



## IMPACT OF GLYCEMIC LEVELS IN TYPE 2 DIABETES ON PERIODONTITIS

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### ABSTRACT

**Aim:** The aim is to study the effect of glyceemic level in Type 2 diabetes and cardiovascular risk factors on periodontal health. **Materials and Method:** Type 2 diabetic and non diabetic patients in the age group of 30-50 years ( $n = 100$ ) were recruited for the study. Periodontal examination included as follows: Probing depth, clinical attachment level (CAL), gingival recession, and bleeding on probing. Periodontitis was diagnosed based on the CAL levels and diabetes was diagnosed based on glycated hemoglobin (HbA1c) levels. **Conclusion:** HbA1c and lipid levels were statistically significant with the severity of periodontitis Smoking and obesity were also found to be significantly associated with the presence of periodontitis). The study concluded that uncontrolled HbA1c levels and elevated cardiovascular risk factors significantly increase the severity of periodontitis in Type 2 diabetes mellitus.

### INTRODUCTION

Diabetes mellitus (DM) is a chronic, noncommunicable disease and also one of the major global public health issues. It is defined as a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. An elevation of blood glucose level (hyperglycemia) is the primary feature of DM and results from a defect in insulin secretion by pancreatic  $\beta$  cells, a decrease in insulin sensitivity, or a combination of both.<sup>[1]</sup> The most common form of DM is DM type 2 (DM2), which accounts for 85% of all diabetes patients.<sup>[2]</sup>

DM currently is the twelfth leading cause of death in the world. The prevalence of DM has risen dramatically in recent years, resulting in a rapid increase in diabetic patients. Asia, in particular, has the highest prevalence of diabetes in the world. Countries exhibiting the fastest rate in diabetic population growth include India and China, among many other developing countries.<sup>[3]</sup>

Hyperglycemia triggers a wide variety of long-term complications in diabetics such as large vessel diseases, cardiomyopathy, and kidney and eye impairments.<sup>[4]</sup> Periodontal disease is the most prevalent oral complication in patients with DM2. The risk of developing periodontitis may be greater in patients with diabetes who have poor glyceemic control than that in patients with well-controlled diabetes. In the Third National Health and Nutrition Examination Survey, which included thousands of Americans, adults with poorly controlled diabetes had an almost three-fold

increased risk of having periodontitis compared with that in adult subjects without diabetes, while subjects with diabetes and good glyceemic control had no significant increase in risk.<sup>[5]</sup>

When oral hygiene is compromised, oral bacteria may form a plaque biofilm, which is resistant to chemicals and immune cells.<sup>[6,7]</sup> Without mechanical debridement, the plaque biofilm matures and causes gingivitis in a few days. Gingivitis represents chronic but reversible inflammation and can be usually treated by proper plaque control. Gingivitis typically extends to irreversible periodontitis for months or years.<sup>[6,7]</sup> This accumulation of plaque acts as a local irritant in the oral cavity.

The evidence of a direct relationship between periodontal disease and diabetes, gathered from thorough reviews, is strong.<sup>[8]</sup> It appears that diabetics have an increased susceptibility to periodontitis that is related to diabetes control,<sup>[9]</sup> and duration of disease.<sup>[10]</sup> The objective of the present study is to determine the correlation between glyceemic control and periodontitis among 35-45 years aged patients with DM2.

### MATERIAL AND METHODS

#### Source of Data

100 Subjects attending the in the dental clinic ahmedabad, Gujarat, India for their routine checkup, and investigation were screened for DM2 and Periodontitis. Patients who fulfilled the eligibility criteria and signed the informed consent were recruited in the study.

## Inclusion and Exclusion Criteria

### Inclusion criteria

Blood sugar controlled only with oral hypoglycemic agents and no systemic antibiotic administration or periodontal treatment within the last 6 months.

### Exclusion Criteria

Patients with known systemic diseases other than DM2, tobacco, and alcohol users and patients who were suffering from an oral disease that need emergency treatment such as endo-perio lesion, periodontal abscess.

### Study Design

Glycemic status was assessed by the glycelated hemoglobin (HbA1c) and fasting blood sugar level. Participating subjects were interviewed for the demographic data, medical history, duration, and the frequency of a drug and were cross-verified with the hospital records.

The oral examination was conducted in dental clinical wing of the diabetic center. Periodontal pocket depth was assessed by measuring PPD with the UNC-15 probe at six points for each tooth, and the arithmetic mean value of all the teeth was considered. Gingival status was assessed by GI, Loe and Silnes<sup>12</sup> Dental plaque was assessed by PI.<sup>13,14</sup>

In the present study, unpaired *t*-test was applied to find the statistical difference in PPD, GI, and PI with glycemic level (HbA1c). Statistical significant was set at  $P = 0.05$ .

## RESULTS AND DISCUSSION

The finding that DM2 subjects have an increased prevalence and severity of periodontitis in our study is in accordance with other studies.

An abundance of recent evidence has consolidated a bidirectional correlation between diabetes and periodontitis. While diabetes is an independent risk factor for periodontitis, periodontitis as a chronic inflammation has a negative impact on the metabolic control of diabetes. DM and periodontitis are common multigenetic and multifactorial chronic diseases with a higher incidence at increased age. Both of the morbidities negatively affect periodontal health and systemic health, thus affecting the quality of life.<sup>[16-18]</sup>

As the value of HbA1c increases there is increase in the mean PPD value. This is because hyperglycemia impairs overall cell function, as insulin is required for glucose to enter cells to provide a source of energy. Furthermore, hyperglycemia alters collagen metabolism, which predisposes to impaired wound healing. In general, hyperglycemia results in the formation of proteins known as advanced glycation end products (AGEs). AGEs may be associated with a state of enhanced oxidative stress, thereby accelerating tissue injury. Finally, hyperglycemia and the imbalance in lipid metabolism impair neutrophil

and monocytes functioning.<sup>[19]</sup> Significant association between DM2 and periodontal disease was found by using the deep periodontal pockets as the clinical parameter for periodontal disease severity.<sup>16</sup> All of these factors may contribute to DM2 predisposing to the pocket formation.

The PI values were minimum in patients having healthy periodontium but increased gradually with the progress of the periodontal disease except for localized periodontitis. Plaque acts as a local irritant in the oral cavity.

Regarding the influence of diabetes on periodontium, there are two schools of thoughts. One school of thought has reported increased severity of periodontal disease in diabetics not related to increased local irritants. According to them angiopathy, abnormal collagen metabolism, abnormal polymorphonuclear cell function, and altered sulcular microbial flora are found in close association with the severity of periodontitis in diabetic patients. These factors reduce the defensive capacity of tissues and may disturb the tissue response to local irritants. Another school of thought recognizes no relationship between diabetes and periodontal disease and maintains that when two conditions exist together, it is a coincidence rather than a specific cause and effect relationship. According to them, the distribution and severity of local irritants affect the severity of periodontal disease in diabetics. In the present study, effect of blood glucose level is directly related to the plaque which is similar to the results shown by Southerland *et al.* Previous pathological study showed diabetic patients had a larger content of lipid-rich plaque compared with nondiabetic patients. The result of the study (Ricardo Faria-Almeida, Ana Navarro, and Antonio Bascones, 2006) showed a statistically significant relationship between PI and HbA1C ( $P = 0.0001$ ) which was similar to our study.<sup>[20-22]</sup>

Although, the sex interaction in the present study did not reach statistical significance, the observation that periodontal disease was apparently more strongly associated with incident type 2 diabetes among women may be worthy of note, given recent findings that inflammation was a stronger predictor of type 2 diabetes in women than in men.<sup>[23]</sup> Rajhans NS found that periodontal disease increases in prevalence and severity with the age of the patient which was similar to the result of our study.<sup>[15]</sup>

Cerda *et al.*<sup>[24]</sup> had concluded that the duration of diabetes was a significant factor for the severity of periodontal disease. Emrich *et al.* stated that the diabetic status was significantly and strongly related to both prevalence and severity of periodontal disease. The severity of periodontal disease was more prevalent in diabetics who had the disease for >5 years. The finding of our study is consistent with the above studies but contradictory with Faulconbridge *et al.*<sup>[25]</sup>

From the present study, it can be speculated that poorer the control and longer the duration of diabetes, the greater will be the prevalence and severity of periodontal disease.

A limitation of this study is that we did not use a population-based sampling scheme to select DM2 patients. However, patients with DM2 regularly visit hospitals to make use of the laboratory facilities. Thus, selection of DM2 patients from the diabetic center does not mean that only a subset of patients with more severe DM2 patients was selected. The present study concludes that diabetes plays a role in the initiation and progression of periodontal disease along with multiple factors. Particularly poor metabolic control, as well as extended duration of diabetes, is a risk factor for periodontitis when extensive local irritants are present on teeth. The dentist can play an important role in diabetic patients overall health care through recognition and treatment of their periodontal needs understanding it as the "Sixth Complication of DM." The better you control your blood sugar level, the less likely you are to develop dental problems and vice versa.

### CONCLUSION

Risk of worsening of periodontal disease is associated with HbA1c in T2DM. Results support the clinical relevance of identifying patients with poor glycemic control and periodontitis and harness the individualized integrative approaches for the management of metabolic conditions and periodontitis by endocrinologists and periodontists. At the level of clinical practice, this concept would possibly reinforce the need for development and implementation of guidelines to screen prediabetes and diabetes in dental and medical primary care settings.

### REFERENCES

- American Diabetes Association. Diagnosis and classification of diabetes mellitus (Position Statement). *Diabetes Care*, 2009; 32 Suppl 1: S62-7.
- Mealy B. Diabetes mellitus. In: Greenberg MS, Glick M, editors. *Burket's Oral Medicine Diagnosis and Treatment*. 10<sup>th</sup> ed. New York: B.C. Decker Inc., 2003; 563-77.
- Awuti G, Younusi K, Li L, Upur H. Epidemiological survey on the prevalence of periodontitis and diabetes mellitus in Uyghur adults from rural Hotan area in Xinjiang. *Exp Diabetes Res.*, 2012; 2012: 1-7.
- Cerbone AM, Macarone-Palmieri N, Saldalamacchia G, Coppola A, Di Minno G, Rivellesse AA. Diabetes, vascular complications and antiplatelet therapy: Open problems. *Acta Diabetol*, 2009; 46: 253-61.
- Tsai C, Hayes C, Taylor GW. Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population. *Community Dent Oral Epidemiol*, 2002; 30: 182-92.
- Feres M, Figueiredo LC. Current concepts in the microbial etiology and treatment of chronic periodontitis. *J Int Acad Periodontol*, 2009; 11: 234-49.
- Highfield J. Diagnosis and classification of periodontal disease. *Aust Dent J.*, 2009; 54 Suppl 1: S11-26.
- Rees TD. The diabetic dental patient. *Dent Clin North Am*, 1994; 38: 447-63.
- Tervonen T, Oliver RC. Long-term control of diabetes mellitus and periodontitis. *J Clin Periodontol*, 1993; 20: 431-5.
- Belting CM, Hiniker JJ, Dummett CO. Influence of diabetes mellitus on the severity of periodontal disease. *J Periodontol*, 1964; 35: 476-80.
- Kiran M, Arpak N, Unsal E, Erdogan MF. The effect of improved periodontal health on metabolic control in type 2 diabetes mellitus. *J Clin Periodontol*, 2005; 32: 266-72.
- Loe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontol Scand*, 1963; 21: 533-51.
- Silness and Loe H. The gingival index, the plaque index and the retention index systems. *J Periodontol*, 1967; 38 Suppl: 610-6.
- GraphPad QuickCalcs: T test calculator. Available from: <http://www.graphpad.com/quickcalcs/ttest1.cfm>. [Last accessed on 2015 Feb 16].
- Rajhans NS, Kohad RM, Chaudhari VG, Mhaske NH. A clinical study of the relationship between diabetes mellitus and periodontal disease. *J Indian Soc Periodontol*, 2011; 15: 388-92.
- Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: An epidemiologic perspective. *Ann Periodontol*, 2001; 6: 99-112.
- Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: A two-way relationship. *Ann Periodontol*, 1998; 3: 51-61.
- Brown LJ, Brunelle JA, Kingman A. Periodontal status in the United States, 1988-1991: Prevalence, extent, and demographic variation. *J Dent Res.*, 1996; 75: 672-83.
- Janket SJ, Jones JA, Meurman JH, Baird AE, Van Dyke TE. Oral infection, hyperglycemia, and endothelial dysfunction. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*, 2008; 105: 173-9.
- Sandholm L, Swanljung O, Rytömaa I, Kaprio EA, Mäenpää J. Periodontal status of Finnish adolescents with insulin-dependent diabetes mellitus. *J Clin Periodontol*, 1989; 16: 617-20.
- Southerland JH, Taylor GW, Offenbacher S. Diabetes and periodontal infection. *Clin Diabetes*, 2005; 23: 171-8.
- Moreno PR, Murcia AM, Palacios IF, Leon MN, Bernardi VH, Fuster V, *et al*. Coronary composition and macrophage infiltration in atherectomy specimens from patients with diabetes mellitus. *Circulation*, 2000; 102: 2180-4.

23. Desvarieux M, Schwahn C, Völzke H, Demmer RT, Lüdemann J, Kessler C, *et al.* Gender differences in the relationship between periodontal disease, tooth loss, and atherosclerosis. *Stroke*, 2004; 35: 2029-35.
24. Cerda J, Vázquez de la Torre C, Malacara JM, Nava LE. Periodontal disease in non-insulin dependent diabetes mellitus (NIDDM). The effect of age and time since diagnosis. *J Periodontol*, 1994; 65: 991-5.
25. Faulconbridge AR, Bradshaw WC, Jenkins PA, Baum JD. The dental status of a group of diabetic children. *Br Dent J.*, 1981; 151: 253-5.