Journal of Advanced Medical and Dental Sciences Research
@Society of Scientific Research and Studies

Journal home page: www.jamdsr.com doi: 10.21276/jamdsr UGC approved journal no. 63854

(e) ISSN Online: 2321-9599; (p) ISSN Print: 2348-6805

Original Article

Assessment of Impaired Glucose Tolerance in Chronic Periodontitis Patients– A Case Control Study

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***ABSTRACT*:**
The treatment of periodontal disease in impaired glucose tolerance and diabetic patients is reported to have a beneficial effect on their glucose control. Studies have indicated that periodontal diseases and diabetes mellitus are closely associated and are highly prevalent chronic diseases with many similarities in pathobiology. These findings suggest diabetes increases the risk of periodontaldiseases, and biologically plausible mechanisms have been demonstrated inabundance. As inflammatory disease, periodontitis may increase insulinresistance in a way similar to obesity, thereby aggravating glycemic control. **Methods:** Total 60 patients of age group 35-55 years with the diagnosis of chronic generalized periodontitis were screened for the purpose of the study using clinicaland radiographic parameters. Oral glucose tolerance was assessed in systemically healthy patients withchronic periodontitisfollowing which, 8 patients were excluded from study as they did not fall under the category of NGT(Non-glucose tolerant) OR IGT(Impaired glucose tolerance). Out of Remaining 52 patients who fulfilled our desired criteria were (23 IGT and 29 NGT) randomly we picked 20 each and assigned them to Group I and Group II respectively (20 each) andenrolled for the study. The relationships between the mean ratio of probing pocket depth, clinical attachment loss, alveolar bone loss and glucose tolerance results were analyzed. **Results:** There was statistically significant differencein mean Probing depth between group I and group II (P=0.001) (i.e. 57.460± 26.79% against 30.595 ± 14.15 %.) The proportion of subjects with IGT increased significantlyin the subjects with higher tertiles of alveolar bone loss (P <0.05). In the study, we found a significant difference in Clinical attachment loss (P=0.009) in group I and II (i.e. 30.879 ± 14.07% compared to 18.668 ±1 4.18% and itsrange was from 7.20% to 57.14%). It showed good correlation with the values of glycaemic control and Clinical Attachment Loss. Analysis revealed that group I patients had Mean Alveolar Bone Loss slightly more as compare to groupII patients (i.e. 26.748±12.86% compared to 21.203±5.24%. There was statistically no significant difference in meanAlveolar Bone Loss between groups. (P=0.082). **Conclusion:** The degree of probing pocket depth, clinical attachment loss is associated with IGT, suggesting that periodontitis is associated with impaired glucose tolerance.
Key words: Diabetes; Impaired glucose tolerance; periodontal disease; risk factor.

Received: 1 February, 2019 Revised: 20 February, 2019 Accepted: 23 February, 2019

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This article may be cited as: Pippal D, Kawadkar A, Shrivastava R, Sakalle D, Jain M, Goutam M. Assessment of Impaired Glucose Tolerance in Chronic Periodontitis Patients– A Case Control Study. J Adv Med Dent Scie Res 2019;7(2):95-100.

INTRODUCTION :

According to International Diabetes Federation, approximately 285 million had diabetes in 2010 which will rise to438 million by 2030.Currently, it represents 6.4% of the world and 24%of Indian adult population that mean every fifth person with diabetes will be anIndian. Surveys reported that the prevalence of IGT in the Indian subcontinent is8.7% in urban and 7.9% in rural areas. The prevalence of diabetes is increasingannually in India and varies by age and racial category.About 85% to 90% ofdiabetic cases are type 2 diabetes, whereas type 1 diabetes constitutes 5%to 10%of patients.[(1)]

Some individuals have glucose levels that do not meet the criteria for diabetes but are too high to be considered normal. Members of this group have a conditioncalled ‘‘prediabetes,’’ a term which encompasses both impaired fasting glucose(IFG) and impaired glucose tolerance (IGT) introduced in 1979 bythe National Diabetes Data Group and agreed by the World Health Organization.[(2)]

EFFECTS OF DIABETES ON THE PERIODONTIUM

Examination of the available data reveals strong evidence that diabetes is arisk factor for gingivitis and periodontitis, and the level of glycemic controlappears to be an important determinant in this relationship. [(3)]Poor metabolic control can increase the severity of gingival inflammation indiabetics, whereas improvement in

glycemic control may be associatedwith decreased gingival inflammation. [4]

MECHANISMS BY WHICH DIABETES MAY INFLUENCE THEPERIODONTIUM:

A large evidence base is available to describe these potentialmechanisms, many of which are strikingly similar to those associated with theclassic diabetic complications, including retinopathy, nephropathy, neuropathy,macrovascular diseases, and altered wound healing. The strength of the evidence has led some to suggest that periodontitis should be listed among the ''classic''complications of diabetes.[5]

The function of immune cells, including neutrophils, monocytes, andmacrophages, is altered in diabetes. Neutrophil adherence, chemotaxis, andphagocytosis are often impaired, which may inhibit bacterial killing in theperiodontal pocket and significantly increase periodontal destruction.[6]

OBJECTIVES:

- To compare Group I with Group II for the level of glucose tolerance.
- To compare Group I and Group II for the alveolar bone loss(radiographically).
- To compare Group I and Group II for Clinical attachment loss.
- To compare Group I and Group II for PPD (Probing pocket depth).

MATERIAL AND METHODS:

Study Population:

The present study was designed as a double blind, non-interventional, cross-sectional, case Control study and the subjects were the patients visiting Department of Periodontics, Rishiraj College of Dental Sciences and ResearchCentre, Bhopal. All the subjects in the study were matched for age and otherparameters. The study was carried out over a period of 6 months after Ethicalclearance was obtained from the Ethical Committee of Rishiraj College of Dental Sciences and Research Centre, Bhopal.

Out of 60 examined patients, 8 wereexcluded on the basis of not falling under the NGT or IGT category. Remainingpatients who fulfilled our desired criteria were (23 IGT and 29 NGT) thenrandomly picked and assigned to Group I (IGT) and Group II (NGT) respectively (20 each) andenrolled for the study.

Examination of Systemic Condition:

The patient were made to observe fast for 9-16 hours (observing fast from 9pmon the previous evening) prior to the test. The following day, collected simulated plasma samples frompatients (who have fasted for minimum 9 hours), taken immediately before drinking a glucose solution, later after two hours. Glucose concentration in blood plasma isanalyzed by time taken for the glucose to decolorize potassium manganate (VII) solution.[7] Glucose reduces purple-pink manganate ions to colourless manganese ions and water.

$MnO4^-(aq) + 8H^+(aq) + 5e^- \rightarrow Mn^{2+}(aq) + 4H_2O(l)$

Overnight Fasting and Fasting 2 Hr Glucose

- Normal = Overnight Fasting ≤ 110 mg/dlor 2hr value ≤ 140 mg/dl
- Diabetes = Overnight Fasting ≥ 140 mg/dl or 2 hr value ≥ 200 mg/dl
- Impaired Fasting Glycaemia = Overnight Fasting 110 - 125 mg/dlor2 hr value< 140 mg/dl
- Impaired glucose tolerance = Overnight Fasting <126 mg/dl or2 hr value between 140-200mg/dl

Examination of Clinical Procedure

Clinical finding were recorded using A UNC 15 Probe for marking the pocketdepth and clinical attachment loss. A maximum of 28 sites (7 sites x 4 quadrant)were measured in each patient. This procedure was followed for both the groups, i.e. Chronic periodontitis patients with Impaired Fasting and normalGlucose Tolerance.

INCLUSION CRITERIA:

1. At least ≥ 16 Teeth are selected (4 Teeth in Each Quadrant).
2. At least ≥ 3 mm Probing Pocket Depth (PPD) are selected in more than30% Sites.
3. At least ≥ 5 mm Clinical Attachment Loss in more than 30% sites.
4. Radiographic evidence of bone loss by using Intra Oral Periapical Radiograph (IOPA).

Probing pocket depths, Clinical Attachment Loss and alveolar bone loss (radiographic) were measured. Each patient was probed in 7 areas:

1. Mesial and distal sites of 1st & 2nd Premolars.
2. Mesial and distal sites of 1st Molar.
3. Only Mesial sites of 2nd Molar (distal sites of second molars wereexcluded to eliminate the influence of third molars, which were frequentlyimpacted).

One investigator measured alveolar bone loss of Chronic GeneralizedPeriodontitis Patient on (IOPA) X-Ray films using (Vernier Caliper) a rulergraduated to 0.5 mm for measuring the ratio of a length from the cemento-enamel junction to the alveolar bone adjacent to the root surface to that of the length fromthe cement-enamel junction to the root apex.

Clinical attachment loss was measured from the cemento-enamel junction to the base of the pocket. Probing pocket depths were measured from the crest of the gingival margin to thebase of the pocket

STATISTICAL ANALYSIS:

Statistical analysis was done using Statistical Package of Social Science (SPSS Version20; Chicago Inc., USA).Quantitative variables were compared using mean values and qualitative variables using proportions.Significance level was fixed at $P < 0.05$. Chi-

square testwas used for non-parametric. The Student's t-test was used to analyze the variation in mean between two groups ofa variable with a normal distribution

RESULTS:

Overnight fasting glucose level(mg/dl): Among group I patients Mean Overnightfasting glucose level was more than normal i.e 118.96 ± 12.29 whileamong group II patients it was 99.46 ± 7.35. Its range was from 83.30 to163.30 mg/dl. There was statistically highly significant difference in meanOvernight fasting glucose level between group I & group II.(P=0.001).

Comparison of mean Overnight fasting glucose level (mg/dl) between group I & group II chronic Periodontitis patients. TABLE-1

Groups	Mean Overnight fasting glucose level (mg/dl)		
	Mean	SD	Range
Group I (Impaired Glucose Tolerance)	118.96	12.29	106.0 - 163.30
Group II (Normal Glucose Tolerance)	99.46	7.35	83.30 - 107.0
TOTAL	109.21	14.04	83.30- 163.30
Student 't' test value	6.087		
Significance 'P' Value	0.001(HS)		

2HrsPost Challenge glucose level(mg/dl): Among group I patients,Mean 2Hrs PostChallenge glucose level was more than normal i.e 158.98 ± 20.50 whileamong group II patients it was 120.60 ± 10.31. Its range was from 103.0 to196.60 mg/dl. There was statistically highly significant difference in mean2Hrs Post Challenge glucose level (mg/dl) between group I & group II .(P=0.001) .

Comparison of mean 2Hrs Post Challenge glucose level (mg/dl) between group I & group II chronic Periodontitis patients.TABLE-2

Groups	Mean 2Hrs Post Challenge glucose level (mg/dl)		
	Mean	SD	Range
Group I (Impaired Glucose Tolerance)	158.98	20.50	134.0 – 196.60
Group II (Normal Glucose Tolerance)	120.60	10.31	103.0 – 137.0
TOTAL	139.79	25.18	103.0 - 196.60
Student 't' test value		7.478	
Significance 'P' Value		0.001(HS)	

Mean Probing depth (%): Among group I patients Mean probing depth (%)was more as compare to normal group II patients i.e 57.460 ± 26.79while among group II patients it was 30.595 ± 14.15. There was statistically highly significant differencein mean Probing depth (%) between group I & group II. (P=0.001).

Comparison of mean probing depth (%) between group I & group II Chronic Periodontitis Patients.TABLE-3

Groups	Mean Probing depth (%)		
	Mean	SD	Range
Group I (Impaired Glucose Tolerance)	57.460	26.79	14.28 – 100.0
Group II (Normal Glucose Tolerance)	30.595	14.15	10.71- 59.25
TOTAL	44.37	25.27	10.71 -100.0
Student 't' test value		3.885	
Significance 'P' Value		0.001(HS)	

Mean Clinical Attachment Loss (%): Among group I patients, Mean ClinicalAttachment Loss (%) was more as compare to group IIpatients i.e. 30.879 ± 14.07whereas it was18.668 ±14.18. Its range was from 7.20% to 57.14%.

There wasstatistically significant difference in mean Clinical Attachment Loss (%)between group I & group II. (P=0.009).

Comparison of mean Clinical Attachment Level (%) between group I & group II chronic Periodontitis patients.TABLE-4

Groups	Mean Clinical Attachment Level (%)		
	Mean	SD	Range
Group I (Impaired Glucose Tolerance)	30.879	14.07	10.71 - 57.14
Group II (Non Glucose Tolerance)	18.668	14.18	7.20 - 72.0
TOTAL	24.773	15.25	7.20 – 57.14
Student 't' test value		2.733	
Significance 'P' Value		0.009(S)	

Alveolar Bone Loss (%): Among group I patients Mean Alveolar Bone Loss (%) wasslightly more as compare to normal group II patients i.e 26.748 ± 12.86 whileamong group II patients it was 21.203 ± 5.24. Its range was from 12.62% to54.93%. There was no significant difference found in mean AlveolarBone Loss (%) between group I & group II. (P=0.082).

Comparison of mean Alveolar Bone Loss (%) between group I & group II chronic Periodontitis patients.TABLE-5

Groups	Mean Alveolar Bone Loss (%)		
	Mean	SD	Range
Group I (Impaired Glucose Tolerance)	26.748	12.86	12.62- 54.93
Group II (Non Glucose Tolerance)	21.203	5.24	13.94 – 30.88
TOTAL	23.975	10.09	12.62- 54.93
Student 't' test value		1.785	
Significance 'P' Value		0.082(NS)	

DISCUSSION:

T. Saito et al in the year 2004[8] explained the relationship between periodontal condition and glucose tolerance in 2180 Hisayama residents aged 40 to 79 years in Japan. They reported a significant relationship between diabetes and PPD. Subsequent study done by M. Duarte et alin 2007[9]evaluated the changes associated with type 2 diabetes and moderate-to-severe chronic periodontitis (probing depth >6 mm, n 20). They too found a significant relationship (p >0.001) between diabetes and PPD and our study was in accordance with this. Similarly, Awartani F et al (2009) [10]investigated the association between glycemic control of type 2 diabetes mellitus (type 2 DM) and severity of periodontal disease. Their results showed a significant association between the PD ≥4 mm and poorly controlled diabetic patients as compared to better-controlled patients. We also found a highly significant association (p >0.001) between probing pocket depth in NGT patients compared to IGT patients.Comparison of mean Probing depth between male & female chronic Periodontitis patients revealed females having more PPD compared to male patients (i.e. 45.730 ± 26.366% against 43.209 ± 24.89%) but statistically, no significant difference in mean Probing depth was found between male & female chronic periodontitis patients. (P=0.761).

P. Engebretson et al in the year 2004[11] demonstrated that glycemic control and GCF levels of IL-1β had significant positive correlation with mean clinical attachment loss (P=0.001).

Faria-Almeida R et al (2006) [12] Conducted a prospective longitudinal study to compare type 2 diabetics and non-diabetics for level of clinical attachment, and gingival recession and found significant association between type 2 diabetes, CAL and gingival recession.

Navarro-Sanchez et al in 2007[13] reported higher mean percentage of sites with a depth of 4–6mm in the diabetic group versus controls and concluded that the clinical improvements were obtained accompanied by a significant reduction in HbA1C values in type 2 diabetic subject .Another study by Fernanda O.B. Correa et al (2008) [62] along similar lines reported that patients with Type II Diabetes Mellitus showed significantly worse clinical parameters than the control group at baseline, there was a significant improvement in the clinical condition following periodontal therapy. Awartani F et alin 2009 [10] investigated the association between glycemic control of Type 2 Diabetes Mellitus and severity of periodontal disease. Their results showed a significant association between the loss of attachment (3-4 mm) as well as more calculus in poorly controlled diabetic patients with clinical attachment loss as compared to better-controlled patients.

Perayil J et al (2014) [14] compared HbA1c levels in individuals without diabetes and with and without periodontitis. They demonstrated a statistically significant

difference in CAL (P < 0.05) in their study between the two groups.

In our study,we found a significant difference in group I and II for CAL (i.e. 30.879 ± 14.07% compared to 18.668 ±1 4.18% and its range was from 7.20% to 57.14%). There was statistically significant difference in mean Clinical Attachment Loss between group I & group II subjects. (P=0.009) which was in accordance with previously done studies.

Alveolar Bone Loss was the third parameter we evaluated using laboratory diagnosis. The comparison among the groups showed group I patients had Mean Alveolar Bone Loss slightly more as compare to group II patients (i.e. 26.748±12.86% compared to 21.203±5.24%. Its range was from 12.62% to 54.93%). There was statistically no significant difference in mean Alveolar Bone Loss between group I & group II patients. (P=0.082). Among female patients, mean Alveolar Bone Loss was more as compared to male patient's (i.e. 24.967±10.26% in female and 23.164±10.26% in male). Though there was statistically no significant difference found in mean Alveolar Bone Loss between male & female chronic periodontitis patients. (P=0.581). Robert G et al (1990)[15] conducted a study to determine the prevalence and incidence of periodontal disease and its relationship with non-insulin-dependent diabetes mellitus (NIDDM) in 2273 Pima Indians by percentage of interproximal crestal alveolar bone loss ascertained from panoramic radiography. They reported a significant result. Whereas in our study, there was statistically non-significant difference in mean Alveolar Bone Loss between group IGT and NGT groups with chronic Periodontitis (P=0.082). Similarly, Grossi et al (1994)(29) reported age as the primary factor associated with severity of radiographic bone loss. The relative risks for the different age categories increased from age 35 to 44 to a risk at age 65 to 74. However, no significant differences were observed in the number of sites radiographic bone loss in a case-control study of individuals 19 to 25 years old with and without diabetes whereas, age was not a differentiating criterion for our study.

Another study done by Tervonen T et al (2000), [16] assessed the degree/ level of marginal alveolar bone loss on panoramic radiographs of maxillary and mandibular molars as the % of the distance between the cemento-enamel junction (CEJ) and the bone crest along the total length of the root in a group of young subjects with type 1 diabetes mellitus (DM) (age range 24–36 years) on 35 individuals. They showed a significant (p>0.05) marginal bone loss in the subjects with complicated DM. They concluded that type 1 DM has a modifying effect on marginal loss of alveolar bone. In another study Lappin DF et al (2009) [17] determined plasma concentrations of bone metabolism marker (nuclear factor-kB ligand (RANKL), osteoprotegerin (OPG), C-terminal telepeptide of type 1 collagen and osteocalcin in type 1 diabetes mellitus patients and non-diabetic and the influence of periodontitis on biomarkers of bone formation in type 1 diabetes mellitus patients (n=563) and nondiabetics (n=538). They concluded that the diabetics were having highly significant values for bone loss biomarker compared to nondiabetic periodontitis patients. (p=0.001). Similarly, Fawad Javed et al in (2014)[18] assessed the effects of glycemic control on self-perceived oral health, periodontal parameters, and marginal bone loss (MBL) in patients with prediabetes in 303 individuals (196 males and 107 females, aged 39 to 46 years). There was significant difference (P < 0.001) in periodontal parameters and marginal bone loss among patients with prediabetes and healthy controls. They concluded that the severity of periodontal parametersand marginal bone loss are worse in patients with prediabetes than controls. Glycemic control significantly reduces the severity of these parameters as well as the state of prediabetes in affected individuals. On the same note,Ji-Hye Kim et al in 2014 [19] examined the effect of the interaction between periodontitis and type 1 diabetes mellitus on alveolar bone, "trabecular bone volume fraction" (BVF) of mandibular condyle and tibia in animals. They reported non-significant alveolar bone loss in the diabetic group which was slightly increased compared with that of the normal group. The results were similar to our study. Subsequent study done by Niels-Christian Reimers Holm et al in (2016)[20] on 291 adults with undiagnosed DM or prediabetes where periodontal examination was performed and radiographic bone were level measured along with HbA1c levels. They found higher proportions of patients with undiagnosed DM and prediabetes in the periodontitis group than in the control group (P = 0.054). They concluded that individuals with undiagnosed DM and prediabetes at early stages of disease, can be identified in the dental office by chairside recordings of HbA1c levels, which may prevent future complications.

CONCLUSION:

Periodontal diseases and diabetes mellitus are closely associated and are highlyprevalent chronic diseases with many similarities in pathobiology. Related antecedent conditions including obesity and insulin resistance are known play animportant role in this relationship. Diabetes increases the risk of periodontaldiseases, and biologically plausible mechanisms have been demonstrated inabundance. As inflammatory disease, periodontitis may increase insulinresistance in a way similar to obesity, thereby aggravating glycemic control. Lessclear is the impact of glycemic control of diabetes on non-diabetes i.e. impairedglucose tolerance and normal glucose tolerance and the mechanisms throughwhich it affects the progress of periodontal disease with an attempt to decipherthe same, we designed a case-control study but with limited resources anddifferent constraints we could not reach a concrete conclusion.

Hereby, we would like to accept the limitation of our study and would like tosuggest further research, to clarify this aspect of the relationship betweenperiodontal diseases and impaired glucose tolerance. Literature is scant when itcomes to Impaired glucose tolerance and normal glucose tolerance andprospective, rigorous, controlled trials with a larger number of patients, in ethnically diverse populations are warranted to establish

these relationshipsbetween Impaired glucose tolerance and normal glucose tolerance and prediabetesand its relation with periodontal disease.

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