Periodontal status and glycaemic control among type 2 diabetic patients- a comparable study between 2 teaching hospitals in 2 geographical zones in Nigeria

Abstract

Background: There is clinical evidence that periodontitis and diabetes mellitus (DM) are interconnected. Thus, periodontitis can promote systemic chronic inflammation that can exacerbate type 2 diabetes mellitus.
Methodology: Self-administered questionnaire was used to collect data. Glycaemic control and periodontal status were evaluated by glycated Hb values and CPI respectively. Data was analysed using the Statistical Package for Social Sciences version 20.0 (IBM SPSS Statistics Armonk New York). Association of glycaemic index with periodontal status was explored by the $\chi 2$ test. Statistical significance was set at $P < 0.05$.
Results: One hundred and eighty-five participants with type 2 diabetes were recruited with a female predominance in both centers. Majority of participants were above the fifth decade. The mean duration of diabetes was 8.97±7.14. 73.3 % of participants in UPTH and 84.7% in LASUTH were out of (p=0.001). One-fourth of participants in LASUTH and 14.2% of participants from UPTH had poor oral hygiene. Twice participants in LASUTH had good glycaemic control compared to those in UPTH (p=0.001). The periodontal status of majority of the participants in the two centers was between CPI score 2 and 4 (p=0.02). The association between good glycaemic control and gender and between good glycaemic control and age were statistically significant (p=0.014; p=0.001).
Conclusion: The periodontal status of participants did not worsen with poor glycaemic control. However, education was significantly associated with extent of control. Periodontal care needs to be incorporated into the management of the diabetics in order to improve their quality of life.

Key words: Demographics, DM, Glycaemic control, Periodontal status.

Introduction

Periodontal disease (PD) is a chronic infectious disease that is caused by gram-negative microorganisms found in dental plaque.[1] These microorganisms cause local inflammation that progresses from gingival inflammation to alveolar bone destruction and loss of periodontal attachment[2.] They can also induce initial infiltrate of inflammatory cells like lymphocytes, macrophages and polymorphonuclear leokocytes.[2]

Microbial components such as lipopolysaccharide (LPS) activate macrophages to synthesize and secrete a variety of pro-inflammatory molecules like tumor necrosis factor-α (TNF-α),

interleukin-1 (IL-1) and prostaglandin E2 (PGE2).[2] Furthermore, they produce toxins that activate T lymphocytes to produce IL-1 and lymphotoxin (LT) which has properties that are similar to those of TNF-α. These cytokines show potent pro-inflammatory and catabolic activities that play important roles in periodontal tissue destruction by collagenolytic enzymes such as metalloproteinases (MMPs).[2] These collagenolytic enzymes are activated by reactive oxygen species and elevate the levels of interstitial collagenase in inflamed gingival tissue.[3] This results in attachment loss that deepens the gingival sulcus and creates a periodontal pocket that contains millions of bacterial that further worsens the destruction.[4-7]

Recent studies have suggested that the effect of PD might not only be limited to the oral cavity but can progress to the body system since the human body acts as a unity and biologic processes in one part of the body can affect other body areas.[9-11]

Diabetes mellitus (DM) on the other hand is a chronic disease that affect all individuals of all ages. If poorly controlled, it can result in hyperglycaemia that can lead to a lot of complications in other organs of the body such as the heart, kidney and eyes.[12]

Elevated glucose levels induce non-enzymatic glycation and oxidation of proteins (collagen, and lipids) resulting in the accumulation of advanced glycation end products (AGEs) in diabetic tissues.[13] AGEs interact with their receptors found on cell surfaces called the receptor for AGE (RAGE) resulting in various pathological changes. The AGE-RAGE interaction in the macrophages causes increased release of pro-inflammatory cytokines like Tumour Necrosis Factor alpha (TNF-α) and Interleukin -1 beta (IL-1β).[14,15]

Diabetes is one of the currently recognized two true risk factors (the other is smoking) for periodontal disease that have been incorporated into the grading component of the new classification of periodontal diseases by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP).[16-17] Furthermore, a bidirectional

relationship has been established between PD and DM by several studies suggesting that periodontitis may be a complication of diabetes and can have an adverse effect on glycaemic control by raising blood glucose levels.[18-20]

This study therefore assessed the periodontal status and glycaemic control of diabetic patients.

Methodology

A descriptive cross-sectional study conducted between July and September 2019 at two outpatient diabetic clinics of two Teaching Hospital in two zones in Nigeria (University of Port Harcourt Teaching Hospital (UPTH) in South-South zone of Nigeria and Lagos State University Teaching Hospital (LASUTH) in Lagos, South West zone in Nigeria). Ethical approval was obtained from the ethics committees of the hospitals.

The inclusion criteria were subjects with type 2 diabetes aged 18years and above diagnosed with diabetes at least 1 year before the study, gave consent to be part of the study and are permanent residents in the study locations. Those with physical or mental challenges, those on xerostomia causing drugs and conditions (anti-hypertensives, anti-depressants, diuretics and radiation therapy), those with chronic systemic diseases such as asthma and epilepsy, and those who smoke or consume alcohol were excluded from the study. One hundred and eighty-five subjects were recruited for this study; 105 from UPTH and 80 from LASUTH

Self-administered questionnaires were used to collect data on demographics. Oral cleanliness was assessed using Simplified Oral hygiene index by Greene and Vermillion and periodontal status was assessed using CPI index. Values of glycated heamoglobin levels in the blood (HbA1c) values were retrieved from patients records and confirmed with laboratory reports. In accordance with the American Diabetes Association (ADA) guidelines, HbA1c < 7% was

taken to indicate good glycemic control, while HbA1c ≥ 7% indicated poor glycemic control.[21]

Simplified Oral Hygiene Index (OHI-S). [22]

The OHI-S is a composite index that scores debris and calculus deposition on selected teeth. It was developed by (Greene and Vermillion in 1964. It is expressed as the sum of the mean debris index (DI-S) and calculus index (CI-S) of the examined teeth. The OHI-S is interpreted as follows: Score 1 (good oral hygiene) = 0.0 – 1.2, Score 2 (fair oral hygiene) =1.3 – 3.0, Score 3 (poor oral hygiene) = 3.1 – 6.0.

Periodontal status was measured using a Community Periodontal Index according to the World Health Organization (WHO) basic methods of oral health surveys.[23] The criteria for the community periodontal index (CPI) are as follows:

Code-0- Coloured band of the probe remains completely visible in the deepest sulcus of the sextant-healthy.

Code-1- Coloured band of the probe remains completely visible in the deepest sulcus of the sextant, some bleeding after gentle probing.

Code-2- Coloured band of the probe still completely visible, but there is bleeding on probing, supragingival or subgingival calculus and/or defective margins.

Code-3- The coloured band is partially submerged. Pocket 4-5 mm deep.

Code-4- The coloured band completely disappears in the pocket, indicating a depth greater than 5.5 mm and a loss of attachment of 3mm or more.

Data was analysed using the Statistical Package for Social Sciences version 20.0 (IBM SPSS Statistics Armonk New York). Continuous variables were described with mean and standard

deviation while nominal variables were described with frequencies. Association of glycaemic index with periodontal status was explored by the $\chi 2$ test. Statistical significance was set at $P < 0.05$.

Results

Study population consisted of one hundred and eighty patients with type 2 diabetes (105 from UPTH and 80 from LASUTH). Mean age was 57.11±13.45 year and mean DM duration was 8.97±7.14years. There was a female predominance in both centers; UPTH (F:M of 1.76:1), LASUTH (F:M of 2.64:1). Table 1a.

Table 1b shows the mean parameters of the study participants

Figure 1 shows the clustered count bar of Hb1Ac by CPI scores. The CPI score among the two groups was majorly 2

Participants glycaemic control and periodontal status showed that twice participants in LASUTH had glycaemic control than those in UPTH (51.3% in LASUCOM vs 26.7 % of participants in UPTH had good glycaemic control). Statistical analysis showed this to be significant (p= 0.001). One-fourth of participants in LASUTH had poor oral hygiene, while 14.2% of participants from UPTH had poor oral hygiene. More participants in UPTH had poor glycaemic control (73.3%). The periodontal status of majority of the participants in the two centers were CPI score between 2 and 4. Statistical analysis showed a statistical significance (p=0.02). Table 2

The association between participants' glycaemic control and periodontal status showed that the oral hygiene status of about two-third of participants with or without glycaemic control in both centers was fair and the periodontal status showed a CPITN score of between 2 and 3 majorly. The CPITN score of participants with good glycaemic control showed a statistical significance (p= 0.03). Table 3.

Association between gender and good glycaemic control was statistically significant (p=0.014). Likewise, the association between age group and good glycaemic control (p=0.001) and poor glycaemic control (0.02). Table 4a.

The associations between glycaemic control and occupation (p=0.10; p= 0.16) and between glycaemic control and education (p=0.10, p=0.11) showed no statistical significance. Table 4b.

Discussion

The bidirectional relationship between PD and DM is well documented, though it is still unclear if it is a causal one or due to their common risk factors.[17,24-27] The interaction could be because diabetes may directly influence the oral microbes leading to dysbiosis or the common inflammatory pathways as inflammatory markers have been reported to be elevated in these two comorbidities. As such, studies have reported a positive effect on glycated haemoglobin levels in the blood (HbA1c) when periodontal therapy is done as it reduces the periodontal inflammatory load.[11,17,25,26] This study reported a good glycaemic control among a quarter and half of participants in UPTH and LASUTH respectively.

The mean age of the study population was Mean age = 57.11±13.45 years and comparable to other studies done among diabetes.[28] More female predominance in this study comparable to some other studies.[28-31]

The glycated (glycosylated) haemoglobin assay (HbA1c) is an indicator of blood glucose levels and therefore a possible prognostic marker that gained widespread acceptance in the 1980s as the laboratory test of choice and is still widely used. [32] It can be measured using a number of differing methods with several internationally adopted standards such as the Diabetes Control and Complications Trial (DCCT) or the International Federation of Clinical Chemistry (IFCC) standard.[33,34] The latter consistently gives lower values (non-diabetic reference range is about 3% to 5% IFCC and 4% to 6% DCCT, with good control in diabetic groups as 5% IFCC and 7% DCCT. The American Diabetes Association (ADA) guidelines, however endorsed that HbA1c < 7% indicates a good glycemic control, while HbA1c ≥ 7% indicates a poor glycemic control.[21]

The participants in this study based on Glycated hemoglobin were classified into three groups. These are well controlled (Glycated hemoglobin <7.0%), moderately controlled

(Glycated hemoglobin 7.0-8.0%) and poorly controlled (Glycated hemoglobin >8.0%). Our study showed no association between duration of diabetes, glycated haemoglobin and periodontal disease severity similar to the findings in other studies.[35,36] This contrasted with other studies that reported association between higher glycated haemoglobin and severe periodontitis.[37-42]

In our study, the number of participants with poor glycaemic control was more than those with good glycaemic control. This correlated with the findings of other studies.[43-45]

Simplified oral hygiene index (OHI-S) measures the cleanliness of the mouth and can be used to classify individuals into good, fair or poor oral hygiene. Majority of our participants had fair oral hygiene. This compares to the study done among T2DM in Lucknow, India that reported that 68.8% of their participants had fair oral hygiene.

The periodontal status of the participants in this study did not worsen with poor glycaemic control. This compares to the study done among diabetics in Harvard Medical School, Boston.[46] The means of participants parameters examined in this study are comparable to those reported by other studies.[46,47]

Community periodontal index is used to detect periodontal diseases. It scores the presence and absence of supra and sub gingival plaque and calculus as well as pocket depth correlating it with the extent and severity of the disease. Two-fifth and about half of those with well controlled and poorly controlled DM respectively had CPI score 2. However, a third of all participants irrespective of glycaemic control had CPI scores 3 and 4. This contrast with another study that recorded that two-fifth of their participants with poorly controlled DM had CPI code 3 and three-fifth had CPI code 4.

Limitation

This study did not assess gingival recession.

Conclusion

The periodontal status of participants did not increase with poor glycaemic index. Participants had CPI scores 0-4 irrespective of Hb1Ac. However, the adverse effect of periodontal infections on diabetes mellitus is potentially explained by resulting increase in systemic inflammation which can contribute to insulin resistance.

Recommendation

There is the need to increase patients' awareness of the link between diabetes mellitus and periodontitis and encourage collaboration between medical and dental professionals for the management of affected individuals.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

References

1. Negrato CA, Tarzia O, Jovanovič L, Chinellato LE. Periodontal disease and diabetes mellitus. J Appl Oral Sci. 2013;21(1):1-12.
2. Sorsa T, Ingman T, Suomalainen K, Haapasalo M, Konttinen YT, Lindy O, et al. Identification of proteases from periodontopathogenic bacteria as activators of latent human neutrophils and fibroblast-type interstitial collagenases. Infect Immun. 1992; 60:4491–4495.
3. Lee W, Aitken S, Sodek J, McCulloch CAEvidence of a direct relationship between neutrophil collagenase activity and periodontal tissue destruction in vivo: role of active enzyme in human periodontitis. J Periodontal Res. 1995; 30:23–33.
4. Geerts SO, Legrand V, Charpentier J, Albert A, Rompen EH. Further evidence of the association between periodontal conditions and coronary artery disease. J Periodontal. 2004; 75:1274–1280.
5. Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis: assembling the players. Periodontal 2000. 1997; 14:33–53.
6. Friedewald VE, Kornman KS, Beck JD, Genco R, Goldfine A, Libby P, et al. The American Journal of Cardiology and Journal of Periodontology editor's consensus: periodontitis and atherosclerotic cardiovascular disease. J Periodontal. 2009; 80:1021–1032.
7. Amar S, Han X. The impact of periodontal infection on systemic diseases. Med Sci Monit. 2003;9:RA291–RA299.
8. Kapila YL. Oral health's inextricable connection to systemic health: Special populations bring to bear multimodal relationships and factors connecting periodontal disease to systemic diseases and conditions. Periodontol 2000. 2021;87(1):11-16.

9. Abusleme L, Morandini AC, Hashizume-Takizawa T, Sahingur SE. Editorial: Oral Microbiome and Inflammation Connection to Systemic Health. Front Cell Infect Microbiol. 2021; 11:780182.
10. Nazir MA, Izhar F, Akhtar K, Almas K. Dentists' awareness about the link between oral and systemic health. J Family Community Med. 2019 Sep-Dec;26(3):206-212.
11. Borgnakke WS, Ylöstalo PV, Taylor GW, Genco RJ Effect of periodontal disease on diabetes: Systematic review of epidemiological observational evidence J Periodontol 2013;84(4): S135–S152.
12. Caton JG, Armitage G, Berglundh T, Chapple ILC, Jepsen S, Kornman KS, et al. A new classification scheme for periodontal and peri-implant diseases and conditions—Introduction and key changes from the 1999 classification J Periodontol 2018;89(1): S1–S8.
13. Schmidt AM, Weidman E, Lalla E, Yan SD, Hori O, Cao R, et al. Advanced glycation end products (AGEs) induce oxidant stress in the gingiva: a potential mechanism underlying accelerated periodontal disease associated with diabetes. J Periodontal Res. 1996; 31:508–15.
14. Lalla E, Lamster IB, Drury S, Fu C, Schmidt AM. Hyperglycemia, glycoxidation and receptor for advanced glycation end products: potential mechanisms underlying diabetic complications, including diabetes-associated periodontitis. Periodontol 2000. 2000; 23:50–62.
15. Lalla E, Lamster IB, Schmidt AM. Enhanced interaction of advanced glycation end products with their cellular receptor RAGE. Implications for the pathogenesis of accelerated periodontal disease. Ann Periodontol. 1998; 3:13–19.

16. Tonetti MS, Greenwell H, Kornman KS Staging and grading of periodontitis: Framework and proposal of a new classification and case definition J Periodontol 2018;89(1): S159–S172.
17. Genco RJ, Graziani F, Hasturk H Effects of periodontal disease on glycemic control, complications, and incidence of diabetes mellitus Periodontology 2000 2020;83:59–65.
18. Winning L, Linden GJ Periodontitis and systemic disease: Association or causality? Curr Oral Health Rep 2017; 4:1–7.
19. Taylor GW Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiological perspective Ann Periodontol 2001;6(1):99–112.
20. Borgnakke WS IDF Diabetes Atlas: Diabetes and oral health—A two-way relationship of clinical importance Diabetes Res Clin Pract 2019; 157:107839.
21. American Diabetes Association. Glycemic Targets: Standards of Medical Care in Diabetes. Diabetes Care 2018; 41(1): S55–S64.
22. Green JC, Vermillon JR. The simplified oral hygiene index. JADA.1964;68:7–13.
23. World Health Organization. Oral Health Surveys, Basic Methods. 4th ed. Geneva, Switzerland: World Health Organization; 1998:1–67.
24. Sanz M, Ceriello A, Buysschaert M, Chapple I, Demmer RT, Graciani F, et al. Scientific evidence on the links between periodontal diseases and diabetes: Consensus report and guidelines of the joint workshop on periodontal diseases and diabetes by the International Diabetes Federation and the European Federation of Periodontology J Clin Periodontol 2018;45:138–149.
25. Genco RJ, Sanz M Clinical and public health implications of periodontal and systemic diseases: An overview Periodontol 2000 2020;83:7–13.

26. Polak D, Saunui T, Nishimura F, Shapira L Diabetes as a risk factor for periodontal disease—plausible mechanisms Periodontol 2000 2020;83(1):46–58.
27. Lakschevitz F, Aboodi G, Tenenbaum H, Glogauer M Diabetes and periodontal diseases: Interplay and links Curr Diabetes Rev 2011;7:433–439.
28. Rapone B, Ferrara E, Corsalini M, Qorri E, Converti I, Lorusso F, Delvecchio M, Gnoni A, Scacco S, Scarano A. Inflammatory Status and Glycemic Control Level of Patients with Type 2 Diabetes and Periodontitis: A Randomized Clinical Trial. Int J Environ Res Public Health. 2021;18(6):3018.
29. Grossi SG, Skrepinski FB, DeCaro T, Robertson DC, Ho AW, Dunford RG, et al. Treatment of periodontal disease in diabetics reduces glycated hemoglobin. Journal of Periodontology 1997;68(8):713-719.
30. Kothiwale SV, Kothiwale VA, Bhargava PV. Effect of Non-Invasive Periodontal Therapy on Glycaemic Control in Type 2 Diabetes Mellitus Patients-A Randomized Control Trial. InDiabetes 2013;62: A229-A229.
31. Santos VR, Lima JA, Miranda TS, Gonçalves TE, Figueiredo LC, Faveri M. Full-mouth disinfection as a therapeutic protocol for type-2 diabetic subjects with chronic periodontitis: Twelve-month clinical outcomes. A randomized controlled clinical trial. Journal of Clinical Periodontology 2013;40(2):155-162.
32. Simpson TC, Weldon JC, Worthington HV, et al. Treatment of periodontal disease for glycaemic control in people with diabetes mellitus. Cochrane Database Syst Rev. 2015;2015(11):CD004714.
33. Florkowski C. HbA1c standardization issues: should New Zealand follow the DCCT or the IFCC position? The New Zealand Medical Journal 2003;116(1171): U395.

34. Hanas R, John G, International HBA1c Consensus Committee. 2010 consensus statement on the worldwide standardization of the hemoglobin A1C measurement. Diabetes Care 2010;33(8):1903-1904.
35. Engebretson SP, Hyman LG, Michalowicz BS, Schoenfeld ER, Gelato MC, Hou W, Seaquist ER, Reddy MS, Lewis CE, Oates TW, Tripathy D, Katancik JA, Orlander PR, Paquette DW, Hanson NQ, Tsai MY. The effect of nonsurgical periodontal therapy on hemoglobin A1c levels in persons with type 2 diabetes and chronic periodontitis: a randomized clinical trial. JAMA. 2013 Dec 18;310(23):2523-32.
36. Kebede TG, Pink C, Rathmann W, Kowall B, Völzke H, Petersmann A, Meisel P, Dietrich T, Kocher T, Holtfreter B. Does periodontitis affect diabetes incidence and haemoglobin A1c change? An 11-year follow-up study. Diabetes Metab. 2018; 44(3):243-249.
37. Chiu SY, Lai H, Yen AM, Fann JC, Chen LS, Chen HH. Temporal sequence of the bidirectional relationship between hyperglycemia and periodontal disease: a community-based study of 5,885 Taiwanese aged 35-44 years (KCIS No. 32). Acta Diabetol. 2015 ;52(1):123-131.
38. Demmer, RT, Jacobs DR. Jr, 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.
39. Ide R, Hoshuyama T, Wilson D, Takahashi K, Higashi T. Periodontal disease and incident diabetes: a seven-year study. Journal of Dental Research 2011; 90: 41– 46.
40. Morita T, Yamazaki Y, Mita A, Takada K, Seto M, Nishinoue N, Sasaki Y, Motohashi M, Maeno M. A cohort study on the association between periodontal disease and the development of metabolic syndrome. Journal of Periodontology 2010; 81: 512– 519.

41. Saito T, Shimazaki Y, Kiyohara Y, Kato I, Kubo M, Iida M, Koga T. The severity of periodontal disease is associated with the development of glucose intolerance in non-diabetics: the Hisayama study. Journal of Dental Research 2004; 83: 485– 490.
42. Xiong X, Elkind-Hirsch KE, Vastardis S, Delarosa RL, Pridjian G, Buekens P. Periodontal disease is associated with gestational diabetes mellitus: a case-control study. Journal of Periodontology 2009; 80: 1742– 1749.
43. Preshaw PM, Alba AL, Herrera D, Jepsen S, Konstantinidis A, Makrilakis K, Taylor R. Periodontitis and diabetes: A two-way relationship. Diabetologia. 2012; 55:21–31.
44. Winning L, Patterson CC, Neville CE, Kee F, Linden GJ. Periodontitis and incident type 2 diabetes: A prospective cohort study. J Clin Periodontol. 2017; 44:266–274.
45. Stoicescu M, Calniceanu H, Țig I, Nemeth S, Tent A, Popa A, Brisc C, Ignat-Romanul I. Significant aspects and correlation between glycemic control and generalized chronic periodontitis in type 2 diabetes mellitus patients. Exp Ther Med. 2021;22(1):671.
46. Shinjo T, Ishikado A, Hasturk H, Pober DM, Paniagua SM, Shah H, Wu IH, Tinsley LJ, Matsumoto M, Keenan HA, Van Dyke TE, Genco RJ, King GL. Characterization of periodontitis in people with type 1 diabetes of 50 years or longer duration. J Periodontol. 2019 ;90(6):565-575. doi: 10.1002/JPER.18-0735.
47. Tinsley LJ, Kupelian V, D'Eon SA, Pober D, Sun JK, King GL, Keenan HA. Association of Glycemic Control with Reduced Risk for Large-Vessel Disease After More Than 50 Years of Type 1 Diabetes. J Clin Endocrinol Metab. 2017 Oct 1;102(10):3704-3711.

Tables

Table 1a. Participants' demographics

Variables	Teaching Hospitals						$\chi 2$	p-value
	UPTH		LASUTH		Total			
	Freq	%	Freq	%	Freq	%		
Sex							**1.57**	**0.21**
Female	67	63.8	58	72.5	125	67.6		
Male	38	36.2	22	27.5	60	32.4		
Age group							**29.82**	**<0.0001***
20-29	2	1.9	2	2.5	4	2.2		
30-39	11	10.4	4	5.0	15	8.1		
40-49	30	28.6	3	3.7	33	17.7		
50-59	30	28.6	21	26.3	51	27.6		
60-69	19	18.1	22	27.5	41	22.2		
>70	13	12.4	28	35.0	41	22.2		
Tribe							**78.29**	**<0.0001***
Hausa	7	6.7	0	0.0	7	3.8		
Igbo	34	32.4	20	25.0	54	29.2		
Yoruba	10	9.5	53	66.2	63	34.0		
Rivers	34	32.4	0	0.0	34	18.4		
Others	20	19.0	7	8.8	27	14.6		
Education							**9.51**	**0.02***
Informal	18	17.1	3	3.8	21	11.4		
Primary	19	18.1	22	27.5	41	22.2		

Secondary	27	25.7	25	31.2	52	28.1		
Tertiary	41	39.1	30	37.5	71	38.4		
Occupation							**15.09**	**0.005***
Civil servant	26	24.8	15	18.7	41	22.2		
Retired	14	13.3	28	35.0	42	22.7		
Farmer	4	3.8	1	1.3	5	2.7		
Self-employed	56	53.3	36	45.0	92	49.7		
Professionals	5	4.8	0	0.0	5	2.7		
Duration of diagnosis (years)							**5.13**	**0.53**
1-5	43	41.0	31	38.7	74	40.0		
6-10	34	32.4	20	25.0	54	29.2		
11-15	17	16.2	14	17.5	31	16.8		
16-20	8	7.7	10	12.5	18	9.7		
21-25	1	0.9	3	3.8	4	2.2		
26-30	1	0.9	2	2.5	3	1.6		
>30	1	0.9	0	0.0	1	0.5		
Total	**105**	**100.0**	**80**	**100.0**	**185**	**100.0**		

Mean age = 57.11±13.45 years; Mean DM duration 8.97±7.14years

Table 1b. Participants' characteristics

Variables	**Mean ± SD; %** **Well controlled (Hb1AC <7%)**	**Mean ± SD; %** **Poorly controlled (Hb1AC ≥7%)**	**Total**
Age (years)	58.1±16.0	56.5211.7	57.11±13.46
Duration (years)	9.0±7.8	8.9±6.8	8.97±7.17
BMI (kg/m^2)	26.9±5.1	27.6±4.9	27.34±5.00
Hb1Ac (%)	6.1±0.6	9.5±2.3	8.22±2.49
Total Cholesterol	2.8±1.1	3.1±1.6	2.95±1.44
LDL (mmol/L)	1.8±1.0	2.1±1.3	1.31±1.99
HDL (mmol/L)	0.8±0.6	0.9±0.7	0.84±0.62
Triglycerides (mmol/L)	1.5±1.0	2.3±1.7	1.99±1.54
OHI-S	2.2±1.1	2.3±1.2	2.25±1.12
CPI	2.2±1.0	2.2±0.9	2.19±0.92

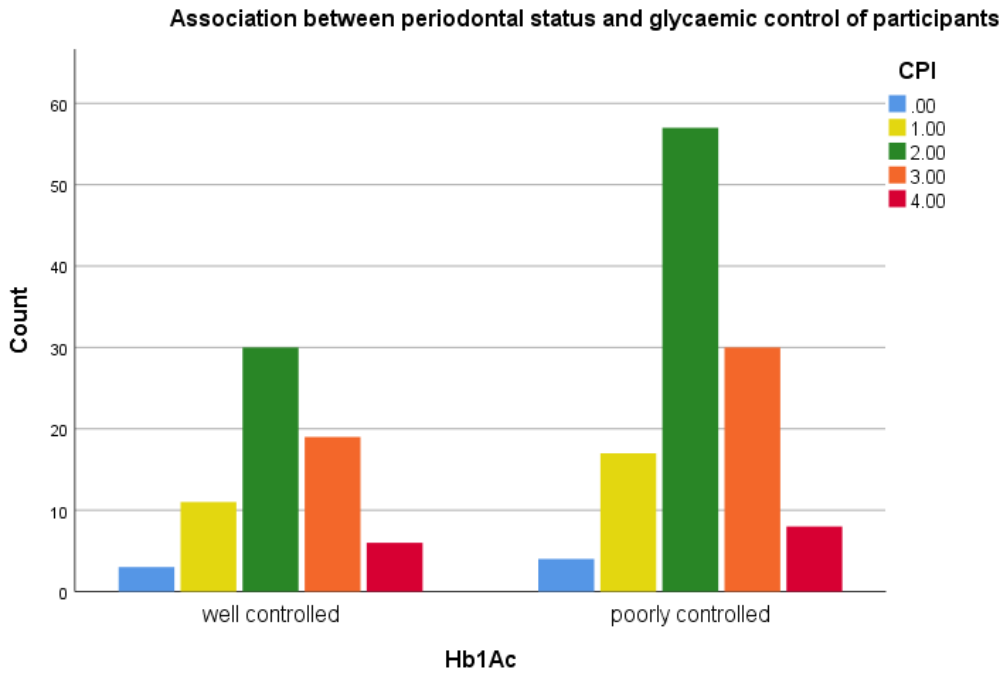

Figure1. Community periodontal index scores with glycated haemoglobin

Table 2. Participants glycaemic and periodontal status

Variables	Teaching Hospitals						$\chi 2$	p-value
	UPTH		LASUTH		Total			
	Freq	%	Freq	%	Freq	%		
HbA1c							**11.73**	**0.001***
<7% (well controlled diabetes)	28	26.7	41	51.3	69	37.3		
≥7% (poorly controlled diabetes)	77	73.3	39	48.7	116	62.7		
OHI-S							**3.59**	**0.17**
Good	24	22.9	18	22.5	42	22.7		
Fair	66	62.9	42	52.5	108	58.4		
Poor	15	14.2	20	25.0	35	18.9		
CPI scores							**11.46**	**0.02***
0	0	0.0	7	8.7	7	3.8		
1	14	13.3	14	17.5	28	15.1		
2	51	48.6	36	45.0	87	47.0		

3	30	28.6	19	23.8	49	26.5		
4	10	9.5	4	5.0	14	7.6		
Total	**105**	**100.0**	**80**	**100.0**	**185**	**100.0**		

Table 3. Association between glycaemic control and periodontal status

Variables		**Teaching Hospitals**						**χ^2**	**p-value**
		UPTH		**LASUTH**		**TOTAL**			
HbA1c		**Freq**	**%**	**Freq**	**%**	**Freq**	**%**		
≤7%	**OHI-S**							**1.07**	**0.59**
Good	7	25.0	12	29.2	19	27.6			
Fair	17	60.7	20	48.8	37	53.6			
Poor	4	14.3	9	22.0	13	18.8			
Total	**28**	**100.0**	**41**	**100.0**	**69**	**100.0**			
≥7%	**OHI-S**							**3.45**	**0.18**
Good	17	22.1	6	15.4	23	91.8			
Fair	49	63.6	22	56.4	71	61.2			
Poor	11	14.3	11	28.2	22	19.0			
Total	**77**	**100.0**	**39**	**100.0**	**116**	**100.0**			
<7%	**CPI scores**							**10.63**	**0.03***
0	0	0.0	3	7.3	3	4.4			
1	1	3.6	10	24.4	11	15.9			
2	12	42.8	18	43.9	30	43.5			
3	11	39.3	8	19.5	19	27.5			
4	4	14.3	2	4.9	6	8.7			
Total	**28**	**100.0**	**41**	**100.0**	**69**	**100.0**			
≥7%	**CPI Scores**							**9.17**	**0.06**
0	0	0.0	4	10.3	4	3.4			
1	13	16.9	4	10.3	17	14.7			

2	39	50.6	18	64.2	57	49.1			
3	19	24.7	11	28.2	30	25.9			
4	6	7.8	2	5.1	8	6.9			
Total	**77**	**100.0**	**39**	**100.0**	**116**	**100.0**			

Table4a. Association between participants glycaemic control and some demographics

Variables	**Teaching Hospitals**						**χ2**	**p-value**
	UPTH		**LASUTH**		**TOTAL**			
HbA1c	**Freq**	**%**	**Freq**	**%**	**Freq**	**%**		
<7%	**Gender**						**6.97**	**0.014***
Female	15	53.6	34	82.9	49	71.0		
Male	13	46.4	7	17.1	20	29.0		
Total	**28**	**100.0**	**41**	**100.0**	**69**	**100.0**		
≥7%	**Gender**							
Female	52	67.5	24	61.5	76	65.5	**0.41**	**0.52**
Male	25	32.5	15	38.5	40	34.5		
Total	**77**	**100.0**	**39**	**100.0**	**116**	**100.0**		
<7%	**Age group (years)**						**19.64**	**0.001***
20-29	2	7.1	1	2.4	3	4.3		
30-39	5	17.9	3	7.3	8	11.7		
40-49	8	28.6	1	2.4	9	13.0		
50-59	7	25.0	7	17.1	14	20.3		
60-69	2	7.1	10	24.5	12	17.4		
>70	4	14.3	19	46.3	23	33.3		
Total	**28**	**100.0**	**41**	**100.0**	**69**	**100.0**		
Mean age± SD	58.10±16.02 years							
≥7%	**Age group (years)**						**13.27**	**0.021***
20-29	0	0.0	1	2.6	1	0.9		
30-39	6	7.8	1	2.6	7	6.0		

40-49	22	28.6	2	5.1	24	20.7		
50-59	23	29.9	14	35.8	37	31.9		
60-69	17	22.0	12	30.8	29	25.0		
>70	9	11.7	9	23.1	18	15.5		
Total	**77**	**100.0**	**39**	**100.0**	**116**	**100.0**		
Mean age± SD	56.52±11.70years							
<7%	**Year of diagnosis (years)**						**3.01**	**0.08**
0-5	16	57.2	17	41.5	33	47.9		
6-10	6	21.4	7	17.1	13	18.8		
11-15	4	14.3	9	21.9	13	18.8		
16-20	2	7.1	4	9.8	6	8.7		
21-25	0	0.0	3	7.3	3	4.4		
26-30	0	0.0	1	2.4	1	1.4		
Total	**28**	**100.0**	**41**	**100.0**	**69**	**100.0**		
Mean duration± SD	6.10± 0.57years							
≥7%	**Year of diagnosis (years)**						**1.25**	**0.54**
0-5	27	35.1	14	35.9	41	35.3		
6-10	28	36.3	13	33.3	41	35.3		
11-15	13	16.9	5	12.8	18	15.6		
16-20	6	7.8	6	15.4	12	10.3		
21-25	1	1.3	0	0.0	1	0.9		
26-30	1	1.3	1	2.6	2	1.7		
>30	1	1.3	0	0.0	1	0.9		
Total	**77**	**100.0**	**39**	**100.0**	**116**	**100.0**		

Mean duration± SD	9.48±2.34years		

Table4b. Association between participants glycaemic control and some demographics

Variables	**Teaching Hospitals**						**χ2**	**p-value**
	UPTH		**LASUTH**		**TOTAL**			
HbA1c	**Freq**	**%**	**Freq**	**%**	**Freq**	**%**		
<7%	**Education**						**6.24**	**0.10**
Informal	3	10.7	1	2.4	4	57.9		
Primary	3	10.7	14	34.1	17	24.6		
Secondary	8	28.6	9	22.0	17	24.6		
Tertiary	14	50.0	17	41.5	31	44.9		
Total	**28**	**100.0**	**41**	**100.0**	**69**	**100.0**		
≥7%	**Education**						**5.96**	**0.11**
Informal	15	19.5	2	5.1	17	14.7		
Primary	16	20.8	8	20/5	24	20.7		
Secondary	19	24.7	16	41.0	35	30.2		
Tertiary	27	35.1	13	33.3	40	34.5		
Total	**77**	**100.0**	**39**	**100.0**	**116**	**100.0**		
<7%	**Occupation**						**7.50**	**0.11**
Civil servant	5	17.9	7	17.1	12	17.4		
Retired	5	17.9	18	43.9	23	33.3		
Farmer	1	3.5	1	2.4	2	2.9		
Self-employed	15	53.6	15	36.6	30	43.4		
Professionals	2	7.1	0	0.0	2	2.9		
Total	**28**	**100.0**	**41**	**100.0**	**69**	**100.0**		
≥7%	**Occupation**						**6.59**	**0.16**

Civil servant	21	27.3	8	20.5	29	25.0		
Retired	9	11.7	10	25.6	19	16.4		
Farmer	3	3.9	0	0.0	3	2.6		
Self-employed	41	53.2	21	33.9	62	53.4		
Professionals	3	3.9	0	0.0	3	2.6		
Total	**77**	**100.0**	**39**	**100.0**	**116**	**100.0**		