Original Research Article

Assessment of periodontal status in adults with diabetes mellitus

Manish Deheriya[1], Anuj Bhargava[2]*, Dushyant Pippal[3], Anupama Ahirwar[3]

[1]Junior Resident, Department of Dentistry, Gandhi Medical College, Bhopal, Madhya Pradesh, India
[2]Assistant Professor, Department of Dentistry, Gandhi Medical College, Bhopal, Madhya Pradesh, India
[3]Senior Resident, Department of Dentistry, Gandhi Medical College, Bhopal, Madhya Pradesh, India

*Correspondence:
Dr. Anuj Bhargava,
E-mail: anujbhargava23@gmail.com

ABSTRACT

Background: Diabetes mellitus is a group of chronic metabolic disorders known to exhibit a myriad of complications over a period of time. Periodontal disease is the sixth most common complication in diabetic patients. The aim of the study was to assess the periodontal status of adult diabetic patients.
Methods: 100 diabetic patients in the age group of 25-80 years fulfilling the inclusion criteria were examined by a calibrated WHO CPI probe to assess their periodontal status as per the scoring criteria of the community periodontal index. Student t test, Chi square test and ANOVA F were applied for statistical analysis. p>0.05 was considered not significant and p<0.01 was considered highly significant.
Results: A prevalence of 73% periodontitis was found in diabetic study population with statistically high significance (p=0.001) found according to age. A total of 52% Shallow pockets and 15% Deep pockets were reported respectively in middle (41-56 years) and older (57-80 years) age groups. Further, 47% male population was found to have statistically significant (p=0.027) more periodontitis (shallow and deep periodontal pockets) compared to female (26%) population.
Conclusions: Within limitations of the study it may be safely concluded that assessment of periodontal status of DM patients revealed chronic periodontal destruction particularly in age groups beyond 40 years in majority of study population depicting that age is significantly associated with the increased prevalence and severity of periodontal disease in patients with diabetes.

Keywords: Diabetes, Periodontitis, Prevalence

INTRODUCTION

Diabetes mellitus (DM) is a group of chronic metabolic disorders characterized by disturbance in carbohydrate, lipid and protein metabolism, originating either due to insufficient production of insulin by beta cells of pancreas (Type 1 DM) or due to increased insulin resistance in the body (Type 2 DM). It is primarily manifested as increased blood sugar over a prolonged period of time.[1] DM is an increasing burden on the economies of many developed and developing countries due to its increasing prevalence. According to report by International Diabetes Federation, around 415 million people worldwide were suffering from diabetes in 2015. This number is estimated to reach 640 million by the year 2040.[2] Researches suggest that every 5th person suffering from diabetes will be Indian and hence the economic burden in India due to diabetes will be highest in the world.[3] In 1970s, the prevalence of diabetes among urban Indians was reported to be 2.1%, and this has now risen to 12.1%. According to the World Health Organization (WHO) projections, the present population of 30 to 33 million diabetics in India will go up to 74 million by 2025. The WHO has issued a warning that India will be the diabetes capital of the World.[4] This rising trend in the incidence can be attributed to shift in lifestyle towards

urbanization leading to reduced physical activity and hence obesity.[5]

DM is known to exhibit a myriad of complications over a period of time. The macrovascular complications include cardiovascular disease, stroke, peripheral artery disease along with microvascular complications comprising of retinopathy, neuropathy and nephropathy.[2] DM also predisposes an individual to higher susceptibility to infections due to reduction in functional activity of neutrophils which are the first line of defence of human immune system. Clinical investigations have demonstrated definite and consistent defect in microbicidal, chemotactic and phagocytic properties of neutrophils in patients suffering from diabetes.[5] This might be one of the reasons that predisposes a diabetic patient to oral infections and other oral manifestations such as xerostomia, glossitis, geographic tongue, taste disorders, gingivitis, periodontitis, dental caries, altered tooth eruption, candidiasis, aspergillosis, mucormycosis, oral lichen planus and delayed wound healing.[6]

Loe stated that periodontal disease is the sixth most common complication in diabetic patients.[7] Periodontitis is also one of the most commonly occurring oral disease worldwide.[8] Periodontal disease (PD) begins with gingivitis, the localized inflammation by bacteria in the dental plaque, progresses to bleeding and swollen gingiva, loss of the gingiva including bone and periodontal ligament (the connective tissue collagen fibers) which further creates shallow to deep periodontal 'pockets' that are a hallmark of periodontitis and can eventually lead to mobility and finally tooth loss. Most common organisms associated with periodontal diseases are *Porphyromonas gingivalis*, *Prevotella intermedia*, *Bacteroides forsythus*, *Campylobacter rectus*, *Actinobacillus actinomycete-mcomitans* and treponemes.[9]

Taylor et al demonstrated that presence of diabetes increases the prevalence, incidence, severity and progression of periodontitis, and periodontal infection is associated with poorer glycemic control in people with diabetes. The study also concluded that treating periodontal infections can be influential in contributing to glycemic control management and possibly to the reduction of the burden of complications of diabetes mellitus.[10]

Although there have been multiple studies to establish a correlation between DM and periodontitis but still their cause and effect relationship remains complex and controversial. One school of thought believes that the probable correlation between DM and periodontitis can be attributed to factors like microvascular alterations, suppressed host immune response, altered subgingival flora which becomes predominantly gram negative, defective collagen metabolism weakening the periodontal fiber structure and non enzymatic glycation. The other school of thought maintains that the presence, distribution and severity of local irritants affects the severity of periodontitis in diabetic patients.[11]

Thus to gain better insight a cross sectional study was planned with an aim to assess the periodontal status of patients suffering from diabetes mellitus using appropriate dental indices.

METHODS

Study design

Current study is a cross sectional prevalence study.

Study population and sample selection

Study participants were enrolled from outpatient block of department of dental surgery, Gandhi Medical College and Hamidia hospital, Bhopal from December 2018-January 2020. Patients pre diagnosed and confirmed with non insulin dependent type II diabetes mellitus (NIDDM) by the consultant Endocrinologist of our institute were selected. Random blood sugar (RBS) estimation of such patients was also done before recording their Periodontal Status.

A total number of 100 patients in the age group of 25-80 years were included in the study based on the inclusion criteria; patients with pre existing and confirmed type 2 diabetes mellitus by endocrionologist, patients with no other systemic diseases other than diabetes, patients with minimum number of 8 teeth present in the oral cavity, patients not on any drug therapy such as phenytoin, nefidipine, cyclosporine etc which may have a direct impact on the gingival health, patient not undergone any periodontal treatment in last 1 year. The exclusion criteria included; patients below 25 years of age, patients with history of Smoking and/or tobacco chewing, patients with medical history of pregnancy or lactation.

Data collection procedure

All those patients who satisfied the inclusion criteria received detailed information regarding the study and further only those patients were included who signed an written informed consent drafted in accordance with the Helsinki declaration of 1975 (revised in 2000). Confidentiality of each study subject was ensured. They were also given the option of withdrawing from the study at any given point of time without assigning any reason. A translated consent form was then completed and signed by the study participants upon agreement to participate.

Methodology

After recording the patient's chief complaint according to the designed and validated case history format, written informed consent was obtained from all the patients that their information would be used in this study.

Clinical parameters and recording of clinical data

A careful oral examination was carried out under all aseptic precautions using a mouth mirror and a World Health Organization (WHO) CPI probe to calculate community periodontal index (CPI) which is an epidemiological dental index to assess the Periodontal Status of a study population.

A WHO CPI probe with calibrated black bands between 3.5 mm, 5.5 mm and rings at 8.5 mm and 11.5 mm along with a 0.5 mm ball tip attached to its working end was used to record CPI Index by a single trained dental examiner. Application of a maximum force not exceeding 20 gms by the examiner was ensured by repetitive training of the examiner by exercising placing the CPI probe under the thumb nail and pressing until the blanching occurs. The training was done before starting the study.[12] It was done to minimize measurement errors.

The entire oral cavity was divided into six sextants;18-14, 13-23, 24-28, 38-34, 33-43 and 44-48.CPI Score recording was done on index teeth with tooth number 17-16, 11, 26-26, 36-37, 31, 46-47. A sextant was only examined if two or more teeth were present. CPI Probe was used as a sensing instrument to determine pocket depth, bleeding on probing and calculus. The Probe tip was inserted gently into the gingival sulcus on the distobuccal surfaces of the indexed teeth and gentle walking of the probe in short upward and downward movements was done till the margins to the mesiobuccal surface of the index teeth readings were recorded using community periodontal index (CPI) scoring system from code 0 to code 4. (code 0:healthy periodontal condition; code 1:gingival bleeding on probing; code 2:supra or sub gingival calculus and/or overhanging of fillings or crown; code 3: pathological pocket of 4-5 mm; code 4: pathological pocket of 6mm deep or more; code X:the index cannot be recorded. For an individual (n=100) the most severe score recorded amongst the 6 sextants was taken as the final CPI score.[13]

Statistical analysis

The data obtained was subjected to statistical analysis with consultation of a statistician. The data so obtained was compiled systematically. A master table was prepared and the total data was subdivided and distributed meaningfully and presented as individual tables along with graphs.

Statistical procedures were carried out in 2 steps; data compilation and presentation and statistical analysis

Statistical analysis was done using statistical package of social science (SPSS Version 20; Chicago Inc., USA). Data comparison was done by applying specific statistical tests to find out the statistical significance of the comparisons. Chi Square test was used in tables to determine demographic distribution and calculate the prevalence of periodontal disease of study subjects (diabetic patients) according to age and gender. Mean number of sextants affected by periodontal disease according to gender among diabetes patients was calculated by Student t test. Further, mean number of sextants affected by periodontal disease according to age among diabetes patients was calculated by application of ANOVA F value. p value was ascertained as, p>0.05; not significant and p<0.01; highly significant (significant at 99.9% confidence level).

RESULTS

Demographic distribution of study subjects (diabetes patients) according to age and gender is revealed in (Table 1 and Figure 1). Total 100 diabetes mellitus patients were selected for the study to find out the prevalence of periodontal disease. Mean age of all patients was 51.41 year and they were in the range of 26-80 year. Mean random blood sugar level of all patients was 213.93 mg/dl (127-524 mg/dl).

Table 1: Demographic distribution of study subjects (diabetes patients) according to age and gender.

Age groups (years)	Male N (%)	Female N (%)	Total N (%)
25-40	8 (8.0)	10 (10.0)	18 (18.0)
41-56	29 (29.0)	18 (18.0)	47 (47.0)
57-80	21 (21.0)	14 (14.0)	35 (35.0)
Total	58 (58.0)	42 (42.0)	100 (100.0)
Chi Square value	1.680		
P value	0.432 (non significant)		
Mean age	51.41 (26-80 years)		
Mean sugar	213.93 (127-524 mg/dl)		
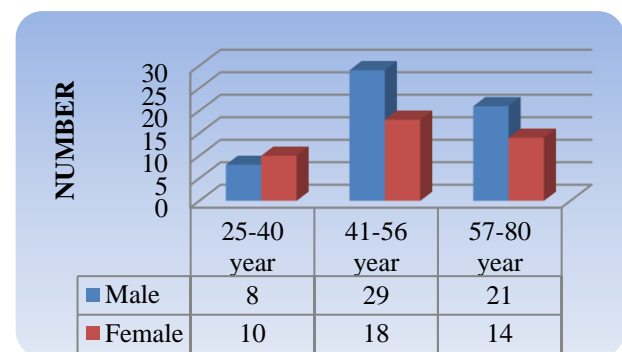
Figure 1: Demographic distribution of study subjects (diabetes patients) according to age and gender.

Orevalence of periodontitis (periodontal disease) according to gender among diabetes patients is revealed in (Table 2). Out of 100 patients, most of 56 (56.0%) had shallow pocket, 17 (17.0%) had deep pocket and 27 (27.0%) had calculus. Out of 56 cases of shallow pocket, 39 (39.0%) were male and 17 (17.0%) were female. Out of 17 cases of deep pocket, 8 (8.0%) were males and 9

(9.0%) were females. There was statistically significant difference found in prevalence of periodontitis (periodontal disease) according to gender (p=0.027).

Table 2: Prevalence of periodontitis (periodontal disease) according to gender among diabetes patients.

Gender	N	CPI score 2 calculus N (%)	CPI score 3 S. pocket N (%)	CPI score 4 deep pocket N (%)
Male	58	11 (11)	39 (39)	8 (8)
Female	42	16 (16)	17 (17)	9 (9)
Total	100	27 (27)	56 (56)	17 (17)
Chi Square value	7.253			
P value	0.027 (significant)			

Prevalence of periodontitis (periodontal disease) according to age among diabetes patients is revealed in (Table 3). Out of 100 patients, most of 56 (56.0%) had shallow pocket, 17 (17.0%) had deep pocket and 27 (27.0%) had calculus. Out of 56 cases of shallow pocket, most of 32 (32.0%) were 41-56 year old, 20 (20.0%) were 57-80 year old. There was statistically highly significant difference found in prevalence of periodontitis (periodontal disease) according to age (p=0.001).

Mean number of sextants affected by periodontal disease according to gender among diabetes patients is revealed in (Table 4). Mean number of sextants affected by bleeding was 0.41±0.95 among male and 0.67±1.09 among female. Mean number of sextants affected by

calculus was 2.84±1.42 among male and 3.02±1.63 among female. Mean number of sextants affected by shallow pockets was 2.45±1.50 among male and 1.95±1.66 among female. Mean number of sextants affected by deep pocket was 2.45±1.50 among male and 1.95±1.66 among female. Statistically no significant difference was found in mean number of sextants affected by periodontal disease according to gender among diabetes patients (p>0.05).

Table 3: Prevalence of periodontitis (periodontal disease) according to age among diabetes patients.

Age (year)	N	CPI score 2 calculus N (%)	CPI score 3 S. pocket N (%)	CPI score 4 deep pocket N (%)
25-40	18	12 (12)	4 (4)	2 (2)
41-56	47	12 (12)	32 (32)	3 (3)
57-80	35	3 (3)	20 (20)	12 (12)
Total	100	27 (27)	56 (56)	17 (17)
Chi Square value	29.466			
P value	0.001(highly significant)			

Mean number of sextants affected by periodontal disease according to age among diabetes patients is revealed in (Table 5). Mean number of sextants affected by bleeding was 0.52±1.020. Mean number of sextants affected by calculus was 2.92±1.50. Mean number of sextants affected by shallow pocket was 2.24±1.57. Mean number of sextants affected by deep pocket was 0.32±0.76. There was statistically high significant difference found in mean number of sextants affected by periodontal disease according to age among diabetes patients (p<0.001).

Table 4: Mean number of sextants affected by periodontal disease according to gender among diabetes patients.

Gender	Number	CPI score 1 bleeding Mean±SD	CPI score 2 calculus Mean±SD	CPI score 3 S. pocket Mean±SD	CPI score 4 deep pocket Mean±SD
Male	58	0.41±0.95	2.84±1.42	2.45±1.50	0.29±0.79
Female	42	0.67±1.09	3.02±1.63	1.95±1.66	0.36±0.72
Student 't' test value		1.227	0.584	1.563	0.412
P value		0.223 (non significant)	0.561 (non significant)	0.121 (non significant)	0.681 (non significant)

Table 5: Mean number of sextants affected by periodontal disease according to age among diabetes patients.

Age (year)	Number	CPI score 1 bleeding Mean±SD	CPI score 2 calculus Mean±SD	CPI score 3 S. pocket Mean±SD	CPI score 4 deep pocket Mean±SD
25-40 year	18	1.11±1.32	3.78±1.35	1.00±1.45	0.11±0.32
41-56 year	47	0.55±1.05	2.96±1.58	2.30±1.61	0.19±0.74
57-80 year	35	0.17±0.568	2.43±1.29	2.80±1.23	0.60±0.88
Total	100	0.52±1.020	2.92±1.50	2.24±1.57	0.32±0.76
ANOVA F value		5.563	5.184	9.074	3.907
P value		0.005 (highly significant)	0.007 (highly significant)	0.001 (highly significant)	0.023 (significant)

DISCUSSION

Periodontal disease (PD) is not a solitary disease but represents a cascade of pathological events which affect the periodontium.[14] The two most common PD are gingivitis which is a reversible inflammation of gingiva and chronic periodontitis.[15] The role of diabetes mellitus and PD has been researched over years. The signs and symptoms of breakdown of tooth supporting structures have been now recognized as sixth complication of DM and are a consequence of hyperglycemia. Thus, glycemic levels play a pivotal role in complications associated with DM.[16-18]

Abundant literature is available which has concluded that diabetes poses as a risk factor for poor oral health and periodontal infection increases the severity of diabetes and adversely affects glycemic control.[10]

Our present descriptive cross sectional study reports the prevalence of periodontitis in adult diabetic patients by assessing their periodontal status using community periodontal index (CPI). The results illustrate that a total of 73% of the diabetic subjects in this study had a CPI score of either 3 (56%; shallow pockets) or 4 (17%; deep pockets) depicting a chronic periodontitis disease state with destruction of tooth supporting structures (bone & attachment loss). Rest 27% of the population had a CPI score of 2. Our study results are well consistent with the work of Emrich et al who reported that odds were approximately 3 times more for people with diabetes to have destructive periodontal disease.[19] Taylor et al described that diabetic subjects had 4 times more risk for severe alveolar bone loss.[20] Majority of study population in our study had evidence of signs and symptoms of chronic periodontitis which is in consonance with another study conducted by RG Nelson in 1990 on 2273 pima Indians aged 15 years and above from Gila river Indian community in USA which concluded that the rate of PD in study participants with non insulin dependent diabetes is 2.6 times (95%, CI, 1.0-6.6, controlled for age and sex) against healthy controls.[14] Grossi et al and Dolan et al also reported that diabetic individuals are twice more prone to have attachment loss than those without it.[21,22]

In Table 2, 47% male population had CPI score of either 3 (39%; shallow pockets) or 4 (8%, deep pockets) as against 26% of females participants who had CPI score of 3 (17%; shallow pockets) or 4 (9%; deep pockets). Statistically, significant difference (p<0.05) was found in prevalence of PD according to gender (p=0.027). Cross sectional studies by Burt BA and Johnson ES are in conformity to our results since these studies also reported that males have a higher predilection for PD.[23,24] Schulze et al investigated gender dependent differences by the comparison of periodontal status and oral hygiene between diabetic and non-diabetic 517 patients (171 non-diabetic, 205 type 2 diabetic with oral and 141 with insulin therapy) and concluded that periodontitis was more severe in males than in females, with exception of patients using insulin. Periodontal status was worse in men mainly due to oral hygiene behavior. Men apparently need much more advise than women.[25] However, the study on pima Indians by RG Nelson in 1990 reported no significant gender incidence rates (incidence-rate ratio 1.0, 95% confidence interval) 0.5-1.9.[14] These results therefore suggest that there is a greater need for regular periodontal evaluation and effective oral hygiene care among males with diabetes than in diabetic females to decrease the risk of developing periodontitis and progression of periodontitis into a more severe form.

52 % of the diabetic study patients (Table 3) in the age group of 41-56 years (32%) and 57-80 years (20%) had a CPI score of 3 with shallow periodontal pockets. Similarly 15% in the age group of 41-56 years (3%) and 57-80 years (12%) had a CPI score of 4 with deep periodontal pockets depicting chronic periodontitis with severe PD (tooth supporting structural damage, bone and attachment loss). Only 6% of patients in age group of 25-40 years had shallow (4%) or deep periodontal (2%) pockets. Thus, there was statistically highly significant difference found in Prevalence of PD according to age. (p=0.001). The results suggest that non insulin dependent diabetes and age are powerful predictors for development of PD.[14]

Grossi et al in 1994 conducted a study to compare the response of periodontal therapy in diabetic and non-diabetic subjects, according to them, age was the single most important factor most associated with severity of attachment loss. The relative risks for the different age categories increased from 1.72 (95% CI: 1.18-2.49) at age 35 to 44 to a risk of 9.01 (95% CI: 5.86-13.89) at age 65 to 74.[22] Similar results were reported by Rajhans et al where prevalence of periodontal disease in diabetic patients was found to be 86.8% (gingivitis 27.3% and periodontitis 59.5%). The authors concluded that the prevalence as well as severity of the periodontal disease increased with increases in age.[11] Association of age and PD have also been reported by other cross sectional studies done by Page RC in 1985 and the National institute of dental research in 1987.[26,27]

According to American diabetic association (ADA) standards of medical care in diabetes, an FBS > than 152 mg/dl which is comparable to an HbA1c of 7% is considered to be poor control. In our study Mean random blood sugar levels of patients was 213.93 mg/dl (127-524 mg/dl). These random blood glucose levels according to ADA correspond to an HbA1c of 9% which depicts poor glycemic control.[28-30] 73% of our study population had either CPI scores of 3 or 4 which are indicative of moderate to severe form of PD possibly attributable to the increased mean random blood glucose levels due to poor diabetic control. Our study findings are consistent with the study of Awartani on 126 Saudi diabetic females who divided study participants into group I (better

control with HbA1c <9%) and group II (poor control with HbA1c >9%).This study concluded that there was a statistically significant association of loss of attachment with PD in poorly controlled diabetic patients and such patients were vulnerable to higher risk for periodontitis.[31]

In our study CPI scores of 0, 1 and 2 were considered as healthy or having gingivitis. CPI scores of 3 and 4 were regarded to have PD. No diabetic study subject had a sextant with CPI index score of 0.However, mean number of sextant (1.11±1.32) with CPI score of 1was found in the age group of 25-40 years of age. Highest mean number of sextants (3.78±1.35) in CPI score of 2 was found in the age groups of 25-40 years. These results suggest that young adults with diabetes are more prone to develop gingivitis. Contrary to these finding mean number of sextants 2.30±1.61 and 2.80±1.23 for a CPI score of 3 was reported in the age group of 41-56 years and 57 to 80 years respectively depicting PD and tooth supporting structural damage. Further, mean number of Sextants in CPI score 4 in all study age groups was as low as 0.32±0.76. There was statistically high significant difference found in Mean number of sextants affected by periodontal disease according to age among diabetes patients (p<0.001). The results of our study are well consistent to a study conducted in Japan by Ohtake et al who demonstrated that none of the study subjects in their study had a CPI score of 0 in the diabetic group and 90% of the diabetic subjects had a CPI score of 3 and 4 and to study of Masayuki et al on 518 community residents in the age group of 20 to 91 years who analyzed the oral health status amongst Japanese adults with and without DM using CPI index.[32] The authors concluded that mean number of sextants with CPI scores 0, 1 and 2 were fewer as compared to sextants with codes 3 and 4 in diabetic group.[33]

Our study reported that 73% of the adult diabetic population had a CPI score of either 3 or 4 depicting a diseased state of periodontium. Moreover, on an average poor glycemic control was also reported in our diabetic study subjects. There is evidence in literature that periodontal infection once established may lead to adversely affecting the glycemic control status in diabetics which may lead to diabetes related complications.[5] The highly vascular inflamed periodontium acts like a reservoir for tumor necrosis factor (TNF-alpha) and other inflammatory mediators like Interleukin: IL-1 6 and IL-1.[34,35] These Interleukin mediators have shown to have effects on glucose and lipid metabolism.[35-37] TNF-alpha, IL-1 and IL-6 are known to be insulin antagonist.[10,37,38] Gram negative anaerobic bacteria specially *Porphyromonas gingivalis* is an etiologic agent of PD. Lipopolysaccharides (LPS) from *P. gingivalis* are known to induce IL-1, TNF-alpha, prostaglandin (PGE2) and matrix metalloproteinase's (MMP's). Persistent rise of IL-1 6, IL-1 and TNF-alpha have effects on liver and pancreatic beta cells. Thus, chronic periodontal inflammation may exaggerate the existing elevated cytokine status in diabetic patients leading to increased severity and complicating glycemic

controls.[39] This is well supported by documentary evidence from the study Collin in which mean glycosalated hemoglobin increased to 0.5% in type 2 diabetic patients with severe periodontitis over a period of 2 to 3 years as compared to mean glycosalated hemoglobin reduction of 0.9% in diabetic patients with less or no periodontal disease.[40] Similarly a study by Nelson in 1990 on pima Indians evidenced that severe periodontitis at baseline had poor glycosalated hemoglobin levels as compared to those with minimum or no periodontitis after a 2 years follow-up. Sufficient literature is available to evidence the bidirectional nature of diabetes and periodontitis.[14]

However, we feel that limited sample size was one of the limitation of this study which could be overcome by having a larger sample size for better statistical analysis in future. Microbiological estimation of pro inflammatory cytokine markers like TNF-alpha, IL-1, IL-6 to evaluate the level of periodontal inflammation with reference to gender and age and its prospective long term effect on glycosalated hemoglobin may be undertaken in prospective studies. Advanced radiological assessment tools to evaluate alveolar bone destruction may be incorporated in long term studies.

CONCLUSION

Within limitations of the study it may be safely concluded that assessment of periodontal status of Diabetes Mellitus patients revealed chronic periodontal destruction particularly in age groups beyond 40 years in majority of study population depicting that Age is significantly associated with the increased prevalence and severity of periodontal disease in patients with diabetes. Further, such patients may present with increased severity of diabetes along with altered complicating glycemic controls possibly due to sustained chronic systemic inflammation due to periodontitis. Thus, regular oral health and periodontal status assessment and treatment planning of diabetic patients is required to lower their risk of developing severe PD and any systemic complications emanating as its outcome.

ACKNOWLEDGEMENTS

REFERENCES

1. Mealey BL, Oates TW. Diabetes mellitus and periodontal diseases. J periodontol. 2006;77(8): 1289-303.

2. Papatheodorou K, Banach M, Bekiari E, Rizzo M, Edmonds M. Complications of diabetes 2017. J Diabetes Res. 2018;2018:3086167.
3. Shetty S, Kohad R, Yeltiwar R, Shetty K. Gingival blood glucose estimation with reagent test strips: a method to detect diabetes in a periodontal population. J periodontol. 2011;82(11):1548-55.
4. Apoorva SM, Sridhar N, Suchetha A. Prevalence and severity of periodontal disease in type 2 diabetes mellitus (non–insulin dependent diabetes mellitus) patients in Bangalore city: An epidemiological study. J Indian Soc. Periodontal. 2013;17(1):25-9.
5. Al Shwaimi E, Idrees M, Berri Z, El-Sakka H, Kujan O. Association between diabetes mellitus and periodontal diseases: a survey of the opinions of dental professionals. Med Princ Pract. 2019;28 (2):141-9.
6. Mauri-Obradors E, Estrugo-Devesa A, Jané-Salas E, Viñas M, López-López J. Oral manifestations of Diabetes Mellitus. A systematic review. Med Oral Patol Oral Cir Bucal. 2017;22(5):e586-94.
7. Mohamed HG, Idris SB, Ahmed MF, Bøe OE, Mustafa K, Ibrahim SO, et al. Association between oral health status and type 2 diabetes mellitus among Sudanese adults: a matched case-control study. PloS one. 2013;8(12):e82158.
8. Acharya P, Kumar M, Saimbi CS. Clinical evaluation of topical metronidazole and chlorhexidine gel following scaling and root planing in patients with chronic periodontitis. J Col of Med Sci-Nepal. 2019;15(1):10-7.
9. Ezzo PJ, Cutler CW. Microorganisms as risk indicators for periodontal disease. Periodontol. 2003;3:24-35.
10. Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiologic perspective. Ann Periodontol. 2001; 6(1):99-112.
11. Rajhans NS, Kohad RM, Chaudhari VG, Mhaske NH. A clinical study of the relationship between diabetes mellitus and periodontal disease. J Indian Soc of Periodontol. 2011;15(4):388-92.
12. Beltrán-Aguilar ED, Eke PI, Thornton-Evans G, Petersen PE. Recording and surveillance systems for periodontal diseases. Periodontol. 2012;60(1): 40-53.
13. Oral health surveys basic method, World Health Organization. Available at https://apps.who.int/ iris/handle/10665/41905. Accessed on 20 July 2020.
14. Nelson RG, Shlossman M, Budding LM, Pettitt Dj, SaadMF, Genco RJ, et al. Periodontal disease and NIDDM in pima Indians. Diabetes Care. 1990;13 (8):836-40.
15. Albander JM, KingmanA. Gingival recession, gingival bleeding, and dental calcus in adults 30 years of age and older in the United States, 1988-1994. J Periodontol. 1999;70(1):30-43.
16. Darre L, Vergnes JN, Gourdy P, Sixou M. Efficacy of periodontal treatment on glycaemic control in diabetic patients:A meta-analysis of interventional studies. Evid based Dent. 2009;10:20-1.

17. Loe H. Periodontal disease the sixth complication of diabetes mellitus. Diabetes care. 1993;16(1):324-34.
18. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care. 2006;29:543-8.
19. Emrich LJ, Shlossman M, GenjoRJ.Periodontal disease in non insulin dependent Diabetes mellitus. J Periodontol. 1991;62(2):123-31.
20. Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M, Knowler WC, et al. Non-insulin dependent diabetes mellitus and alveolar bone loss progression over 2 years. J Periodontol. 1998;69(1): 76-83.
21. Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, et al. Assessment of risk for periodontal disease, Risk indicators for attachment loss. J Periodontol. 1994;65(3):260-7.
22. Dolan TA, Gilbert GH, Ringelberg ML, Legler DW, Antonson DE, Foerster U, et al. Behavioral risk indicators of attachment loss in adult Floridians. J Clin Periodontol. 1997;24(4):223-2.
23. Burt BA, Ismail AI, Eklund SA. Periodontal disease, tooth loss, and oral hygiene among older Americans. Community Dent Oral Epidemiol. 1985;13(2):93-6.
24. Johnson ES, Kelly JE, Van Kirk LE. Selected dental findings in adults by age, race and sex, United states,1960-1962. Vital Health Stat 11. 1965;11:1-35.
25. Schulze A, Busse M. Gender differences in periodontal status and oral hygiene of non-diabetic and type 2 diabetic patients. Open Dent J. 2016; 10:287-97.
26. Page RC. Oral health status in the United States: Prevalence of inflammatory periodontal disease. J Dent Educ. 1985;49(6):354-67.
27. Beck JD, Katz RV, Page RC, Greene JC, Löe H. Oral health of US adults: NIDR 1985 national survey. J Public Health Dent. 1987;47(4):198-205.
28. Haghighatpanah M, Nejad ASM, Haghighatpanah M, Thunga G, Mallayasamy S. Factors that correlate with poor glycemic control in type 2 diabetes mellitus patients with complications. Osong Public Health Res Perspect. 2018;9(4):167-74.
29. American Diabetes Association. Glycemic targets in Standards of Medical Care in Diabetes-2017. Diabetes Care. 2017;40(1):S48–S56. Available at: https://care.diabetesjournals.org/content/40/Supplement_1/S48. Accessed on 2 September 2020.
30. Fasil A, Biadgo B, Abebe M. Glycemic control and diabetes complications among diabetes mellitus patients attending at University of Gondar Hospital, Northwest Ethiopia. Diabetes Metab Syndr Obes. 2018;12:75-83.
31. Awartani FA. Evaluation of the relationship between type 2 diabetes and periodontal disease. Saudi Med J. 2009;30(7):902-6.
32. Ohtake T, Takahashi R, Ohyabu Y, Minamisono N, Kuzuyama T, Oshishi K, et al. A study on community periodontal index of treatment needs

(CPITN) in type 2 diabetic patients. J Jpn Soc Periodontal. 2005;47(1):28-35.

33. Ueno M, Takeuchi S, Oshiro A, Shinada K, Ohara S. Association between diabetes mellitus and oral health status in Japanese adults. Int J Oral Sci. 2010;2(2):82-9.

34. Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: a two-way relationship. Ann Periodontol. 1998;3(1):51-61.

35. Offenbacher S, Katz V, Fertik G, Collins J, Boyd D, Maynor G, et al. Periodontal infection as a possible risk factor for preterm low birth weight. J Periodontol. 1996;67:1103-13.

36. Feingold KR, Soued M, Serio MK, Moser AH, Dinarello CA, Grunfeld C. Multiple cytokines stimulate hepatic lipid synthesis in vivo. Endocrinology. 1989;125(1):267-74.

37. Ling PR, Istfan NW, Colon E, Bistrian BR. Differential effects of interleukin-1 receptor antagonist in cytokine- and endotoxin-treated rats. Am J Physiol. 1995;268(2.1):E255-61.

38. Feingold KR, Grunfeld C. Role of cytokines in inducing hyperlipidemia. Diabetes. 1992;41(2):97-101.

39. Grossi SG. Treatment of periodontal disease and control of diabetes: an assessment of the evidence and need for future research. Ann Periodontol. 2001;6(1):138-45.

40. Collin HL, Uusitupa M, Niskanen L, Kontturi-Närhi V, Markkanen H, Koivisto AM, et al. Periodontal findings in elderly patients with non-insulin dependent diabetes mellitus. J Periodontol. 1998;69(9):962-6.