

Retrospective Study of the Prevalence of Type 2 Diabetes Mellitus and Severity of Periodontal Disease in Chronic Periodontitis Patients

Waleed Ahmed Ismail¹, Siti Lailatul Akmar Zainuddin^{2*}, Romaisa Arshad Khokhar³,
Haslina Taib⁴, Basaruddin Ahmad⁵, Azlina Ahmad⁶

1. Department of Periodontics, School of Dental Sciences, Universiti Sains Malaysia, Kelantan.
2. Department of Periodontics, School of Dental Sciences, Universiti Sains Malaysia, Kelantan.
3. Department of Oral Pathology, Islamic International Dental College, Riphah International University, Islamabad.
4. Department of Periodontics, School of Dental Sciences, Universiti Sains Malaysia, Kelantan.
5. Department of Medical Statistics, School of Dental Sciences, Universiti Sains Malaysia, Kelantan.
6. School of Dental Sciences, Universiti Sains Malaysia, Kelantan.

Abstract

Diabetes mellitus is a long-standing risk factor for chronic periodontitis. Both the diseases share a two-way relationship, the common factor being the inflammatory-mediated pathway. The aim of this study was to assess the prevalence of type 2 diabetes mellitus and the severity of chronic periodontitis, among chronic periodontitis patients, who attended the Periodontics Clinics at Hospital Universiti Sains Malaysia, Kelantan in 2017.

It was a retrospective, cross-sectional study. The data was taken from patient records who attended the Dental Clinics at Hospital USM in the year 2017 and were diagnosed with chronic periodontitis.

166 patients with chronic periodontitis were recruited for the study. The medical history and periodontal charting of the chronic periodontitis subjects were retrieved from their records, categorizing them into test group (type 2 diabetes mellitus and chronic periodontitis) and control group (chronic periodontitis only). Statistical analysis was done using SPSS 24.0 and independent t-test was used for the comparison of mean scores.

Overall, 47 patients (28.31%) of chronic periodontitis had type 2 diabetes mellitus whereas 119 patients (71.69%) were non-diabetic. The mean age of diabetics with chronic periodontitis was also greater than the non-diabetics, with a slight predominance of male subjects. Similarly, all the periodontal parameters were far worse in diabetics with chronic periodontitis as compared to the non-diabetics.

This study demonstrated that the prevalence of chronic periodontitis patients with type 2 diabetes mellitus in Hospital Universiti Sains Malaysia for the year 2017 was lower than chronic periodontitis patients without type 2 diabetes mellitus. Also, it was deduced that type 2 diabetes mellitus has an adverse effect on the periodontal apparatus as periodontal status of chronic periodontitis patients suffering from type 2 diabetes mellitus was worse than non-diabetic chronic periodontitis patients.

Clinical article (J Int Dent Med Res 2020; 13(2): 595-600)

Keywords: Chronic periodontitis, diabetes mellitus, periodontal parameters, prevalence, retrospective.

Received date: 03 July 2019

Accept date: 08 October 2019

Introduction

Diabetes mellitus is a serious, chronic disease characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or

both. Type 2 diabetes mellitus (T2DM) is the most common form. Diabetes of all types can lead to long-term complications including retinopathy, nephropathy, autonomic and peripheral neuropathy, cardiovascular symptoms and sexual dysfunction¹. Diabetes was the eighth leading cause of death among both males and females. The total burden of deaths from high blood glucose in 2012 was estimated to 3.7 million, which includes 1.5 million diabetes deaths, and 2.2 million deaths from

*Corresponding author:

Dr. Siti Lailatul Akmar Zainuddin
Department of Periodontics, School of Dental Sciences, Universiti
Sains Malaysia Health Campus, 16150, Kelantan
E-mail: lailatul@usm.my

cardiovascular diseases, chronic kidney disease, and tuberculosis related to higher-than-optimal blood glucose². According to World Health Organization (WHO) survey, the number of people with diabetes has risen from 108 million in 1980 to 422 million in 2014. The global prevalence of diabetes among adults over 18 years of age has risen from 4.7% in 1980 to 8.5% in 2014 and has been rising more rapidly in middle- and low-income countries.

Over the past decade, there has been an increasing prevalence of T2DM among adults aged ≥ 30 years in Malaysia. In 2011, the fourth Malaysian National Health and Morbidity Survey (NHMS IV) reported that the prevalence of T2DM increased to 20.8%, affecting 2.8 million individuals, compared with the third National Health and Morbidity Survey (NHMS III), which reported a prevalence of 14.9% in 2006³. Similarly, American Diabetes Association reports that in 2015, 30.3 million Americans, or 9.4% of the population, had diabetes⁴.

Periodontal disease is a chronic inflammatory disease of the supporting tissues of the teeth and it is caused by bacterial plaque that harbors periodontal pathogenic microorganisms such as *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Prevotella intermedia* and others⁵. Chronic periodontitis is characterized by bleeding and inflamed gingiva, pocket formation, loss of connective tissue attachment and alveolar bone resorption which can eventually result in tooth loss. The disease has significant impact on quality of life in terms of aesthetic, mastication and speech as the teeth may become progressively mobile and may exfoliate spontaneously if treatment is not provided⁶.

In 2010, Kassebaum and co-workers found that severe periodontitis was the sixth-most prevalent condition, affecting 10.8% or 743 million people worldwide⁷. The recent National Oral Health Survey of Adults (NOHSA) 2010 carried out in Malaysia found that 94% of dentate Malaysians adults have some forms of periodontal disease varying from mild gingivitis to severe periodontitis. More worrying, the prevalence of severe periodontitis in Malaysia showed a sharp increase among adults from 7.2% in 2000 to 25.3% in 2010. Prevalence of chronic periodontitis had been on the rise year after year especially after year 2000 which was clearly stated in NOHSA 2010.

Diabetes mellitus and periodontal disease are both common, chronic conditions and multiple studies have demonstrated that diabetes mellitus (type 1 and type 2) is an established risk factor for periodontitis⁸⁻¹⁰. In the review of dental records, it was evident that patients with chronic periodontitis had more co-morbidities, including diabetes (27.8%), cardiovascular diseases (5.8%), hypertension (28.3%) and others (10.4%)¹¹.

Many reviews suggested two-way relationship between periodontitis and diabetes mellitus^{9,12}. Improved glycemic control may alleviate periodontal disease. In turn, control of periodontal disease was postulated to enhance glycemic control. Many studies^{9,13,14} showed that scaling and root planing (SRP) improve the glycemic control in periodontal patients. SRP and topical antibiotic also showed favorable clinical and glycemic control in chronic periodontitis with T2DM^{15,16}.

The current knowledge indicates that enhanced release of proinflammatory cytokines (interleukin-1 β , interleukin-6 and tumor necrosis factor- α), an altered receptor activator of nuclear factor kappa-B ligand/osteoprotegerin ratio, advanced glycation end-product/receptor for advanced glycation end-product interactions, increased production of reactive oxygen species and increased leukocyte-endothelial cell interaction play crucial roles in the two-way relationship between diabetes and periodontitis. These complex changes, resulting from the presence of diabetes, modify the local inflammatory reaction in the periodontium of patients with diabetes, leading to a proinflammatory state in the gingival tissue and the microcirculation¹⁷.

The main purpose of this study was to describe the prevalence of T2DM in patients with chronic periodontitis attending periodontal clinic from the Record Unit HUSM in 2017.

Materials and methods

Study Design

This was a retrospective, cross-sectional study design conducted among patients who attended Dental Clinics at HUSM in the year 2017 and were diagnosed with chronic periodontitis.

Ethical Consideration

The ethical approval was obtained from

The Human Research Ethics Committee of USM (JEPeM) with ID number USM/JEPeM/17030192.

Data Collection

During this study, data of 166 diagnosed patients of chronic periodontitis was analyzed and retrieved using their records which were obtained from the Records Unit. There were 92 (55%) males and 74 (45%) females.

All information such as patients' socio-demographic data (age, gender and race), medical history and periodontal parameters (plaque score, gingivitis score, probing pocket depth and clinical attachment level) were collected.

All the selected patients were adults aged between 18-70 years. Only the patients diagnosed with mild to moderate chronic periodontitis i.e., with a mean probing pocket depth >3 and <5 mm (mild), and ≥5 and <7 mm (moderate)¹⁸ were selected. For the test group, only the patients clinically diagnosed with T2DM at least 6 months prior were selected.

Pregnant and lactating women, patients with acute or chronic medical disorders and those who had undergone periodontal therapy at least 3 months prior to data collection were excluded from the sample.

Data and Statistical Analysis

All data was analyzed using SPSS version 24.0 statistical software. Continuous data was summarized as mean with standard deviation (SD) while discrete data (categorical) as mean percentage with standard deviation (SD). Continuous variables were compared by analysis of variance (ANOVA) and the significance of mean difference between the groups was done by independent t-test. Categorical variables were also compared by independent t-test. *P* value of 0.05 was taken as indicative of statistical significance.

Results

The total data collected was 166 samples which all were diagnosed cases of chronic periodontitis. There were 47 diabetic (28.31%) and 119 non-diabetic (71.69%) chronic periodontitis patients, respectively. The number of chronic periodontitis subjects with diabetes mellitus were considerably lower as compared to chronic periodontitis subjects without diabetes mellitus (Table 1).

The mean age of diabetics was 56.43

years (SD 11.70) and that of non-diabetics was 43.46 years (SD 11.39) (*p*=0.002). Among the diabetic subjects, there were 28 males (59.57%) and 19 females (40.43%) as compared to 64 males (53.78%) and 55 females (46.22%) (*p*=0.498) among the non-diabetic subjects. There were predominantly more males than females in both the groups but the ratio of males to females for each group was almost similar. The result for race shows that majority of the subjects were Malay while a small number of the total population constituted of Chinese. There were a total of 43 Malay (91.49%) and 04 Chinese (8.51%) in the diabetic group whereas the non-diabetic group consisted of 107 Malay (89.92%) and 12 Chinese (10.08%) (*p*=1.000) subjects (Table 1).

Demographic Characteristics	Cumulative Samples (n = 166)	Groups		<i>p</i> value
		Diabetic (n = 47)	Non-Diabetic (n = 119)	
Age [mean (SD), in years]	47.16 (12.81)	56.43 (11.70)	43.46 (11.39)	0.002
Gender	92 (55.42%)	28 (59.57%)	64 (53.78%)	0.498
Male, n (%)	74 (44.58%)	19 (40.43%)	55 (46.22%)	
Female, n (%)				
Race	150 (90.36%)	43 (91.49%)	107 (89.92%)	1.000
Malay, n (%)	16 (9.64%)	04 (8.51%)	12 (10.08%)	
Chinese, n (%)				

Table 1. Demographic Data of Study Subjects.

Values represent the means and standard deviations or numbers (%) of subjects/sites.

The clinical parameters retrieved consisted of plaque score, gingivitis score, probing pocket depth and clinical attachment level for chronic periodontitis subjects with and without diabetes. The mean plaque score for the diabetic group was 65.71% (SD 18.14) and for the non-diabetic group, it was 41.98% (SD 20.36) (*p*=0.043). The mean gingivitis score for the diabetics was recorded as 72.37% (SD 11.78) and 33.25% (SD 14.57) (*p*<0.001) for the non-diabetics. Both average plaque and gingivitis score results show higher score by percentage in chronic periodontitis subjects with diabetes mellitus compared to non-diabetic subjects. Similarly, the probing pocket depth and clinical attachment levels were greater in values for chronic periodontitis subjects with diabetes mellitus as compared to their non-diabetic counterparts. For the diabetic group, the mean probing pocket depth was 5.12mm (SD 0.82) and it was 4.21mm (SD 0.36) (*p*<0.001) for the non-

diabetic group. The mean clinical attachment level for the diabetics was 5.53mm (SD 0.78) while for non-diabetics, it came out to be 4.57mm (SD 0.41) ($p < 0.001$; Table 2).

Clinical parameters	Diabetic	Non-Diabetic	p value
Plaque score [mean (SD), in %]	65.71 (18.14)	41.98 (20.36)	0.043
Gingivitis score [mean (SD), in %]	72.37 (11.78)	33.25 (14.57)	<0.001
Probing pocket depth [mean (SD), in mm]	5.12 (0.82)	4.21 (0.36)	<0.001
Clinical attachment level [mean (SD), in mm]	5.53 (0.78)	4.57 (0.41)	<0.001

Table 2. Periodontal Parameters of Study Subjects.

Values represent the means and standard deviations or numbers (%) of subjects/sites.

Discussion

The two-way relationship between chronic periodontitis and diabetes mellitus has been cemented by numerous prior studies^{9,19}, each one being a risk factor for the other. This relationship basically relies on the inflammation pathway that is a factor in the pathogenesis of both chronic periodontitis as well as diabetes mellitus. The inflammation process results in the release of certain mediators which are the effector to bring about the changes in the normal healthy state of the cells or tissues and cause them to become diseased. If these mediators can be controlled or their effects be toned down, this can result in the decrease or possible prevention of the harmful effects of a disease, in this case being chronic periodontitis and/or diabetes mellitus.

The total number of chronic periodontitis subjects for the prevalence study were 166, out of which 47 (28.31%) were diabetic and 119 (71.69%) were non-diabetic. This is almost in conjunction to the study done in Saudi Arabia which estimated the prevalence of DM in adults to be 23.7%²⁰. But the result of the present study was in contrast to the study done in Malaysia in 2013 which reported a prevalence of merely 3.2% of diabetic subjects among chronic periodontitis subjects²¹. This difference might owe to a larger sample size used for the latter study, or the fact that there has been a sharp rise in the prevalence of diabetes in the last five years in Malaysia (particularly among the Kelantan population).

There was a significant difference in the mean ages of the test and control groups. The

mean age for diabetics with chronic periodontitis was greater than that of non-diabetics with chronic periodontitis. There is a marginal significance in this result, depicting that DM type-II has a late onset in life as compared to chronic periodontitis. This also depicts that chronic periodontitis is prevalent among the middle-age groups.

According to gender distribution, in both the groups, there were predominantly more males than females. But overall, there was no significant difference in the male to female ratio in both the groups. Therefore, it can be concluded that there is no difference in the prevalence of either T2DM or chronic periodontitis in accordance to gender. In another prevalence study done in 2017, the mean age for the subjects was reported at 50 years, which is similar to our study and strengthens the fact that chronic periodontitis is more common among middle-aged people, but they had a slight majority of females than males²². Although the male to female ratio difference between this study and ours is almost negligible, the minute difference may be accounted for by regional changes and all the factors associated with it, for example, environment, diet etc.

For both the groups, the subjects were predominantly Malays, accounting for almost 90% of total sample size. Other subjects were all Chinese. This can be explained by the fact that the total population of Malaysia consists majorly of the Malay race, followed by the Chinese group, then the Indians and the rest follow²³. Specifically in Kelantan, 95% of the total population is Malay, followed by Chinese to be the second most predominant race²⁴.

Generally, all the periodontal parameters were significantly higher in subjects with diabetes and chronic periodontitis as compared to subjects with chronic periodontitis alone. These results prove that the severity of chronic periodontitis is markedly increased due to T2DM. A recent study conducted in Saudi Arabia was in line with our results, showing significantly higher levels of all periodontal parameters in chronic periodontitis subjects with diabetes as compared to the control group²⁵. Another study conducted in Italy in 2005 supported our results, showing marked increase in periodontal parameters of diabetics as compared to the controls²⁶.

These results go on to signify that diabetes mellitus plays an important role in

furthering the periodontal destruction of a patient. The periodontal status of diabetic subjects is worse than non-diabetic subjects with chronic periodontitis. From these results, it can be concluded that hyperglycemia worsens the periodontal status of subjects as compared to normoglycemic individuals. Primary explanation for this is that chronic hyperglycemia increases the production of proinflammatory cytokines, such as interleukin-6 (IL-6) and matrix metalloproteinases (MMPs), by human gingival fibroblasts (HGFs) as compared to normal glucose²⁷. Another explanation for this result is that the interaction between advanced glycosylated end-products and their receptors is significantly higher in inflamed periodontal tissues with induced hyperglycemia than in normoglycemic individuals. This explanation was proved in a study conducted on rats in which diabetes and periodontitis was induced in the rats²⁸. Another study reported that hyperglycemia increases the expression of Toll-like receptors (TLRs) in periodontal tissues, which contribute to a greater inflammatory response in hyperglycemic subjects and, resultantly, to periodontal disease as well²⁹. This suggests that as the severity of hyperglycemia rises, the periodontal inflammatory response is also expected to increase. Another study suggested that diabetes results in changes in the function of immune cells, including neutrophils, monocytes and macrophages³⁰. This will eventually predispose to chronic inflammation, progressive tissue breakdown and diminished tissue repair capacity. This, in turn, causes chronic periodontitis to aggravate.

A new theory emerged, stating that the presence of untreated periodontal infection can increase the risk of poor glycemic control. Data from a prospective study on subjects with T2DM indicated that individuals with severe periodontitis had diabetes for a longer duration and higher fasting glucose levels³¹. A study by Kiran, Arpak³² found that the glycemic control had improved in T2DM subjects after a three month non-surgical periodontal therapy as compared to diabetic subjects without periodontal therapy. This proves the two-way relationship between T2DM and chronic periodontitis as both affect the severity of each other.

Conclusions

This present study demonstrated that the prevalence of chronic periodontitis patients with type 2 diabetes mellitus in Hospital USM Periodontics clinic in the year 2017 was lower than chronic periodontitis patients without type 2 diabetes mellitus. But the prevalence might vary from year to year and nothing constant can be concluded about the prevalence of diabetes mellitus in chronic periodontitis patients from these results. Probably an assessment of more than one year and with larger population will give a better idea of the prevalence of diabetes mellitus in patients with chronic periodontitis.

Another deduction from this study was that the periodontal status of chronic periodontitis patients suffering from type 2 diabetes mellitus was worse than non-diabetic chronic periodontitis patients. This signifies that hyperglycemia is a root cause of an exaggerated inflammatory response which causes an increase in the release of proinflammatory cytokines, causing further degradation of the periodontal apparatus.

Declaration of Interest

The authors report no conflict of interest and the article is not funded or supported by any research grant.

References

1. American Diabetes A. Diagnosis and Classification of Diabetes Mellitus. *Diabetes care*. 2010;33(Suppl 1):62-9.
2. Faisal GG, Radeef AS. Depression, Anxiety and Stress among Diabetic and Non-Diabetic patients with Periodontitis. *Journal of International Dental and Medical Research*. 2017;10(2):248-52.
3. Hussein Z, Taher SW, Gilcharan Singh HK, Chee Siew Swee W. Diabetes Care in Malaysia: Problems, New Models, and Solutions. *Annals of Global Health*. 2015;81(6):851-62.
4. Economic Costs of Diabetes in the U.S. in 2012. *Diabetes care*. 2013;36(4):1033-46.
5. Kusumaningsih T, Luthfi M, Moffan MD. Macrophages Analysis on Gingival Tissue of Diabetic Rats after Insulin Leaf Extract Administration. *Journal of International Dental and Medical Research*. 2018;11(1):308-11.
6. Newman, M. G., In Takei, H. H., In Klokkevold, P. R., & In Carranza, F. A. Carranza's clinical periodontology. 2015:875-6.
7. Kassebaum NJ, Bernabé E, Dahiya M, Bhandari B, Murray CJL, Marcenes W. Global Burden of Severe Periodontitis in 1990-2010: A Systematic Review and Meta-regression. *Journal of dental research*. 2014;93(11):1045-53.
8. Demmer RT, Squillaro A, Papapanou PN, Rosenbaum M, Friedewald WT, Jacobs DR, Jr., et al. Periodontal infection, systemic inflammation, and insulin resistance: results from the continuous National Health and Nutrition Examination Survey (NHANES) 1999-2004. *Diabetes care*. 2012;35(11):2235-42.
9. Abdullah B, Shari NF, Faisal GG, Radeef AS, Suhaila MA. Assessment of Illness Perception of Diabetic Patients with Periodontitis. *Journal of International Dental and Medical Research*. 2017;10(1):100-7.

10. Pradhan S, Goel K. Interrelationship between diabetes and periodontitis: a review. *JNMA; journal of the Nepal Medical Association*. 2011;51(183):144-53.
11. Mohd-Dom TN, Aljunid SM, Manaf RA, Muttalib KA, Asari ASM. Periodontal status and provision of periodontal services in Malaysia: are we meeting population needs? *BMC Health Services Research*. 2012;12(Suppl 1):8.
12. Lalla E, Papapanou PN. Diabetes mellitus and periodontitis: a tale of two common interrelated diseases. *Nature reviews Endocrinology*. 2011;7(12):738-48.
13. Madianos PN, Koromantzios PA. An update of the evidence on the potential impact of periodontal therapy on diabetes outcomes. *Journal of clinical periodontology*. 2018;45(2):188-95.
14. Moeintaghavi A, Arab HR, Bozorgnia Y, Kianoush K, Alizadeh M. Non-surgical periodontal therapy affects metabolic control in diabetics: a randomized controlled clinical trial. *Australian dental journal*. 2012;57(1):31-7.
15. Agarwal G, Kaczorowski J, Hanna S. Care for Patients with Type 2 Diabetes in a Random Sample of Community Family Practices in Ontario, Canada. *International Journal of Family Medicine*. 2012;2012:7.
16. 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):777-9.
17. Sonnenschein SK, Meyle J. Local inflammatory reactions in patients with diabetes and periodontitis. *Periodontology 2000*. 2015;69(1):221-54.
18. Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ. Prevalence of periodontitis in adults in the United States: 2009 and 2010. *Journal of dental research*. 2012;91(10):914-20.
19. Preshaw PM, Alba AL, Herrera D, Jepsen S, Konstantinidis A, Makrilakis K, et al. Periodontitis and diabetes: a two-way relationship. *Diabetologia*. 2012;55(1):21-31.
20. Al-Nozha MM, Al-Maatouq MA, Al-Mazrou YY, Al-Harhi SS, Arafah MR, Khalil MZ, et al. Diabetes mellitus in Saudi Arabia. *Saudi medical journal*. 2004;25(11):1603-10.
21. Hassan A, Alam MK. Systemic conditions in patients with periodontal disease. *International Medical Journal*. 2013;20(3):363-6.
22. Ziukaite L, Slot D, Cobb C, Coucke W, Van der Weijden G. Prevalence of diabetes among patients diagnosed with periodontitis: A retrospective cross-sectional study. *International Journal of Dental Hygiene*. 2018;16(2):305-11.
23. Stenson M. Class, race, and colonialism in West Malaysia. UBC Press; 2011:73-85.
24. Ramasundrum V, Mohd Hussin Z, Tan CT. Public awareness, attitudes and understanding towards epilepsy in Kelantan, Malaysia. *Neurol J Southeast Asia*. 2000;5:55-60.
25. Alasqah M, Mokeem S, Alrahlah A, Al-Hamoudi N, Abduljabbar T, Akram Z, et al. Periodontal parameters in prediabetes, type 2 diabetes mellitus, and non-diabetic patients. *Braz Oral Res*. 2018;32:81.
26. Bunjaku V, Popovska M, Grcev A, Mrasori S, Kameri A, Silamniku Z, Dragidella F. Non-surgical Periodontal Treatment and Low Level Laser Therapy (LLLT) Outcomes for Patients Suffering from Type 2 Diabetes Mellitus, Obesity and Chronic Periodontitis. *Journal of International Dental and Medical Research*. 2017;10(2):214-21.
27. Bullon P, Newman HN, Battino M. Obesity, diabetes mellitus, atherosclerosis and chronic periodontitis: a shared pathology via oxidative stress and mitochondrial dysfunction? *Periodontology 2000*. 2014;64(1):139-53.
28. Chang P-C, Chien L-Y, Yeo JF, Wang Y-P, Chung M-C, Chong LY, et al. Progression of Periodontal Destruction and the Roles of Advanced Glycation End Products in Experimental Diabetes. *Journal of periodontology*. 2013;84(3):379-88.
29. Promsudthi A, Poomsawat S, Limsricharoen W. The role of Toll-like receptor 2 and 4 in gingival tissues of chronic periodontitis subjects with type 2 diabetes. *Journal of periodontal research*. 2014;49(3):346-54.
30. Nassar H, Kantarci A, Van Dyke TE. Diabetic periodontitis: a model for activated innate immunity and impaired resolution of inflammation. *Periodontology 2000*. 2007;43:233-44.
31. Saremi A, Nelson RG, Tulloch-Reid M, Hanson RL, Sievers ML, Taylor GW, et al. Periodontal disease and mortality in type 2 diabetes. *Diabetes care*. 2005;28(1):27-32.
32. Kiran M, Arpak N, Unsal E, Erdogan MF. The effect of improved periodontal health on metabolic control in type 2 diabetes mellitus. *Journal of clinical periodontology*. 2005;32(3):266-72.