A Study of Periodontal Disease and Diabetes Mellitus in Abuja Nigeria.

[1*]IKIMI Nathan U. [2]ONIGBINDE OlubunmiO. [2]SORUNKE ModupeoreE.
[3]ADETOYE Johnson O. [1]ENEBONG Deborah J.

[1]Dental and Maxillofacial Department, State House Medical Centre, State House, Abuja FCT, Nigeria
[2]Department of Preventive Dentistry, Faculty of Dentistry, Lagos State University Teaching Hospital, Lagos Nigeria.
[3]Dental Clinic, Garki General Hospital, Garki Abuja, FCT Nigeria.
Corresponding author: Dr. Nathan U.Ikimi;

Abstract
Background:*Periodontal disease is essentially a combination of gingivitis, which is aninflammatory condition of the gingiva and periodontitis, which refers to the destruction of the periodontal ligament, alveolar bone and cementum, a destructive process that could lead to tooth loss. Studies confirm that diabetics are three times more likely to develop periodontal disease than the control.*
Objectives:*To examine the oral hygiene status of diabetic patients and evaluate the extent of periodontal disease in them.*
Methodology:*This is an observational case-controlled study among diabetic subjects with type 1 and 2 diabetes mellitus attending a diabetic clinic inAbuja, Nigeria. Social demographic characteristics were recorded; presence of plaque and calculus were noted down; pocket depths and clinical attachment loss were measured and recorded in a periodontal chart. Data were analyzed descriptively and comparatively using SPSS version 20.*
Results:*Mean plaque index in diabetic patients was 2.01±0.09 and 1.64±0.07 in non-diabetics; mean pocket depth in diabetic patients was 3.4±0.9 and 2.5±0.7 in non-diabetics; mean clinical attachment loss in diabetic patients was 3.1±0.7 and 2.3±0.6 in non-diabetics.*
Discussion:*The results in this study shows that the oral hygiene status and periodontal disease is more pronounced in diabetics than non-diabetics and this is indicated by the increase in plaque index, pocket depths and clinical attached loss in this study group; this is in agreement with results from other studies in Nigeria and other parts of the world.*
Conclusion: *Diabetes mellitus, a systemic condition, contributes negatively to periodontal disease and this could lead to tooth loss.*
Key Words: *Calculi; Dental Plaque Index; Diabetics;Gingivitis; Periodontitis.*

I.   Introduction

Periodontal disease consist of gingivitis which could be described as aninflammatory disorder of the gingivaand periodontitis which is the destruction of the periodontal attachment apparatus.[1]Periodontal disease is initiated by an overgrowth of a group of microorganismthat are present in bacterial plaque which according toSocransky et al[2]are made up of three microaerophilic species (*Aggregatibacter*[previously called *Actinobacillucus*]*actinomycetemcomitans, Campylobacter rectus* and *Eikenellacorrodens)* and seven anaerobic species (*Porphyromonasgingivalis, Bacteroidesforsythus, Treponema denticola, prevotella intermedia,Fusobacterium nucleatum, Eubacterium and spirochetes).*These bacteria existing in plaque release antigenic substances and these stimulates both cell-mediated and humoral responses which while designed to be protective, also cause tissue damage, destroy matrix components, fibroblast, affect production of collagenand damagesthe junctional epithelium[3,4].Thus, if this inflammatory response and the immunologically mediated pathway triggered by the presence of dental plaque continue uninterrupted, it would lead to chronic periodontitis[5].

Studies have reported that a higher incidence of periodontal disease exist among diabetics than inhealthy controls[6, 7]; besides,when diabetic subjects with type 1 and type 2 diabetes mellitus were analysed together without distinction of diabetes typesand compared with the control, young and adult subjects with diabetes mellitusdisplayeda greater incidenceof periodontal disease[8-10].Furthermore, the elevated blood sugar

---

level in diabetics leads to delayed wound healing, inability to respond adequately to attack from infections, bone loss and eventually in most cases, tooth loss [11,12].It has also been reported that susceptibility to periodontitis is increased by approximately threefold in people with diabetes mellitus[13]andthat an undesirable bi-directional relationship exist between periodontal disease and diabetes mellitus such that a poor periodontal status has a negative effect in achieving glyceamic control and a hyperglyceamic environment increases susceptibility to periodontal diseases[7,13].

In Nigeria, like other parts of the world,a higher prevalence of periodontal disease has been reported among diabetics [14-17] and anestimated 40-80 person with diabetes mellitus in 2,000 adult populations visiting a dental practice in Nigeria has periodontal disease, while about half are unaware of their diabetic condition [17].Therefore, the higher prevalence of periodontal disease in diabetic patients than non-diabeticsand the lack of awareness among the general population of the negative effects of diabetes mellitus on periodontal tissuemake it imperative to conduct a study in diabetic patients to create awareness among diabeticsin particular and the general population of their periodontal status and the susceptibilityof diabetic patients to periodontal disease with its accompanying consequences [17]. Also, to create awareness among medical doctors, health workers and other stake-holders of the harmful relationship between diabetes mellitus and periodontal diseases; and to provide information to help health professionals understand the treatment needs of diabetic patients with periodontal diseases and render adequate, efficient and timely health services.

Objectives
1. To evaluate the relationship of diabetes mellitus and oral hygiene status as shown by the presence of plaque and calculus.
2. To investigate the relationship between diabetes mellitus and periodontal diseases as indicated by increased pocket depths and clinical attachment loss.

II. Methodology

This study consists of 201 diabetic patients and an equal number of controls in the diabetic clinic of Garki General Hospital, in the federal capital territory, Abuja Nigeria. Ethical clearance was obtained from the Federal Capital Territory (FCT) Health, research and ethical committeeto carry out the study. This is an observational case-controlled study with diabetic patients aged 16 years and above with a history of Type 1 or 2 diabetes mellitus (DM) of not less than 6 months; an equal number of non-diabetic patients, matched for age and sex were recruited as control. In both groups, only subjects having 10 or more teeth present were included.Excluded were patients with systemic conditions that would give false readings because of the effects of these conditions on the gingiva e.g. lupus, blood diseases and HIV/AIDS. Additionally, confirmed smokers, diabetics who have been on antibiotics or anti-inflammatory medications in the previous three months and pregnant women were excluded from this study. Sociodemographic data and medical History which included types of diabetes mellitus, medical conditions like hypertension, kidney disease, drug history, were all collected from patient'scase file.

Using the World Health Organization (WHO) graduated probe, with patient seated in upright position under an overhead fluorescent light,the jaws were divided into sextants and index teeth (11, 16, 17, 26, 27, 31, 36, 37, 46 and 47) were examined in each sextant by a single examiner after reaching a substantial agreement of 0.70-0.80 consistency; in the absence of index teeth, it was agreed that the only standing tooth should also be examined and recorded in a Community Periodontal Index of Treatment Need (CPITN) chart. The presence of Plaque and Calculus, Pocket depths, Clinical attachment loss and number of Teeth present or absent were recorded [16, 17].

Data was entered and analyzed using Statistical Package for Social Science (SPSS) 20(IBM Corp., Armonk, NY, USA). Continuous variables were presented as Mean, while categorical variables were presented using Frequency and Percentage. Mean comparison of Plaque Index, Pocket depth, Clinical attachment Loss and Missing teeth based on blood sugar level was carried out using student's independent t – test. Probability value (p) ≤ 0.05 (less than 0.05) was used and considered statistically significant.

III. Results

Among the 201 diabetic patients and non-diabetic patients in this study, 99 were diabetic males (49.3%) and 102 (50.7%) were diabetic females while the non-diabetic were 96 males (47.8%) and 105 (52.2%) females; with age ranging from 35-74 years and a mean age of 49.6 ± 10.3 for diabetics and 48.4 ± 10.5 for non-diabetics. Age at onset of diabetes mellitus was compared. **Details of comparison in Figure 1.**
Mean plaque index for diabetic and non-diabetic patients was compared and the difference isstatistically significant. **Details in Table 1.**

Mean calculus index in diabetic and non-diabetic patients was also compared and the difference is statistically significant. **Detailsin Table 2.**

Mean pocket depth for diabetic patients was compared with non-diabetic patients and the difference was found to be statistically significant. **Details in Table 3**.

The mean clinical attachment loss in diabetic and non-diabetic patients was both compared and the difference is statistically significant with P-value0.002. **Details in Table 4.**

IV. Discussion

Oral health is affected by a number of factors including metabolic disorders such as diabetes mellitus (DM)[20].The mean age for diabetic patients and non-diabetic patients in this study indicates an early on-set of diabetes mellitus among respondents (Fig 1).The genetic make-up of Negros might be a contributory factor to early onset of DM and this is supported by observations from Sub-Saharan African countries where age range of 45-64 years has been reported to be diabetic [16, 21].Diabetes mellitus results in susceptibility to periodontal disease and once gingivitis becomes established, there is insulin resistance and prolong hyperglycaemia[22].Thus, an early on-set of diabetes mellitus implies an early on-set of periodontal disease too. The prolong hyperglycaemia in diabetic state leads to the formation of Advanced Glycation End-products (AGE) and this in turn binds to a specific cellular receptor known as the Receptor for AGE (RAGE)[23]. The binding of AGE and RAGE causes a sequence of pro-inflammatory events that are self-sustaining and AGE present in the gingiva of diabetic may be associated with a state of enhanced oxidative stress and this might be thepossible mechanism for accelerated periodontal tissue injury [23]. The accumulation of AGE causes cross linking of collagen molecules to each other and to circulating proteins; this promotes plaqueformation and accumulation withbasement membrane thickening [24].Additionally, the bi-directional relationship that exist between diabetes mellitus and periodontal disease comes into play such that the thickening of the capillary basement membrane in hyperglycaemic surroundings further deteriorate oxygen diffusion, reduce metabolic waste removal and decreases the speed of polymorphonulcear leucocytes' migration as well as diffusion of antibodies and thus, the destructive process of periodontal disease continues uninterrupted in the diabetic state[25].

Dental plaque is the principal aetiological factor that initiates gingivitis; it has been reported that a mouth left for 10 to 20 days to accumulate plaque without any form of cleaning will result in periodontal disease [1].The mean plaque and mean calculus Index score recorded in this study for diabetic patients (Table 1 and 2) is similar to results of Rajhans et al who reported a mean plaque and calculus index scores of 1.22 ± 0.55 and 1.27 ± 0.60respectively for diabetic patients and non-diabetics[26].A higher pocket depth for diabetic patients as compared to non-diabetics (Table 3) was recorded in this study and this is in agreement with previous study in Nigeria [12]. However, this is different from theresearch in Columbia where a lower pocket depth of 2.62mm was reported for diabetics [27].Periodontal attachment loss has been found to occur more often in moderate and poorly controlled diabetic subjects than those with good control [28]. A statistically significant difference (p-value 0.02) in the total mean clinical loss of attachment between diabetics and non-diabeticswas observed in this study (Table 4) and thisis consistent with the results of Liu et al[28]who gave an overall average attachment loss of 3.03mm with only 6% of the sites having a clinical attachment loss of 7mm or more in diabetic patients. AlsoChen [29], in the United States of America reported that an average loss of attachment 3 to 6mm was present in 24.2% of the total 1500 patients', while more than 6mm were present in 15.4% patients.The reduced synthesis of collagen by fibroblasts, glycosylation of existing collagen at wound margins, defective remodeling of collagen resulting in poorly cross-linked collagen and the speedy degradation of newly synthesized collagen by the enzyme collagenase might all be contributoryfactors for poorer gingival health in diabetics, increased attachment loss and the resultant tooth loss[11, 30].

Whenpatients are aware of the oral complications of diabetes mellitus and are able to maintain a well-controlled blood sugar level and regular dental visitations to maintain periodontal health, they do not lose more teeth than healthy individualsirrespective of age;also the associated risk of periodontitis in diabetics would be under control[14, 17, 29].

V. Conclusion

The Mean Plaque Index score and Calculus Index score were significantly higher in the diabetic group suggesting a correlation between poor oral hygiene and diabetes mellitus.Also, mean pocket depth and clinical attachment loss were significantly higher in diabetics and this implies that periodontal disease has a direct relationship with diabetes mellitus.

Within the limitations of this study, periodontal disease which couldlead to tooth loss isdirectly connected to diabetes mellitus. Thus, it is important to create awareness among this group of patients and the general public by carrying out regular oral health education. The diabetic patient usually visits the medical doctor more

than the dentist; therefore, the medical doctor needs to have adequate knowledge of the relationship of periodontal disease and diabetes mellitus so as to carry out prompt dental referrals.

It is our recommendation that further studies should be done to assess the knowledge of the medical doctor on the role of the dental surgeon in the management of diabetic patients and his willingness to collaborate with the dentist and fill gaps in knowledge.

Acknowledgement

Conflict of interest
There is no conflict of interest to declare

References:

[1]   Kinane DF. Causation and pathogenesis of periodontal disease. *Periodontol* 2000. 2001; 25: 8–20
[2]   Socransky SS, Haffajee AD. Dental biofilms: difficult therapeutic targets. *Periodontol* 2000. 2002; 28: 12–55
[3]   Kinane DF, Berglundh T, Lindhe J. Host-Parasite Interactions in Periodontal Disease In: Lindhe J, Karring T, Lang NP, *ClinicalPeriodontology and Implant Dentistry*, (4th.ed) Manhattan, Wiley Publishing, 2003; 150-175.
[4]   Joshi VM, Vandana KL. The detection of eight putative periodontal pathogens in adult and rapidly progressive periodontitis patients: An institutional study. *Indian J Dent Res*. 2007; 18 (3):6-10
[5]   Parameters of care-American academy of Periodontology. *Journal ofPeriodontology*. 1996; 1 (1): x-xiii. doi:10.1902/annals.1996.1.1.x.
[6]   Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes. N Engl*JMed*. 1992; 326:242–50.
[7]   Teng YT, Taylor GW, Scannapieco F, Kinane DF, Curtis M, et al. Periodontal health and systemic disorders. *J Can DentAssoc*. 2002; 68:188–92.
[8]   Lee W, Aitken S, Sodek J, McCulloch CA. Evidence of a direct relationship between neutrophil collagenase activity and periodontal tissue destruction in vivo: role of active enzyme in human periodontitis. *JPeriodontal Res*. 1995;
[9]   Jansson H, Lindholm E, Lindh C, Groop L, Brattahall G. Type 2 diabetes and risk for periodontal disease: a role for dental health awareness. *J ClinPeriodontal*. 2006; 33: 408–414.
[10]   Demmer RT, Jacobs Jr DR, Desvarieux M. Periodontal disease and incident type 2 diabetes: results from the First National Health and Nutrition examination Survey and its epidemiologic follow-up study. *Diabetes Care*. 2008;31 :1373–1379
[11]   Oliver RC, Tervonen T. Periodontitis and tooth loss: Comparing diabetics with the overall population. *J Am Dent Assoc*. 1993 Dec; 124(12):71-6
[12]   Ogunbodede EO, Fatusi OA, Akintomide A, Kolawole K, Ajayi A. Oral health status in a population of Nigerian diabetics. *J Contemp Dent Pract* 2005; 6(4):75-84
[13]   Preshaw PM, Alba AL, Herrera D, Jepsen S, Konstantinidis A, et al. Periodontitis and diabetes: a two-way relationship. *Diabetologia*. 2012 Jan; 55(1): 21–31.
[14]   Taiwo JO. Oral health education needs of diabetic patients in Ibadan. *Afr JMed and Med Sci*. 2000; 29(3-4):269-74
[15]   Lamster IB, DePaola DP, Oppermann RV, Papapanou PN, Wilder RS. The relationship between oral Health and Diabetes mellitus. *JADA* 2008; 139(10): 1389-1397.
[16]   Chinenye S, Uloko AE, Ofoegbu EN, Fasanmade OA, Fasanmade AA, et al. Profile of Nigerians with Diabetes mellitus- Diabcare Nigerian study group (2008); Results of a multicentre study. *Indian J EndocrMetab* 2012; 16(4):558-64
[17]   Azodo CC. Current trends in the management of diabetes mellitus: The Dentist's Perspective. *Journal of Postgraduate Medicine* 2009; 11(1):113-129.
[18]   Silvestre FJ, Miralles L, Llambes F, Bautista D, Solá-Izquierdo E, et al. Type 1 diabetes mellitus and periodontal disease: Relationship to different clinical variables. *Med Oral Path Oral Cir Bucal*. 2009; 14 (4):E175-9.
[19]   O'Leary TJ, Drake RB, Naylor JE. The plaque control record. *JPeriodontol*. 1972; 43(7):38
[20]   Khalid ABA. Diabetes Mellitus and its Oral Complications: A Brief Review. *Pakistan Oral & Dent*. 2006 Jan; 1(1): 145-156.
[21]   King H, Aubert RE, Herman WH. Global burden of Diabetes, 1995-2025. *Diabetic Care* 1998; 21 (9): 1414-1431
[22]   Zambon JJ, Reynolds H, Fisher JG, Shlossman M, Dunford R, et al. Microbiological and immunological studies of adult periodontitis in patients with non-insulin dependent diabetes mellitus. J Periodontol. 1988; 59: 23–31.
[23]   Lalla E, Lamster IB, Feit M, Huang L, Spessot A, et al. Blockage of RAGE suppresses periodontitis-associated bone loss in diabetic mice. *JClin Invest* 2000; 105(3):1117-1124.
[24]   Cianciola LJ, Park BH, Bruck E, Mosovich L, Genco RJ. Prevalence of periodontal disease in insulin-dependent diabetes mellitus (juvenile diabetes) *J Am Dent Assoc*. 1982; 104:653–660.
[25]   Ulrich P, Cerami A. Protein glycation, diabetes and aging. *Recent ProgHorm Res*. 2001; 56(2):1-21.
[26]   Rajhans NS, Kohad RM, Chaudhari VG, Mhaske NH. A clinical study of the relationship between diabetes mellitus and periodontal disease. *Journal of Indian Society of Periodontology*, 2011; 15 (4), 388–392. http://doi.org/10.4103/0972-124X.92576.
[27]   Manson JD, Eley BM. The effect of systemic factors in periodontal tissues. *Outline of Periodontics*, (4th.ed) Scotland. Wright Publishing, 2000; 90-106.

[28]   Liu R, Bal HS, Desta T, Krothapalli N, Alyassi M, Luan Q, *et al*. Diabetes Enhances Periodontal Bone Loss through Enhanced Resorption and Diminished Bone Formation.*J Dent Res*. June 2006; 85(6); 510-514.
[29]   Chen I. The Surgeon General's report on oral health: Implications for research and education. *NY State Dent J* 2000; 66(9): 38-42.
[30]   Piwowar A, Knapik-Kordecka M, Warwas M. Concentration of leukocyte elastase in plasma and polymorphonuclear neutrophil extracts in type 2 diabetes. *ClinChem Lab Med*. 2000; 38(12):1257-61

FIGURES AND TABLES
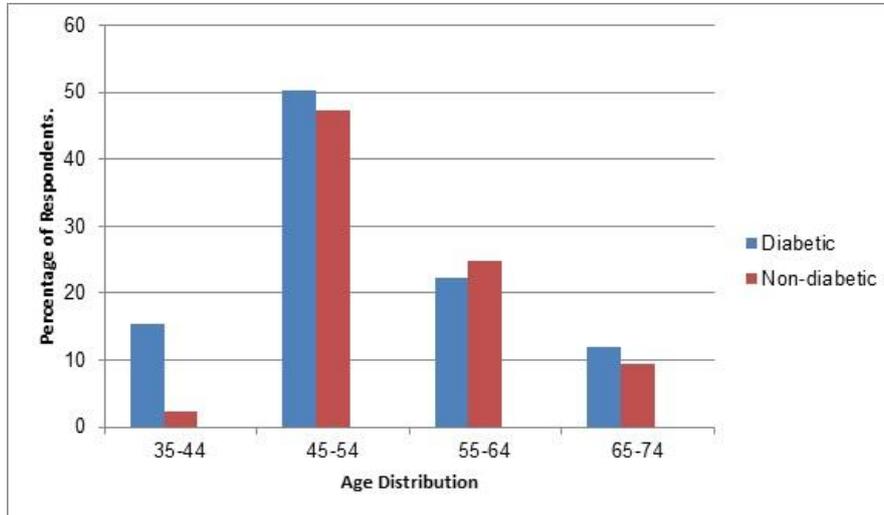
Figure 1: Comparison of Diabetic and Non-diabetic status according to age.

Table 1: Mean Plaque Index of diabetic and non-diabetic patients

	Mean	Standard deviation	Mean Diff.	P-value
Diabetic patients (201)	2.01	0.09	0.37¶	0.031*
Non diabetic patients (201)	1.64	0.07		
Total	1.83	0.08		

*P-value 0.031(Statistically significant)

¶ Diabetic patients had a significantly higher plaque level .

Table 2: Mean Calculus Index of diabetic and non-diabetic patients

	Mean	Standard deviation	Mean diff.	P-value
Diabetic patients (201)	2.06	0.11	0.40§	0.035*
Non diabetic patients (201)	1.66	0.08		
Total	1.86	0.10		

* P-value 0.035 (Statistically Significant)

§= The mean value in Calculus Index was significantly higher in the diabetic patients.

Table 3: Mean Pocket Depth of diabetic patients (PD) according to age-groups

	Pocket Depth (mm)¶		Mean difference (mm)	p-value
	Diabetic	**Non-diabetic**		
Age group (years)				
35-44	3.0±0.9	2.1±0.6	0.9	0.000*
45-54	3.3±0.7	2.4±0.9	0.9	0.000*
55-64	3.5±0.7	2.7±0.8	0.8	0.000*
65-74	3.8±1.1	2.7±1.0	1.1	0.005*
Overall	3.4±0.9	2.5±0.7	1.2	0.000*

*Statistically Significant @ P-value <0.005

¶ = Both diabetic and non-diabetic patients had increased pocket depths which increased with age but diabetic patients had a significantly higher pocket increase than non-diabetic patients.

Table 4: Mean Clinical Attachment Loss (CAL) in Diabetic and Non-patients.

	CAL (mm)		Mean difference (mm)	p-value
	Diabetic	**Non-diabetic**		
Age group (years)				
35-44	2.5±0.5	2.0±0.5	0.5	0.045*
45-54	2.8±0.6	2.2±0.5	0.6	0.010*
55-64	3.2±0.8	2.4±0.7	0.8	0.000*
65-74	3.5±1.0	2.5±0.9	1.0	0.005*
Overall[§]	3.1±0.7	2.3±0.6	0.8	0.002*

*Statistically Significant @P-value <0.005

§= There was a significant increase in clinical attachment loss which was more in diabetic patients than non-diabetic patients.

.