the degradation of the tissues surrounding and supporting the tooth, which is brought on by bacterial invasion, are the primary characteristics of periodontal disorders.

An inflammatory disease with many underlying causes, chronic periodontitis is characterised by the slow degradation of the tissues supporting teeth. Dysbiotic biofilms in tooth plaque are often linked to this disorder [2] According to [3] , this kind of periodontitis is mostly a Gram-negative infection that causes significant inflammation and permits the spread of bacteria and their byproducts, such as lipopolysaccharides, throughout the vascular system of the body.

Comprehensive investigations demonstrate a strong association between periodontitis and diabetes. Due to a heightened immune response to the bacteria, this

kind of periodontal disease is characterised by its correlation with both microbial causes and the host's response. Cytokines and other inflammatory markers are released as a result, contributing to the degradation of our own tissues. As a non-communicable disease, periodontitis has several risk factors and determinants with a number of other illnesses, including heart disease, excessive blood pressure, and cancer [4].

Hyperglycemia, the main symptom of diabetes mellitus, is a set of disorders that affect how proteins, fats, and carbohydrates are metabolised. It is known to be genetically and phenotypically heterogeneous. These conditions are characterised by either decreased insulin sensitivity in the liver and muscles, which leads to an incorrect release of insulin, or altered activity of pancreatic β-cells [5]. Insulin resistance and insufficient reserve insulin production are the two main causes of type 2 diabetes. On the other hand, type 1 diabetes often results from the immune system destroying pancreatic β-cells, which completely stops the generation of insulin. Type 2 diabetes is frequently the result of increased adipocyte release of free fatty acids, which is brought on by adiposity and inhibits glucose uptake, glycogen synthesis, and glycolysis. Many obese people tend to increase their insulin output to combat their insulin resistance, but an increase in β-cell apoptosis decreases the ability to produce insulin in obese adults [5].

The degree of hyperglycemia and periodontitis are significantly correlated. The exact processes that connect these two disorders are unclear, but they include elements of neutrophil activity, cytokine dynamics, and immune function. There is mounting evidence that diabetes and periodontitis are correlated in both directions, with diabetes raising the risk of developing periodontitis and periodontal inflammation influencing glycaemic control. According to [6], people with diabetes who have severe periodontitis have twice the rate of macroalbuminuria and a threefold increased risk of end-stage renal disease.

2. Materials and Methods

This study is based on an extensive review of the scientific literature concerning the relationship between diabetes mellitus and periodontal diseases. Over 200 English-language studies published within the last five decades were analyzed to explore the clinical and biological mechanisms linking these two conditions. The authors critically examined variations in diagnostic criteria, glycaemic control assessment, and methods of evaluating diabetic complications, which often make comparisons across studies difficult. Epidemiological data, microbial profiles, inflammatory markers, and glycaemic response to periodontal treatment were reviewed to understand the bidirectional influence between diabetes and periodontitis.

The review incorporated findings from experimental, clinical, and epidemiological studies that investigated periodontal disease prevalence in diabetic populations, particularly individuals with type 1 and type 2 diabetes. The role of hyperglycemia in exacerbating periodontal tissue degradation, immune response dysfunction, and delayed wound healing was examined. Additionally, studies on the impact of periodontal therapy on glycaemic control, including meta-analyses and randomized controlled trials, were considered to evaluate whether periodontal treatment contributes to better diabetes management. The review also included microbial studies assessing oral microbiota differences in diabetic and non-diabetic individuals to highlight potential pathogenic interactions.

3. Results and Discussion

Periodontal disease

A persistent bacterial infection that damages the gums and the tooth's supporting bone is called periodontal disease (PD). The presence of anaerobic Gram-negative bacteria in the plaque that sticks to the teeth causes this syndrome [7]. These bacteria have the capacity to cause a localised inflammatory response over time, which might develop into a chronic issue. Alveolar bone is destroyed and tissue connection to the teeth is lost as a consequence of gum inflammation. Components of the microbial plaque

that might stimulate the infiltration of inflammatory cells, including lymphocytes, macrophages, and polymorphonuclear leukocytes (PMNs), are what propel this process [8].

LPS, a key component of microbial activity, causes macrophages to produce a wide variety and substantial amount of pro-inflammatory chemicals. These mostly consist of prostaglandins, especially PGe2, as well as other enzymes and cytokines including TNF-α and IL-1. Furthermore, it is known that bacterial toxins may activate T cells, which results in the creation of IL-1 and lymphotoxin (LT), a chemical that is similar to TNF-α. According to [8], these cytokines work in tandem with metalloproteinases (MMPs), which use collagenolytic enzymes as precursors, to degrade periodontal tissues because of their strong pro-inflammatory and pro-catabolic properties. Interstitial collagenase levels rise as a consequence of reactive oxygen species stimulating collagenolytic enzymes in the inflamed gingival tissue [9].

Diabetes Mellitus

According to [10], a variety of metabolic diseases are included in diabetes mellitus. Diabetes must be understood as a major metabolic disorder of the carbohydrates, lipids, and proteins that is associated with high blood glucose levels because of either insufficient insulin action or an overabundance of insulin response. Weight loss, increased thirst, frequent urination, hazy eyesight, and in some cases, excessive appetite are common signs of these illnesses if left untreated. Acute hyperglycemia may occur in individuals with nonketotic hyperosmolar syndrome and ketoacidosis, in addition to the serious long-term microvascular consequences linked to chronic hyperglycemia, such as peripheral and autonomic neuropathy, nephropathy, and retinopathy [11].

There are other types of diabetes, such as Type 1, Type 2, gestational diabetes, and others. According to the American Diabetes Association, only 5–10% of people with diabetes in 2021 had type 1 diabetes, which is an autoimmune reaction that attacks the pancreatic α-cells, completely stopping the generation of insulin. Although teens account for the bulk of Type 1 diabetes occurrences, adults account for a lower percentage [12].

Type 2 diabetes, which makes up 90–95% of all cases, is characterised by either low insulin production or insulin resistance, with one of these factors perhaps predominating. Type 2 diabetes is a complex disease that is impacted by a number of risk factors, such as age, obesity, bad lifestyle choices, and prior gestational diabetes. Although it mostly affects the elderly, children and teenagers may also be affected. According to [13], the diagnosis of gestational diabetes mellitus occurs when hyperglycemia is first identified during pregnancy.

Exocrine pancreatic diseases (like pancreatitis and cystic fibrosis), monogenic diabetes syndromes (like neonatal diabetes and maturity-onset diabetes of the young), and drug or chemical-induced diabetes (like post-organ transplantation, glucocorticoid use, and HIV/AIDS treatment) are among the many conditions with different aetiologies that cause hyperglycemia [12]. A condition known as prediabetes is characterised by slightly raised blood glucose levels that do not meet the diagnostic criteria for diabetes but indicate a higher risk of developing diabetes and cardiovascular disease (CVD) in the future. It is often associated with hypertension, dyslipidaemia, and obesity, especially visceral or abdominal fat [12].

Relationship between the oral microbiota and diabetes

Few research have examined the connections between oral microbiota and diabetes, despite the fact that many have examined the inflammatory mechanisms that link periodontitis and diabetes. According to one research, the levels of a number of periodontal infections, such as A. actinomycetemcomitans, Campylobacter rectus, Capnocytophaga spp., E. corrodens, F. nucleatum, and P. intermedia, were similar in diabetes and nondiabetic individuals. However, compared to their non-diabetic counterparts, a higher proportion of diabetics in this study had P. gingivalis [14].

P. gingivalis and P. intermedia seemed to be more common in periodontitis cases among young Japanese patients with type 1 diabetes mellitus than in healthy people[15]. Although the biological ramifications are still to be determined, the results show that this intersubject variability is reflected in the differences in subgingival biofilm microbial compositions between diabetes and non-diabetic persons. Furthermore, diabetes's impact on the local environment of the periodontal pocket may exacerbate circumstances that encourage the development of certain bacterial species.

Researchers have also looked at the connection between obesity and the mouth cavity's bacterial flora. 98.4% of the overweight participants had the periodontal pathogen Selenomonas noxia, with a relative abundance exceeding 1.05% of the total bacterial population, according to a study that included 313 women with body mass indices ranging from 27 to 32 kg/m² (compared to 232 healthy control subjects) [16]. The fascinating topic of whether oral bacteria may be contributing to the pathophysiology linked with obesity was raised when S. noxia was detected at levels higher than 1.05%. This presence showed a sensitivity of 98.0% and a specificity of 80.0% in predicting obesity. According to [17], the idea of "infectobesity" implies that gut bacteria may impact host metabolism by influencing insulin resistance and inflammation. This suggests that the gut microbiota of an obese person is more efficient at extracting energy per unit of food consumed than that of a lean person.

Animal studies have shown that modifying the gut microbiota—by means of prebiotics, certain diets, or natural antibiotics—may affect insulin resistance associated with diabetes and sensations of fullness. Nevertheless, it is still unknown how oral periodontal bacteria mediate these effects [18].

Diabetes and pre-diabetes in relation to periodontitis

According to research, "Individuals with diabetes have a 2- to 4-fold greater risk for developing periodontitis than do non-diabetic individuals, and similar to other complications related to diabetes, this risk progresses with the duration of the disease and the level of glycaemic control attained by the patient" [19] cemia raises the risk of periodontitis in diabetics by causing a number of cellular alterations. According to [9], these alterations include extended inflammatory responses, delayed wound healing, microvascular alterations, weakened defenses against periodontal infections, and impaired new bone production and repair. Patients with type 1 diabetes had a significant decrease in their insulin requirements after receiving periodontal care [20]. Accordingly, it has been suggested that diabetic individuals with periodontitis can find it difficult to keep their blood sugar levels under control, which might result in more difficulties [19],[21]. Studies have shown that people with diabetes with severe periodontitis are more likely to die than those with little or no periodontitis[22]. This implies that diabetes people may be more susceptible to periodontitis [20],[22]. The results of the little research on the relationship between pre-diabetes and periodontitis paint a contradictory picture. Some research has identified a positive link between deep periodontal pockets and alveolar bone loss, whereas other studies have found no correlation at all [20].

Diabetes and periodontitis: pathologic mechanisms

In the context of periodontitis, a chronic inflammatory disease marked by necrosis and the uncontrolled release of mediators primarily triggered by dental plaque within periodontal tissues, chemokines, cytokines, and T cell regulatory cytokines suggest that IL-12 and IL-18 are important mediators studied. The following mediators have also been studied: TNF-α, RANKL, MMP-8, MMP-9, IL-1β, IL-6, and PGE2. Additionally, [23] point out that MMP-13 has also been researched.

Nowadays, it is clear that extremely individual-specific, intricate, and varied cytokine networks play a role in the pathophysiology of periodontitis. Individual differences in the kind of inflammatory response are caused by the interaction of genetic, environmental, and epigenetic variables. Accordingly, patterns and rates of disease development will be impacted by a widespread inflammatory response in the

periodontal tissues [24], [25] found that among individuals with insulin-dependent diabetic mellitus (IDDM), the prevalence of periodontitis was associated with a higher deterioration in glycaemic control over a two-year period. individuals with periodontitis were more likely to develop ketoacidosis, retinopathy, and neuropathy than diabetic individuals without the condition. According to [26], those with neurological impairments also had more severe instances of gingivitis than people without such impairments.

The Impact of Periodontal Therapy on the Management of Diabetes

The impact of treating periodontitis on diabetics' capacity to control their blood sugar levels has been studied. Diabetes patients with periodontitis who got therapy were compared to those who did not get treatment or whose intervention was delayed in a number of randomised controlled studies. The findings of these research have been extensively reviewed in up to seven systematic reviews and meta-analyses to far. According to the data from these evaluations, periodontitis therapy of any kind has been consistently associated with a 0.4% reduction [27], [28].

These investigations included a Cochrane review, which showed that nonsurgical periodontal therapy reduced HbA1c by around 0.4%. Although a 1% drop in HbA1c may not seem like much, it has important clinical ramifications since it is linked to a quantifiable reduction in the risk of diabetes-related complications[27]. Furthermore, treating periodontal disease is a simple medical procedure free of the negative effects sometimes associated with other diabetic drugs.

It is acknowledged that not all clinical research evaluating the impact of periodontal therapy on glycaemic management have produced identical findings. The failure of periodontal care to improve glycaemic control in a recent large multicenter study with more than 500 patients serves as a noteworthy example [29]. There are two primary reasons why this specific research has drawn criticism: it included individuals who already had a favourable HbA1c (glycaemic control) level, which was out of proportion to the intervention's possible advantages).

Diabetes and periodontal therapy

Managing periodontitis may improve glycaemic control in diabetics, according to several clinical investigations [30], [31]. According to a recent meta-analysis, periodontal therapy may result in significant drops in HbA1c levels when it comes to the glycaemic management of diabetic patients [28]. However, the meta-analysis's authors stressed that care should be used when interpreting these findings since several of the included studies had inadequate and inconsistent methodology.

4. Conclusion

In conclusion, there may be a negative cycle generated by the interaction between diabetes and periodontitis, yet this loop may be halted by treating periodontitis. To assess how periodontal care improves diabetes patients' glucose management, more intervention studies must be done out.

REFERENCES

[1] S. Wild, G. Roglic, A. Green, R. Sicree, and H. King, "Global prevalence of diabetes: estimates for the year 2000 and projections for 2030," *Diabetes Care*, vol. 27, no. 5, pp. 1047–1053, 2004.

[2] P. N. Papapanou, V. Baelum, W.-M. Luan, P. N. Madianos, X. Chen, O. Fejerskov, et al., "Subgingival Microbiota in Adult Chinese: Prevalence and Relation to Periodontal Disease Progression," *J. Periodontol.*, vol. 68, no. 7, pp. 651–666, Jul. 1997.

[3] B. L. Mealey and R. Klokkevold Perry, *Periodontal Medicine: Carranza's Clinical Periodontology*, New Delhi: Elsevier, 2004.

[4] M. B. Agholme, G. Dahllöf, and T. Modéer, "Changes of periodontal status in patients with Down syndrome during a 7-year period," *Eur. J. Oral Sci.*, vol. 107, no. 2, pp. 82–81, 1999.

[5] B. L. Mealey and T. W. Oates, "Diabetes mellitus and periodontal diseases," *J. Periodontol.*, vol. 77, no. 8, pp. 1289–1303, 2006.
[6] P. M. Preshaw, A. L. Alba, D. Herrera, S. Jepsen, A. Konstantinidis, K. Makrilakis, and R. Taylor, "Periodontitis and diabetes: a two-way relationship," *Diabetologia*, vol. 55, no. 1, pp. 21–31, Jan. 2012.
[7] C. A. Negrato and O. Tarzia, "Buccal alterations in diabetes mellitus," *Diabetol. Metab. Syndr.*, vol. 2, p. 3, 2010.
[8] T. Sorsa, T. Ingman, K. Suomalainen, M. Haapasalo, Y. T. Konttinen, O. Lindy, et al., "Identification of proteases from periodontopathogenic bacteria as activators of latent human neutrophils and fibroblast type interstitial collagenases," *Infect. Immun.*, vol. 60, pp. 4491–4495, 1992.
[9] W. Lee, S. Aitken, J. Sodek, and C. A. McCulloch, "Evidence of a direct relationship between neutrophil collagenase activity and periodontal tissue destruction in vivo: role of active enzyme in human periodontitis," *J. Periodontal Res.*, vol. 30, pp. 23–33, 1995.
[10] American Diabetes Association, "Diagnosis and Classification of Diabetes Mellitus," *Diabetes Care*, vol. 33, Suppl. 1, pp. S62–S69, 2010.
[11] American Diabetes Association, "Diagnosis and classification of diabetes mellitus," *Diabetes Care*, vol. 37, Suppl. 1, pp. S81–S90, 2014.
[12] American Diabetes Association, "Classification and diagnosis of diabetes: Standards of Medical Care in Diabetes," *Diabetes Care*, vol. 44, Suppl. 1, pp. S15–S33, 2021.
[13] P. J. Turnbaugh, R. E. Ley, M. A. Mahowald, V. Magrini, E. R. Mardis, and J. I. Gordon, "An obesity-associated gut microbiome with increased capacity for energy harvest," *Nature*, vol. 444, pp. 1027–1031, 2006.
[14] H. Thorstensson, G. Dahlen, and A. Hugoson, "Some suspected periodontopathogens and serum antibody response in adult long-duration insulin-dependent diabetics," *J. Clin. Periodontol.*, vol. 22, pp. 449–458, 1995.
[15] K. Takahashi, F. Nishimura, M. Kurihara, et al., "Subgingival microflora and antibody responses against periodontal bacteria of young Japanese patients with type 1 diabetes mellitus," *J. Int. Acad. Periodontol.*, vol. 3, pp. 104–111, 2001.
[16] J. M. Goodson, D. Groppo, S. Halem, and E. Carpino, "Is obesity an oral bacterial disease?" *J. Dent. Res.*, vol. 88, pp. 519–523, 2009.
[17] R. E. Ley, P. J. Turnbaugh, S. Klein, and J. I. Gordon, "Microbial ecology: human gut microbes associated with obesity," *Nature*, vol. 444, pp. 1022–1023, 2006.
[18] A. Vrieze, F. Holleman, E. G. Zoetendal, W. M. de Vos, J. B. L. Hoekstra, and M. Nieuwdorp, "The environment within: how gut microbiota may influence metabolism and body composition," *Diabetologia*, vol. 53, pp. 606–613, 2010.
[19] H. L. Collin, M. Uusitupa, L. Niskanen, V. Kontturi-Narhi, H. Markkanen, A. M. Koivisto, et al., "Periodontal findings in elderly patients with non-insulin dependent diabetes mellitus," *J. Periodontol.*, vol. 69, no. 9, pp. 962–966, 1998.
[20] T. Saito, M. Murakami, Y. Shimazaki, S. Matsumoto, and Y. Yamashita, "The extent of alveolar bone loss is associated with impaired glucose tolerance in Japanese men," *J. Periodontol.*, vol. 77, no. 3, pp. 392–397, 2006.
[21] K. M. Karjalainen, M. L. Knuuttila, and K. J. von Dickhoff, "Association of the severity of periodontal disease with organ complications in type 1 diabetic patients," *J. Periodontol.*, vol. 65, no. 11, pp. 1067–1072, 1994.
[22] A. Saremi, R. G. Nelson, M. Tulloch-Reid, R. L. Hanson, M. L. Sievers, G. W. Taylor, et al., "Periodontal Disease and Mortality in Type 2 Diabetes," *Diabetes Care*, vol. 28, no. 1, pp. 27–32, 2005.
[23] P. M. Preshaw and J. J. Taylor, "How has research into cytokine interactions and their role in driving immune responses impacted our understanding of periodontitis?" *J. Clin. Periodontol.*, vol. 38, pp. 60–84, 2011.
[24] D. F. Kinane, P. M. Preshaw, and B. G. Loos, "Host-response: understanding the cellular and molecular mechanisms of host-microbial interactions–consensus of the Seventh European Workshop on Periodontology," *J. Clin. Periodontol.*, vol. 38, pp. 44–48, 2011.
[25] G. W. Taylor, B. A. Burt, M. P. Becker, R. J. Genco, M. Shlossman, W. C. Knowler, et al., "Severe periodontitis and risk for poor glycemic control in patients with non-insulin-dependent diabetes mellitus," *J. Periodontol.*, vol. 67, pp. 1085–1093, 1996.
[26] I. Rosenthal, H. Abrams, and R. Kopczyk, "The relationship of inflammatory periodontal disease to diabetic status in insulin-dependent diabetes mellitus patients," *J. Clin. Periodontol.*, vol. 15, no. 7, pp. 425–429, 1998.
[27] W. J. Teeuw, V. E. A. Gerdes, and B. G. Loos, "Effect of periodontal treatment on glycaemic control of diabetic patients: a systematic review and meta-analysis," *Diabetes Care*, vol. 33, pp. 421–427, 2010.

[28] L. Darré, J.-N. Vergnes, P. Gourdy, and M. Sixou, "Efficacy of periodontal treatment on glycaemic control in diabetic patients: a meta-analysis of interventional studies," *Diabetes Metab.*, vol. 34, no. 5, pp. 497–506, 2008.
[29] T. C. Simpson, I. Needleman, S. H. Wild, D. Moles, and E. J. Mills, "Treatment of periodontal disease for glycaemic control in people with diabetes," *Cochrane Database Syst. Rev.*, CD004714, 2010.
[30] D. C. Rodrigues Jr., M. Taba Jr., A. B. Novaes, S. L. Souza, and M. F. Grisi, "Effect of non-surgical periodontal therapy on glycemic control in patients with type 2 diabetes mellitus," *J. Periodontol.*, vol. 74, no. 9, pp. 1361–1367, 2003.
[31] M. Kıran, N. Arpak, E. Ünsal, and M. F. Erdoğan, "The effect of improved periodontal health on metabolic control in type 2 diabetes mellitus," *J. Clin. Periodontol.*, vol. 32, no. 3, pp. 266–272, 2005.