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Open Access Journal of Dental Sciences
ISSN: 2573-8771

Would Better Control of Diabetes Improve Oral/Periodontal (Gum) Health? A Short Narrative Review of how Dental Practitioners can contribute

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Review Article
Volume 6 Issue 1
Received Date: February 16, 2021
Published Date: March 10, 2021
DOI: 10.23880/oajds-16000291

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Abstract

The World Health Organisation has listed diabetes to be the seventh leading cause of death, with more than 422 million adults having the condition. It is therefore paramount to understand its pathophysiology, examine its systemic effect and more specifically, the impact on periodontal health. The aim of this short narrative review is to assess how patient's control can play a role in maintaining a better periodontal health, and how a General Dental Practitioner can positively contribute. The overall consensus illustrates a relationship that poorly controlled diabetes could worsen the periodontal tissue leading to destruction and deterioration of gum health.

Keywords: Diabetes; Periodontal health; Periodontitis; Oral hygiene; Gum; HbA1c

Abbreviations: WHO: World Health Organisation; PD: Periodontal Disease; AGE: Advanced Glycation End-Products; CRP: C-Reactive Protein; DM: Diabetes Mellitus.

Introduction

It has been estimated that the number of people living with Diabetes Mellitus worldwide is 422 million and it is the seventh leading cause of death as reported by the World Health Organisation (WHO). Diabetes Mellitus (DM) is a condition which collates metabolic disorders characterised by elevated levels of glucose in the blood, also known as hyperglycaemia. This increase of glucose is a result of malfunction of pancreatic β cells or resistance to the normal function of insulin in the human body [1]. Long untreated hyperglycaemia can cause systemic complications leading to long term deterioration of many organs in the body including the eyes, heart, kidneys, nerves and vascular system [1,2].

The different pathologies of diabetes give rise to three main classifications; Type I (Insulin dependent), Type II (Non-Insulin dependent) and Gestational diabetes (a diabetic state acquired during pregnancy) [1].

Dentists appreciate the long-term complications of diabetes in relation to the oral cavity, including dry mouth, candida infections, delayed wound healing and a secondary risk factor for Periodontal Disease (PD) [2]. A review by Leo in 1993 listed periodontitis as the sixth most significant complication of diabetes [3]. Periodontitis is defined as inflammation and destruction of the underlying supporting tissue of the teeth [4], characterised by periodontal pocket

formation, loss of connective tissue attachment and alveolar bone resorption [5] in response to plaque/biofilm formation on tooth surface. More evidence from both cross-sectional and longitudinal studies has proven the association between DM (both Type I and II) and PD [6-9]. Taylor, et al. [10] demonstrated that the presence of Periodontal Disease in diabetic patients has a role in their glycaemic control [11], and its treatment can improve the patient's metabolic control, therefore consolidating a two-way relationship between the two conditions [9,11-13]. Recent evidence base has determined that his two-way relationship is linked by several mechanisms, where the importance of the host response in the periodontal pathology is firmly confirmed [2,13].

Risk factors for Periodontal Disease, What are the implications?

Diabetes has indisputably been confirmed as one of the factors that increase the signs of periodontitis [2,5,7,9,14-16], with some studies suggesting up to a three-fold [1,2] increase in risk. Other established risk factors for PD include cigarette smoking, stress and poor diet. Evidence illustrates that hyperglycaemia is biologically linked to periodontal tissue destruction by disturbing and altering the immuno-inflammatory response. These alterations are displayed by the production of local mediators such as cytokines (e.g interleukins) [2,17] which highlights the clinical signs of the disease, particularly in Type II diabetes [18]. In a normal body homeostasis in relation to hyperglycaemia; glycosylation of proteins occurs frequently which result in the formation of advanced glycation end-products (AGE). The rate of AGE formation is markedly elevated in people with diabetes [2]. RAGE is a receptor for AGE, and the binding of these receptors advance the increase in the levels of inflammatory mediators (e.g IL-1B, TNF-α and IL-6) which can all promote the tissue change within the periodontium [17] and further periodontal tissue destruction [1,2,19]. Glycohemoglobin is formed continually in the human body, by the binding of haemoglobin and glucose [1]. This forms HbA1c. The level of circulating HbA1c in the body provides an estimate of an individual's two-three months glycaemic control [8]. Thus, high levels reflect the increase of this inflammatory marker, leading to diabetic complications [1].

Another important source of cytokines is adipose tissue. Adipose tissue is abundant in obesity which is one of the manifestations of Type II diabetes. It is evident now, that adipose tissues play a role in the active endocrine secretion of cytokines, such as IL-6 and TNF-α13, and adipokines (e.g leptin and adiponectin). The levels of leptin are directly proportional to the number of adipose tissues; playing an important part in the inflammatory response, which in return correlates to the increased risk of developing Type II diabetes, and inevitably PD. Adiponectin, however, has an opposite effect to that of leptin; whereby, it is an anti-inflammatory. It is indirectly proportional to the number of adipose tissue present, therefore low numbers are associated with periodontal changes [2,20,21].

Longitudinal studies of the relationship between diabetes and Periodontal Disease started as long as forty years ago, by Cohen, et al. [22]. Recent to that, Seppälä, et al. [7], went to study the effect of Type I diabetes on PD over two years. It looked into subjects from three different departments of Medicine where they investigated a variety of dental examinations focused on the periodontium, comparing poor and well controlled glycaemic patients. Seppälä's subjects were aged from 35-56 years [7]. Of the 50 subjects initially participating, only 22 completed the study. A very similar pattern of subjects leaving the experiment was shown in a cohort study, Cohen, et al. [22]. The reason for this study dropout rate was thought to be due to diabetic patients requiring urgent treatments as oppose to long term regular check-ups; observed by Thorstensson, et al. [23]. The only set back is the small number of participants. This reduces the validity of the study. However, the study did conclude that the incidence of Periodontitis was higher in poorly controlled diabetic individuals to that of controlled. These results were in conformity with previous studies [7]. Kaur, et al. looked into the association between Type I and Type II DM as to whether there is a risk of a deteriorating effect to the periodontal tissue health [15]. The research viewed the homogenous adult population in North Germany. The data was collected to examine if there was an increased prevalence to the extent of periodontal damage in those who had diabetes in comparison to those who are non-diabetics. Results were significant (Table 1) and the association of both diseases to PD were plausible. As this was a cross-sectional designed study, it lacked details that impacted on its conclusion; such as glucose intolerance tests, previous periodontal treatments, and previous glucose tests. More importantly, data on whether previous teeth were extracted due to worsening periodontal state was not noted, as the remaining dentition may not represent the long-term periodontal status. This particular study was distinctive, due to the high number of recruited participants; more than 4,000 individuals. Hideaki Hayashida, et al. [8] went to look further into the matter but in a different perspective. They examined the relationship of periodontitis and the levels of HbA1c in non-diabetic subjects. The study was also cross sectioned to small community-based residents, lacked the number of participants and mainly focused on elderly patients. It was concluded that periodontal characteristics were related to the high levels of HbA1c in non-diabetic individuals [8]. A large number of clinical research studies were undertaken to study the extent of diabetes mellitus as a risk factor for PD.

Authors	Study Design	Aims	Sample Size/ Population	Diabetes Type	Duration	Intervention Diabetes	Outcome Measures/ Results	Conclusions
Seppälä, et al. [7]	Longitudinal study	Evaluate links between Type I diabetes and Periodontal Disease	Finish population 38 diabetics	Type I	2 years	Insulin	-	Poorly Controlled Type I diabetics had more gingivitis, BOP, tooth attachment loss, bone loss and gingival recession than Controlled Type I diabetics.
Kaur, et al. [15]	Population based	Determine whether both Type I and Type II diabetes mellitus are associated with increased prevalence and extent of Periodontal Disease and tooth loss compared with non-diabetic subjects	German population 145 Type I diabetics and 2647 non-diabetics 182 Type II diabetics and 1314 non-diabetics	Type I and II	3.5 years	-	-	There is an association between both Type I diabetics and Type II diabetics with increased severity of periodontitis and tooth loss compared with non-diabetics.
Hideaki Hayashida, et al. [8]	-	Analyse the periodontal status and glycosylated haemoglobin (HbA1c) level in non-diabetic subjects to investigate the relationship between periodontitis and glucose control in non-diabetics	Japanese population 141 Subjects	-	1 year	-	-	Significant relationship between periodontal status and HbA1c levels in non-diabetics.
Khader, et al. [16]	Meta-Analysis of 18 studies	Assess the association between Diabetes Mellitus and Periodontal Diseases by comparing the extent and severity of Periodontal Diseases between diabetics and non-diabetics	18 comparative cross-sectional studies, 3 cohort studies and 2 clinical trials 6 studies in USA, 6 studies in Finland and remainder in Australia, Argentina, England, Sweden, France, Turkey and Mauritius 1835 diabetics and 17410 non-diabetics	Type I and II	-	-	-	Diabetics had significantly higher severity but the same extent of Periodontal Disease than non-diabetics, having a higher severity of gingival disease measured by Gingival Index, Probing Pocket Depths, Clinical Attachment Loss, Bone Loss and BOP score

Janket, et al. [24]	Meta- Analysis of 10 studies	Quantify the effects of periodontal treatment on HbA1c level among diabetic patients	456 patients	Type I and II	2 weeks to 5 years	-	-	Average decrease in HbA1c level: 0.38% for all studies 0.66% when restricted to Type II diabetic patients 0.71% if antibiotics were given to them
Al-Mubarak, et al. [27]	-	Assess the response of diabetics to scaling and root planing treatment and subgingival oral irrigation as adjunctive therapy	USA population 52 diabetic subjects with adult Periodontal Disease	Type I and II	12 weeks	Patients were on the same type and dose of diabetic oral hypoglycemic agents and insulin along with the same controlled diet	PPD was >5 mm but, 8 mm in at least one site in 4 teeth in at least 2 different quadrants Teeth showed no profound mobility or furcation involvement	Scaling, root planing and adjunctive therapy may be of value in establishing a healthy periodontium in diabetics
Commisso, et al. [9]	-	Observe the oral health condition in a diabetic Type II population in connection with glycaemic control and lifestyle.	Italian population 118 patients	Type II	3 months	Metformin - 65.3% Insulin secretagogue -26% Insulin - 47.5% Glitazoni - 1.7%	-	Poor oral health care was observed in the diabetic populaion
Fitzsimmons, et al [28]	-	To determine the independent and combined associations of Interleukin 1B (Il 1B) and C-Reactive Protein (CRP) in gingival crevicular fluid in periodontal patients	Australian population 939 people (430 with diagnosed Periodontal Disease and 509 controls) National Survey of Adult Oral (NSAOH)	-	3 years	-	-	Greater odds of having periodontitis was associated with higher amounts of IL-1b and CRP

Santos, et al. [29]	Literature review	Evaluate the impact of periodontitis on the diabetes – related inflammatory status	-	Type I and II	-	-	-	Inflammatory response in periodontitis can contribute to the overall low-grade inflammation that occurs in diabetes; adversely affects glycaemic control.
Taylor, et al. [10]	Longitudinal study	Testing the hypothesis that severe periodontitis in persons with non-insulin-dependent diabetes mellitus incresass the risk of poor glycaemic control.	Indian population 105	Type II	6 years	-	Clinical loss of periodontal attachment using Ramfjord index teeth. OPG rad to assess bone levels using modified Schei technique.	Results strongly suggest that severe periodontitis at baseline increase risk of poorer glycaemic control.
Cohen, et al. [22]	Longitudinal observations	The quantitative difference in the progression of Periodontal Disease in diabetic and non-diabetic females. The longitudinal significance of local factors which may initiate, contribute to, and/or modify the progress of Periodontal Disease in the diabetic and non-diabetic female.	USA population 39 females (21 diabetic)	Type I and II	2 years	-	Gingival score Horizontal tooth mobility	The diabetic patients had a significantly higher periodontal score (more gingival involvement and greater loss of attachment) than non-diabetic group at every examination.
Thorstensson, et al. [23]	-	Investigate dental care habits and knowledge of oral health in age and sex matched adults, long and short duration insulin-dependent, diabetics and non-diabetics	Swedish population 266 (86 non-diabetic)	Long and short insulin dependant	2 years	Insulin	-	Diabetes mellitus had several effects on dental health.

Table 1: Comparison table of previous studies, including aims, settings and findings.

A number of meta-analyses were conducted to obtain a valid concensus of the relationship. Khaders, et al. [16] undertook a cross-sectional comparison study, comparing 18 literatures from 1970-2003 of the periodontal statuses in diabetics compared to non-diabetics. The use of a very strict inclusion criteria for the subjects in an attempt to qualify and quantify any significance was undertaken, and it was concluded that DM severely increases the extent of PD [16].

However, a smaller meta-analysis of ten interventional studies [24] assessing the impacts of periodontal treatment on diabetic control, found a non-significant reduction in HbA1c following the provision and intervention of periodontal treatment. It should be noted that this meta-analysis had a more lenient inclusion criteria, allowing for studies with smaller sample sizes. Furthermore, with studies focused on a Type I diabetic majority cohort, this can lead to further criticism of the outcomes. With Type I diabetic patients being more cautious of their glycaemic control of insulin to that of Type II patients [24], their HbA1c would be less likely to be significantly altered, thereby affecting the conclusion of the analysis. Khaders, et al. [16] utilising a stricter inclusion criteria, greater patient cohort (1,835 diabetic and 17,410 non-diabetic participants with ages ranged from 5 to 78 years) with a wider demographic (including patients from America, Argentina, England, Sweden, France, Turkey and Mauritius) improved the reliability and quality of the papers findings.

Evidence of How Dentists Can Improve Glycaemic Control

Periodontal treatment may be approached in either a surgical or non-surgical manner. Treatment aims at reduction in the subgingival oral biofilm in order to achieve homeostasis [25]. This can be achieved via debridement using scalers coupled with intensive oral hygiene instruction and patient motivation. Emerging evidence suggests that periodontal treatment in conjunction with systemic antibiotics may provide significantly improved outcomes for patients with severe periodontitis than non-surgical therapy alone [25,26]. The effect of treating PD on diabetic patients is promising with a positive impact on glycaemic control [2]. Many studies tested this relationship in many perspectives. The meta-analyses of Janket, et al. [24] illustrated that after a periodontal intervention, HbA1c levels reduced by 0.38%. Other researchers went as far as providing systemic antibiotics (e.g doxycycline) to lower the inflammation markers such as C-Reactive Protein (CRP) and other immuno-inflammatory mediators. Those studies were criticised for a potential systemic bias as the antibiotics provided an external measure to accommodate the high levels of the inflammatory markers as oppose to dental intervention alone [24]. This type of arbitration would likely lead to over-estimation of the positive effect of the glycaemic control.

Normal interventions such as scaling and root debridement with subgingival irrigations were studied by Al-Mubarak [27]. The team looked into both types of DM in a randomised controlled trial. Measurements included systemic mediators (TNF-α and IL-1B) and HbA1c. Although the number of participants was small (52 subjects) it was conducted in a single diabetic centre, and showed significant results. It has proved that such interventions can play a positive value into periodontal health in diabetics [27]. However, the study overlooked if the subjects were smokers or not. Smoking has systemic implications which could manipulate the final results [28]. Commiso [9] undertook a study observing oral health and Type II diabetic glycaemic control. The study was small and unconvincing. Of the initial 118 patients that took part, only 9 patients went to be tested. Regardless of their significance in reduction of the HbA1c levels [9]; this is strongly criticised to show any significance due to the low number of participants that undertook the study, as they do not reflect a great spectrum of patient's data. An Australian population was studied by Fitzsimmons, et al. [28]. The research looked at 939 subjects, 430 of whom had periodontitis, and sought to examine the link between local and systemic mediators (IL-1B and CRP) on the periodontal health. Their results proved significant, with a number of mediators also showing a significant association with a history of diabetes. The limitations to this study were their periodontal measurements; being inclusive to 3 sites instead of the normal 6, and their reliance on the subject's declaration of medical and social history without confirming it from patient records [28]. Santos, et al. [29] had also shown that dental intervention could improve periodontal status with Type II diabetics. However, their results were based on a very short intervention time compared to a normal eight-week intervention. Hence a more longitudinal long-term data is required to reflect a true extent of periodontal association and glycaemic control [29].

Conclusion

The management of diabetic patients requires a multidisciplinary approach. This relationship of a better diabetic control and a healthier periodontal state is plausible. Evidence based research has clearly identified this relationship in the past years [1,30], and has more recently focused on the immuno-inflammatory mediators. The reviews that were studied had a mix of results. The overall concensus (Table 1) illustrates a relationship that, poorly controlled diabetes could worsen the periodontal tissue leading to destruction and deterioration of gingival health [31-36].

The limitations of the study are that it is only a narrative review and not a systematic one. Although the article used a few systematic reviews, it seeks to generalise the findings rather than provide exact figures, percentages or flowcharts. The study did not aim to make a connection between the age of the diabetic patient and Periodontal Disease, but between diabetes, Periodontal Disease and oral hygiene. Another limitation is the small sample compared to the large diabetic population. The number of studies considered was, therefore, limited and difficult to generalise given their different

arrangements. The present report cannot predict the length of time diabetes can cause or increase Periodontal Disease. There are no studies showing an increase in periodontal risk with the aging of the diabetic patient. There is no critical age or age limit from which diabetes has been shown to worsen periodontal status, as there are no studies to approximate how many years after the onset of diabetes, the Periodontal Disease becomes detectable and to what extent it correlates with the patient's age. More substantive studies are needed to address all these questions and chart these aspects so the clinician may have treatment protocols and guidelines in place, depending on the age of the disease and the patient.

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