Diabetes Mellitus; Its Impact on Periodontal Health and Dental Caries

Nur Izzati Najwa Ramli[1], Syarifah Nur Izzati Alkaff Syed Mohamad Nadzir Alkaff[1], Ghasak Ghazi Faisal[2], Lina Hilal Al Bayati[3]*

1. Student, Kulliyyah of Dentistry, International Islamic University Malaysia.
2. Lecturer, Unit of Basic Medical Science, Kulliyyah of Dentistry, International Islamic University Malaysia.
3. Lecturer, Unit of Periodontics, Kulliyyah of Dentistry, International Islamic University Malaysia.

Abstract

To study the relationship between diabetes mellitus, periodontal health status and dental caries.

A case-control study involving 42 diabetic patients and 42 non-diabetic patients was conducted at the dental clinic, Kulliyyah of Dentistry, International Islamic University Malaysia. Full mouth periodontal examination was performed and their decay, missing, filling, total score (DMFT) was obtained.

The percentage of diabetic patients that were diagnosed with periodontitis was 88.1% compared to 59.5% in the control group and it was statistically significant ($p<0.05$). In comparing periodontitis in both groups, the bleeding on probing (BOP) and pocket depth (PD) showed no significant difference ($p>0.05$) while the clinical attachment loss (CAL) showed significant difference ($p<0.05$). The mean values for decay, missing and filled teeth in the diabetic group were 2.24±1.94, 8.52 ±6.13and 3.76± 2.79 respectively. Meanwhile, in the control group the mean values were 1.83±1.92, 4.79±3.80and 2.79 ±2.76 respectively. Among all three mean values, missing teeth was the only one that showed significant difference ($p<0.05$) However, the total DMFT score between the two groups showed statistically significant result ($p<0.05$).

Diabetic patients are more prone to periodontal destruction and tooth loss and they may also be at a higher risk of developing dental caries. Regular dental follow up are required for diabetic patients.

Clinical article (J Int Dent Med Res 2016; 9: (3), pp. 164-168)

Keywords: Diabetes mellitus, plaque score, bleeding on probing, pocket depth, clinical attachment loss, DMFT.

Received date: 29 September 2016 **Accept date:** 07 November 2016

Introduction

Diabetes mellitus is a growing public health concern and a common chronic disease affecting all age groups. The World Health Organization (WHO) has recently declared it to be pandemic[1]. In Malaysia, the prevalence of diabetes is also on the upward trend[2, 3].

Many chronic macrovascular and microvascular complications of diabetes have been reported in the literature and oral manifestations and complications is reported recently as a major complication of diabetes mellitus[4].

A number of inflammatory diseases and soft tissue pathologies in oral cavity are associated with diabetes mellitus[5]. These include periodontal diseases, dental caries, salivary dysfunction, oral mucosal diseases, oral infections, taste and other neurosensory disorders[6].

The oral complications of diabetes mellitus, particularly from poorly controlled disease, are numerous and devastating[7]. These complications include xerostomia, an increased susceptibility to bacterial, viral, and fungal infections (oral candidiasis), poor wound healing, gingivitis, periodontal disease, periapical abscesses, taste impairment and burning mouth syndrome[8].

Corresponding author:
Dr. Ghasak Ghazi Faisal
Pathologist, Basic Medical science Unit
Kulliyyah of Dentistry, International Islamic University Malaysia
Jalan Sultan Ahmad Shah
Kuantan 25150
Pahang, Malaysia
E-mail: drghasak@iium.edu.my

Nationwide surveys have demonstrated that people with diabetes, especially poorly controlled diabetes, have a significantly higher prevalence of severe periodontitis.[9]Several mechanisms have been proposed to explain the increased susceptibility to periodontal diseases, including alterations in host response, subgingivalmicroflora, collagen metabolism, vascularity, gingival crevicular fluid and heredity patterns.[10]

The relationship between diabetes and dental caries has been investigated, but no clear association has been clarified. It is important to note that patients with diabetes are susceptible to oral sensory, periodontal and salivary disorders, which could increase their risk of developing new and recurrent dental caries.[11] For example, several studies have reported a greater history of dental caries in people with diabetes.[12]Factors for caries development include the traditional elements (for example, *Streptococcus mutans* levels, previous caries experience), as well as poor metabolic control of diabetes[13] underscoring the need for dental professionals to follow up all patients with diabetes on a regular basis for new and recurrent dental decay. Caries risk is also heightened by xerostomia. People with diabetes are more likely to experience xerostomia owing to the side-effects of some medications and poor blood glucose control.[6]

Materials and methods

This is a case controlstudy conducted on diabetic patients attending the Kulliyyah of Dentistry Polyclinic, International Islamic University Malaysia. A total of 42 diabetic patients and 42 age and sex matched control patients attending the Periodontal Clinic were selected purposively depending on the inclusion and the exclusion criteria.

Inclusion criteria for diabetic patients sample:

1) Patient who have been diagnosed with diabetes mellitus by the medical practitioner for at least one year.
2) Subjects aged 18 years old and above.
3) Able to give written consent.
4) Males and females.
5) Not on any antibiotic treatment for the past month.

Exclusion criteria

1) Pregnant patient.
2) Smoker patient.
3) Edentulous patient.
4) Patients with other systemic systemic illness.

Inclusion and exclusion criteria for control group

The same as above except that the patient is non-diabetic.

Ethical approval and consent

Ethical approval number (IREC 250) was obtained from the IIUM Research Ethics Committee prior to commencing the study. All patients were informed verbally and were given the patient information sheet to explain about the purpose of the study. A written informed consent was obtained from the patient after explaining the details of clinical procedures prior to participation.

The patients were asked to fill the patient's data form that was provided**.** The data includes gender, age, race, level of education, income per month. For the diabetic patients, we collected data on the disease duration, type of treatment, complications of diabetes mellitus and regular follow-up with the diabetic clinic.

Clinical examination

Random blood sugar test was conducted to measure patient's blood sugar level. All patients underwent a full-mouth clinical examination at six sites per tooth (third molars excluded), using a manual periodontal probe. The periodontal parameters included the following assessments:

a) Probing depth (PD): the distance of the free gingival margin to the base of the probeable pocket, recorded to nearest mm.
b) Clinical attachment loss (CAL): the distance of cemento-enamel junction to the base of the probeable pocket, recorded to the nearest mm.
c) Bleeding on probing (BOP) and dental plaque (PS)

The patients were also evaluated for the decayed, missing and filled teeth index (DMFT).

Statistical analysis

All data that has been recorded were analyzed using SPSS version 20.0.

Results

Overall Periodontal Health Status

The results of the periodontal examination of all the included patients are shown in table

1.The plaque score (PS) is significantly higher in the control group than the diabetic group with the mean PS being 77.3±20.76 and 67.46%.±22.77 for the control and diabetic groups respectively. The bleeding on probing percentage (BOP) between the two groups was proven to be not significant with *p*> 0.05. The mean percentage of BOP for diabetic group was 55.11%±23.43while the control group was 60.03%±21.95.

The percentage of probing depth of 1 to 3 mm was statistically significant with *p*< 0.05. The mean percentages were 76.13%±17.5 and 86.45%±13.7 for diabetic and control groups respectively. However for probing depth ≥4mm, the diabetic group showed a statistically significant higher value than the control group (22.73%±17.65 and 12.67%±12.4) respectively. The result for clinical attachment loss (CAL) showed significant different for the two groups with *p*-value <0.05. The mean value for the percentage of (CAL) of 1 to 3 mm for the diabetic group was 22.32% while the mean for control was 8.99%. For the CAL≥ 4mm, the mean percentage for diabetic group was 39.84% and 13.45% in the control group.

	DM status	Mean±Standard deviation	*p*-value
PS (%)	DM Control	67.46±22.77 77.33±20.76	*p*< 0.05
BOP (%)	DM Control	55.11±23.43 60.03±21.95	*p*> 0.05
PD 1 to 3mm (%)	DM Control	76.13±17.52 86.54±13.77	*p*< 0.05
PD ≥ 4mm (%)	DM Control	22.73±17.65 12.66±12.41	*p*< 0.05
CAL 1 to 3mm (%)	DM Control	22.32±21.96 8.99±9.34	*p*< 0.05
CAL ≥ 4mm (%)	DM Control	39.84±29.15 13.45±14.63	*p*< 0.05

Table 1. The periodontal parameters recorded during clinical examination of all patients.

The severity of periodontitis

Among the 42 diabetic patients, 37 of them were diagnosed with periodontitis while 25 out of 42 control patients were diagnosed with periodontitis and this is statistically significant *p*<0.05. Table 2 summarizes the clinical periodontal characteristics of the patients diagnosed with periodontitis. The plaque score (PS) showed significant difference between the two groups in which the diabetic group was proven to have a lower plaque score compared to the control group. The mean percentage of plaque score for the diabetic group was 68.86%±23.27 while the control group was 80.84%±21.08. The percentage of bleeding on probing (BOP) between the two groups was not significant with *p*> 0.05. The mean percentage of BOP for diabetic group was 57.98%±23.30 while the control group was 64.84%±21.32. The percentage of probing depth of 1 to 3 mm was statistically not significant (*p*> 0.05). The mean percentages for both groups were 73.00%±16.27 and 78.75%±12.68 respectively. The same result goes to percentage of probing depth ≥4mm which was also not significant. The mean percentage for diabetic group was 25.71%±16.67 while for control group was 19.94%±10.98.
However, as we expected, the result for clinical attachment loss (CAL) showed significant different for the two groups *p*<0.05. The mean value for the percentage of (CAL) of 1 to 3 mm for the diabetic group was 24.86%±22.18while the mean for control was 12.83%±9.96. For the CAL ≥4mm, the diabetic patients also showed a statistically higher score of 44.61%±27.73 versus 19.23%±14.70for the control group.

	DM Status	Mean±Std. Deviation	*p*-value for difference
PS (%)	DM Control	68.86±23.27 80.84±21.08	*p*< 0.05
BOP (%)	DM Control	57.98±23.30 64.84±21.32	*p*> 0.05
PD 1 to 3mm (%)	DM Control	73.00±16.27 78.75±12.68	*p*> 0.05
PD ≥ 4mm (%)	DM Control	25.71±16.67 19.94±10.98	*p*> 0.05
CAL 1 to 3mm (%)	DM Control	24.86±22.18 12.83±9.96	*p*< 0.05
CAL ≥ 4mm (%)	DM Control	44.61±27.73 19.23±14.70	*p*< 0.05

Tabel 2. The periodontal parameters of patients with periodontitis.

DMFT Index

Table 3 summarizes the DMFT (decay, missing, filled, and total) index recorded during the examination. The decay status between the diabetic and control group was statistically not significant *p*> 0.05. The mean decay value for the diabetic group was 2.24±1.936 while for the control group was 1.83±1.924. Meanwhile, the missing teeth between both diabetic and control group showed statistically significant difference with *p*< 0.05. The mean missing teeth in the diabetic group was 8.52±6.126 which was higher than the control group 4.79±3.803 and the difference is statistically significant *p*< 0.05. As

for the filled teeth, no significant difference was found between the two groups with *p*-value > 0.05.

The total DMFT index was proven to be significantly higher in the diabetic group with *p*< 0.05. The mean total DMFT in the diabetic group was 14.52±6.922 far higher than 9.40±3.87 in the control group.

	DM status	Mean±Std. Deviation	*p*-value
Decay status	DM	2.24±1.93	*p*> 0.05
	Control	1.83±1.92	
Missing teeth	DM	8.52±6.12	*p*< 0.05
	Control	4.79±3.80	
Filled teeth	DM	3.76±2.79	*p*> 0.05
	Control	2.79±2.76	
Total DMFT value	DM	14.52±6.92	*p*< 0.05
	Control	9.40±3.87	

Table 3. DMFT index recorded during examination.

Discussion

In our study, we assessed the periodontal status of diabetic and non-diabetic patients by recording the full mouth charting of the percentage of plaque score (PS), bleeding on probing (BOP), probing depth and clinical attachment loss (CAL). From the results mentioned above, the plaque score of the diabetic patients were lower compared to the control patients but the result also showed that that diabetic patients have more periodontal destruction than the control patients. The result proved that even with lower plaque score, diabetic patients tend to have a severe periodontal destruction, while for the control patients, the periodontal destruction was mainly caused by poor plaque control. Over the past 15 years, there have been several reports and studies showing that poor oral health, especially the extent and severity of periodontal disease, are associated with a range of systemic conditions[14]

In this study, the BOP showed no significant difference between both groups, and this proved that both groups suffer from periodontal inflammation. The probing depth between all the patients involved were proven to be significant, however the result was not the same if we compared among periodontitis patients only. The 37 periodontitis patients in diabetic groups showed no significant difference in probing depth compared to 25 periodontitis patients in the control group. On the other hand, the diabetic group produced significantly greater amount of CAL compared to control group regardless if we compare them among all the patients or among periodontitis patients only. During recent years, the influence of systemic factors on the pathogenesis of periodontal disease has been assessed, and consistent evidence has been proved establishing both type 1 and type 2 diabetes mellitus as risk factors for the initiation and progression of periodontal disease[15,12].

Several mechanisms have been proposed to explain the greater incidence and severity of periodontal disease in diabetic subjects[16]. These include polymorphonuclear dysfunction, vascular changes, altered collagen and glycosaminoglycan synthesis, deregulated cytokine production and the formation of advanced glycation end-products (AGE). AGE through their receptors induced the expression of pro-inflammatory cytokines. Therefore AGE-receptor interaction amplifies the magnitude of cytokine expression and response, which may further amplify the progression and severity of periodontitis[17]. In our study, all 42 diabetic patients involved were type 2 diabetes patients. Type 2 diabetes, formerly known as noninsulin-dependent diabetes, represents the most common type of diabetes which is caused by resistance to insulin combined with a failure to produce enough insulin to compensate this resistance. In studies carried out on Pima Indians, a population suffering from a very high incidence of type 2 diabetes, diabetic subjects had a significantly greater risk of progressive bone loss compared to control subjects[18].

We assessed the caries status of the patient by recording their DMFT (decay, missing, filled, and total) index. The occurrence of dental caries in patients with diabetes mellitus has been studied, but no specific association has been identified. The relationship between dental caries and diabetes mellitus is complex. Children with type 1diabetes often are given diets that restrict their intake of carbohydrate-rich, cariogenic foods, whereas children and adults with type 2 diabetes, which often is associated with obesity and intake of high-calories and carbohydrate-rich food, can be expected to have a greater exposure to cariogenic foods[19, 20].

In our study, we found no significant difference in decay and filled teeth among the

diabetic and control group. In contrast, missing teeth and the total DMFT index showed statistically significant difference. Most of the diabetic patients have lost their teeth and these have been reflected by significant number of missing teeth. This consequently leads to lower number of decay and filled teeth in diabetic patients. However, in our study we were not able to identify the causes of the missing teeth as it may be due to caries, periodontal disease or other causes. All in all, the significantly higher total DMFT index in diabetic patients was mainly due to a great significant difference of missing teeth among the diabetic subjects. Although, we could not finda consistent pattern regarding the relationship of dental caries and diabetes, however, the reduction in salivary flow that occurs in patients with diabetes is a risk factor for dental caries[21], so we can conclude that diabetic patients generally have a higher risk for dental caries but this higher risk is masked by the early tooth loss that occurs in these patients making it not possible to determine the exact prevalence of dental caries in these patients. Our results are consistent with previous studies that concluded that diabetic patients have higher risk factors for dental caries.[22,23,24]

Conclusions

We can conclude from our study that diabetic patients have more severe periodontitis and that diabetes mellitus negatively affects oral health as reflected by the higher DMFT scores in diabetic patients.

Acknowledgements

We would like to thank the Kulliyyah of Dentistry, International Islamic University Malaysia and the staff working at the dental polyclinic in their help in recruiting patients for this study.

Declaration of Interest

The authors report no conflict of interest, the research was funded by a research grant from the International Islamic University Malaysia, grant number: EDW B 14-108-0993.

References

1. Global report on diabetes. World Health Organization, WHO press, Geneva, 2016.
2. Cheah WL, Lee PY, Lim PY, Fatin Nabila AA, Luk KJ, Nur Iwana AT. Perception of Quality of Life among People with Diabetes. Malaysian Family Physician 2011; 7:21-29.
3.Letchuman GR, Wan Nazaimoon WM, Wan Mohamad WB, Chandran LR, Tee GH, Jamaiyah H, *et al*. Prevalence of diabetes in the Malaysian national health morbidity survey III.Medical J Malaysia. 2010 Sep; 65(3):180-6.
4. Ira BL, Evanthia L, Wenche, George WT. The Relationship Between Oral Health and Diabetes Mellitus. The journal of American dental association 2008; 139(suppl 5):19S-24S
5.Chapple IL, Genco R. Diabetes and periodontal diseases: consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases. Journal of clinical periodontology. 2013;40(s14).
6. Sandberg GE, Sundberg HE, Fjellstrom CA, Wikblad KF. Type 2 diabetes and oral health: a comparison between diabetic and non-diabetic subjects. Diabetes research and clinical practice 2000; 50(1): 27-34.
7.Bajaj S, Prasad S, Gupta A, Singh VB. Oral manifestations in type-2 diabetes and related complications. Indian journal of endocrinology and metabolism. 2012;16(5):777.
8. Khalid AR. Diabetes Mellitus and Its Oral Complications: A Brief Review. Pakistan Oral & Dental Journal.2006; 26 (1): 97-100.
9. Tsai C, Hayes C, Taylor GW .Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population. Community Dental Oral Epidemiology, 2002; 30: 182–92.
10. Brian LM. Periodontal Disease and Diabetes. The journal of the American dental association 2006; 137: 26S-31S.
11. Ship, Jonathan A. Diabetes and oral health: an overview. The Journal of the American Dental Association 2003; 134: 4S-10S.
12. Moore PA, Weyant RJ, Etzel KR, et al .Type 1 diabetes mellitus and oral health: assessment of coronal and root caries. Community Dental Oral Epidemiology 2001; 29: 183–94.
13. Twetman S, Johansson I, Birkhed D, Nederfors T. Caries incidence in young type 1 diabetes mellitus patients in relation to metabolic control and caries-associated risk factors. Caries Research 2002 36(1):31–5.
14. Griffin SO, Barker LK, Griffin PM, Cleveland JL, Kohn W. Oral health needs among adults in the United States with chronic diseases. The journal of the American dental Association 2009;140(10):1266-1274.
15. Duarte PM, De Oliveira MC, Tambeli CH, Parada CA, Casati MZ, Nociti FH. Overexpression of interleukin-1β and interleukin-6 may play an important role in periodontal breakdown in type 2 diabetic patients. Journal of periodontal research 2007; 42(4):377-81.
16. Preshaw PM, Alba AL, Herrera D, Jepsen S, Konstantinidis A, Makrilakis K, Taylor R. Periodontitis and diabetes: a two-way relationship. Diabetologia. 2012;55(1):21-31.
17. Katz J, Bhattacharyya I, Farkhondeh-Kish F, Perez FM, Caudle RM, Heft MW. Expression of the receptor of advanced glycation end products in gingival tissues of type 2 diabetes patients with chronic periodontal disease: a study utilizing immunohistochemistry and RT-PCR. Journal of clinical periodontology 2005; 32(1):40-4.
18. Irwin C, Mullally B, Ziada H, Allen E, Byrne PJ. Risk Factors and Susceptibility in Periodontitis. Dental update 2007; 34(5): 270-2.
19. Taylor GW, Manz MC, Borgnakke WS. Diabetes, periodontal diseases, dental caries, and tooth loss: a review of the literature. Compendium of continuing education in dentistry 2004 ;3:179-84.
20. Twetman S, Johansson I, Birkhed D, Nederfors T. Caries incidence in young type 1 diabetes mellitus patients in relation to metabolic control and caries-associated risk factors. Caries Research 2002; 36(1):31–5.
21. Jawed, Muhammad, et al. Protective effects of salivary factors in dental caries in diabetic patients of Pakistan. Experimental diabetes research 2012 (2012).
22. Siudikiene J, Machiulskiene V, Nyvad B, Tenovuo J, Nedzelskiene I. Dental caries and salivary status in children with type 1 diabetes mellitus, related to the metabolic control of the disease. European journal of oral sciences 2006 ;114(1):8-14.
23. Sandberg, Gun E., etal. Type 2 diabetes and oral health: a comparison between diabetic and non-diabetic subjects. Diabetes research and clinical practice 2000; 50(1): 27-34.
24. Iqbal S, Kazmi F, Asad S, Mumtaz M, Khan AA. Dental caries and diabetes mellitus.Pakistan Oral & Dental Journal. 2011;31(1).