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Original Research Article

# Effects of non-surgical periodontal therapy (NSPT) in prediabetes patients with periodontitis - A randomised control trial

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ABSTRACT

**Aim:** To clinically and microbiologically evaluate the association of periodontitis and pre-diabetes.
**Materials and Methods:** The trial was designed as a randomized controlled clinical trial with a sample size of total of 100 with 1:1 gender ratio. Test group taken were patients with chronic periodontitis with prediabetes and Control group were patients with periodontitis without prediabetes. Body mass index (BMI), Periodontal Probing Depth (PPD),Bleeding on probing (BOP),Clinical Attachment Loss(CAL) using UNC colour coded periodontal probe were recorded. The microbial load in pre-diabetes patients was assessed using samples collected from the periodontal pockets ≤5mm. Fasting Blood sugar (FBS), Post Prandial blood sugar (PPBS), Fasting Insulin were recorded at baseline, 6 weeks,12 weeks and after Non-surgical periodontal therapy (SRP) after taking consent from the patient. The data collected were entered into Microsoft excel 2018. Statistical analysis was done using IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY: IBM Corp. Descriptive analyses were done. Student's t-test, repeated measures of ANOVA were used for determination of the significance of HOMA-IR, HOMA- β mean differences between and within the groups.
**Results:** In both test and control groups, there was a significant difference ($p<0.0001$) in probing depth, CAL, gingival bleeding index from baseline to the post treatment (NSPT) till 12 week period. When these clinical parameters were evaluated and correlated with HOMA-IR, HOMA-β, BMI, FBS, post prandial blood sugar at regular intervals, significant ($p<0.0001$) decrease in HOMA-IR, HOMA-β, BMR, post prandial blood sugar was observed in test group when compared to control group.
**Conclusion:** There is a significant association between prediabetes state and periodontitis. Early diagnosis of periodontitis and a proper treatment in prediabetic group can prevent the progression of prediabetic condition to diabetes and vise versa.



## 1. Introduction

Diabetes mellitus is a highly prevalent metabolic disorder, constituting a serious public health burden. A predicted 300 million people are going to suffer from diabetes by 2025.[1] India has one of the highest incidence rates for diabetes with more than 62 million suffering from diabetes and a projected increase to 80 million by 2030.[2]

Pre diabetes means your blood glucose levels are higher than normal but not high enough to be diagnosed as diabetes. Pre diabetes usually occurs in people who already have some insulin resistance or whose beta cells in the

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pancreas aren't making enough insulin to keep blood glucose in the normal range. Without enough insulin, extra glucose stays in bloodstream rather than entering your cells and over time, patient can develop type 2 diabetes.[2]

Hyperglycemia is known to favor pro-inflammatory priming of periodontal tissues thus contributing to the earlier onset of gingivitis, the initial and reversible stage of periodontitis, in patients with diabetes.[3] Periodontitis elevates the systemic inflammation leading to a state of insulin resistance precipitating into hyperglycemia and advanced glycation end product accumulation.[4]

Persistent hyperglycemic state and advanced glycation end product besides causing the macrovascular and microvascular complications of diabetes also result in the impaired gingival fibroblast synthesis, defective phagocytic activity of mononuclear and polymorphonuclear cells.[5] This may result in the loss of periodontal fibers and supporting alveolar bone.[5] These findings led to consider periodontitis as the sixth complication of diabetes mellitus.[6] The association between periodontal disease and diabetes has been explored in several studies over the years, and it is generally accepted that periodontal disease is more prevalent and more severe in persons with diabetes than in non diabetic persons.

The impact of Diabetes mellitus on periodontal health has been addressed in numerous cross-sectional and longitudinal studies and Diabetes mellitus is currently considered an established risk factor for Periodontal disease. Although there is significant heterogeneity among these studies in design and definition of periodontal disease, increasing evidence suggests that poorly controlled Diabetes mellitus correlates with higher prevalence, severity, and progression rate of Periodontal disease when compared to controlled Diabetes mellitus or health.[7–11]

CRP is an acute-phase protein mainly produced by the liver in response to infection or tissue damage.[12] Connecting peptide (C-peptide), produced in equal amounts to insulin, is known to be a useful marker of beta-cell function and can be used to assess endogenous insulin secretion.[13] Obesity (body-mass index [BMI]≥30 kg/m2) is the strongest risk factor for Type 2 Diabetes mellitus and is associated with metabolic abnormalities resulting in insulin resistance.[6] Insulin and C-peptide responses to oral glucose load are found significantly higher in smokers than non-smokers. In addition to increased insulin resistance, smoking also showed dyslipidaemia and make subjects prone to atherosclerosis. Smokers had higher fasting triglycerides and lower high density lipoprotein cholesterol levels, and an increased proportion of small dense low density lipoprotein particles. Fibrinogen levels and plasminogen activator inhibitor 1 activity were also seen elevated in smokers.[6]

In the present study, the association of periodontitis and pre-diabetes was evaluated clinically and microbiologically. Clinically, the probing depth, CAL, Bleeding on Probing were evaluated at baseline and postoperatively (NSPT-Non Surgical Periodontal Therapy) at 6 weeks and 12 weeks of intervals. The microbial load in pre-diabetes patients was assessed using samples collected from the periodontal pockets ≤5mm by gram staining. The effect of periodontal therapy in pre-diabetes conditions was assessed using Homeostasis Model assessments of IR and β-cell function (HOMA-IR and HOMA-β). BMR, FBS, post prandial blood sugar, Fasting insulin.

## 2. Materials and Methods

The study was designed as a randomized controlled clinical trial to clinically study the relationship between pre-diabetes mellitus and periodontitis by evaluating HOMA-IR & HOMA-β .Subjects for the study were selected from Out-Patient section of the Department of Periodontology, MNR Dental College & Hospital, Sangareddy District, Telangana and were followed for 6 weeks and 12 weeks period after the post NSPT as baseline. The current randomised clinical trial was done on 100 participants. Systemically healthy (Non-diabetic) male and female patients of age group 20-50 years with complaints of periodontal disease having probing pocket depth of ≤5mm and CAL ≤4mm were included in the study. Medically compromised patients, subjects who underwent radiotherapy or chemotherapy in the past 12 months, patients having uncontrolled periodontal disease, having ≤15 teeth and Pregnant and lactating women were excluded.

### *2.1. Sample size calculation*

Sample size calculation was performed using G power 3.1 software. The calculations were based on the probability of Pre diabetes between Periodontitis and Non-Periodontitis groups with an alpha level of 0.05, a desired power of 80%. The estimated sample size was 84 subjects. Considering 20% loss of follow up before completion of the trial, a total of 100 (50 in each group) subjects were included in the study. The study was designed as a single blind, randomized controlled clinical trial where in the relationship between pre-diabetes mellitus and periodontitis was studied by evaluating HOMA-IR & HOMA-β with following groups: Test group: patients with chronic periodontitis with prediabetes and Control group: patients with chronic periodontitis without prediabetes This blind was not broken until this clinical trial was completely finished at the end of 12 weeks.

PPD and CAL, Bleeding on probing were recorded at the baseline, at the end of 6 weeks and 12 weeks using a UNC-15 color-coded periodontal probe. An alginate impression was taken and custom acrylic stent limited to the occlusal $2/3^{rds}$ of the clinical crown were used as fixed reference position (i.e. junction of vertical groove and lower border of

the stent). A groove was prepared in the stent to standardize the probing angulation throughout the study period.

### 2.2. Patient protocol

Each patient was prepared for the study with NSPT (Non Surgical periodontal Therapy) which included scaling and root planing (SRP), occlusal adjustment and oral hygiene instructions. On completion of the initial examination and thorough phase-I therapy, the patients were reassessed at 6 weeks and 12 weeks.

Blood samples were collected from the patient and the parameters - Fasting blood glucose, post prandial blood sugar, Fasting insulin are measured at baseline, 6 weeks and 12 weeks.

### 2.3. HOMA-IR and HOMA-β cell function

The homeostasis model assessment index, HOMA-IR and HOMA-β were used for measurement of insulin resistance and beta cell function (insulin secretion capacity), respectively. Both indexes were calculated using the following formula:

HOMA-IR = Fasting plasma glucose (mg/dL) × Fasting insu lin (mIU/L)/ 05

HOMA-β = 360 × Fasting insulin (mIU/L)/ [Fasting plasma glucose (mg/dL)-63].

### 2.4. Microbiological assessment

Plaque samples are collected from mesiobuccal pocket (PPD 5-7mm) and gram staining was done.

### 2.5. Statistical analyses

The data collected were entered into Microsoft excel 2018. Statistical analysis was done using IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY: IBM Corp. Descriptive analyses were done. Student's t-test, repeated measures of ANOVA were used for determination of the significance of HOMA-IR, HOMA-β mean differences between and within the groups. Multivariate logistic regression analysis was performed for calculation of odds ratios (OR) for the prevalence of both prediabetes and IFG by periodontitis category after adjustment of all possible covariates.

## 3. Results

A total of 100 sample, 50 each male and female are studied. Among them 26 were below 30yrs age. Most of them are having brushing habit of once a day,1/5 were smokers,1/4 were alcoholic. Around 50% were had physical activity. 80% were of more than 25 BMI.

This duration of study was 12 weeks. PPD and CAL, Bleeding on probing were recorded at the baseline to confirm chronic periodontitis, NSPT done to the all the

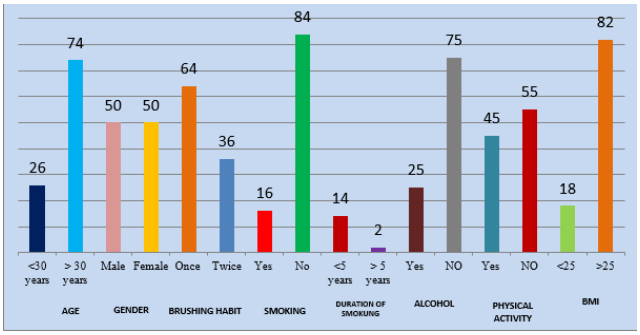


**Fig. 1:**

patients after recording BMR, blood parameters like FBS, post prandial blood sugar, Fasting insulin. These are recorded at 6 weeks and 12 weeks post NSPT and data are studied to correlate with HOMA MODEL.

The homeostasis model assessment index, HOMA-IR and HOMA-β were used for measurement of insulin resistance and beta cell function (insulin secretion capacity), respectively and the results are as follows.

Table 1 On intergroup comparison of Microbial load between Control Group and Test group, a highly statistically significant difference (p<0.0001***) was noted in the test group from baseline to 6 weeks and 12 weeks. However, control group showed no statistically significant difference (p<0.14) from baseline to 6 weeks and 12 weeks.

Table 2 On intergroup comparison of BMI between Control Group and Test group a highly statistically significant difference (p<0.0001***) was noted in the test group from baseline to 6 weeks and 12 weeks. However, control group showed no statistically significant difference (p<0.14) from baseline to 6 weeks and 12 weeks

Table 3 On comparison of Periodontal probing depth between Control Group and Test group a highly statistically significant difference (p<0.0001***) was noted in the test group from baseline to 6 weeks and 12 weeks. Similarly, control group too showed a statistically significant difference (p<0.002*) from baseline to 6 weeks and 12 weeks.

Table 4 On comparison of Clinical Attachment Loss between Control Group and Test group a highly statistically significant difference (p<0.0001***) was noted in the test group from baseline to 6 weeks and 12 weeks. Similarly, control group too showed a highly statistically significant difference (p<0.0005***) from baseline to 6 weeks and 12 weeks.

Table 5 On comparison of Gingival bleeding Index between Control Group and Test group a highly statistically significant difference (p<0.0001) was noted between both the groups from baseline to 6 weeks and 12 weeks

Table 6 On intergroup comparison of Fasting blood sugar values between Control Group and Test group no significant difference was noted in both the groups from baseline to 6

**Table 1:**

| | Timeline | Mean | Standard Deviation | F Stat | P value |
|---|---|---|---|---|---|
| | Microbial Load Base Line | 74 | 5.65 | | |
| **Control Group** | After 6 weeks | 71 | 4.96 | 11.6 | 0.14 |
| | After 12 weeks | 68 | 3.58 | | |
| | Microbial Load Base Line | 78 | 6.23 | | |
| **Test group** | After 6 weeks | 71 | 5.25 | 84.2 | **0.0001*** ** |
| | After 12 weeks | 66 | 4.22 | | |

**Table 2:**

| | Timeline | Mean | Standard Deviation | F Stat | P value |
|---|---|---|---|---|---|
| | BMI base line | 28.11 | 2.45 | | |
| **Control Group** | After 6 weeks | 28.06 | 2.11 | 11.6 | 0.14 |
| | After 12 weeks | 27.24 | 1.96 | | |
| | BMI base line | 28.32 | 2.32 | | |
| **Test group** | After 6 weeks | 27.8 | 2.01 | 84.2 | **0.0001*** ** |
| | After 12 weeks | 27.5 | 1.99 | | |

**Table 3:**

| | Timeline | Mean | Standard Deviation | F stat | P Value |
|---|---|---|---|---|---|
| | Periodontal probing depth base line | 5.52 | 0.51 | | |
| **Control group** | After 6 weeks | 5.25 | 0.33 | 11.5 | **0.002*** |
| | After 12 weeks | 5.02 | 0.15 | | |
| | Periodontal probing depth base line | 5.55 | 0.28 | | |
| **Test group** | After 6 weeks | 5.13 | 0.6 | 73.9 | **0.0001*** ** |
| | After 12 weeks | 5.01 | 0.2 | | |

**Table 4:**

| | Time line | Mean | Standard Deviation | F stat | P Value |
|---|---|---|---|---|---|
| | CAL Baseline | 2.42 | 0.09 | | |
| Control Group | After 6 weeks | 2.27 | 0.19 | 17.56 | 0.0005*** |
| | After 12 weeks | 2.16 | 0.24 | | |
| | CAL Baseline | 2.66 | 0.63 | | |
| Test Group | After 6 weeks | 2.33 | 0.28 | 66.55 | 0.0001*** |
| | After 12 weeks | 2.16 | 0.25 | | |

**Table 5:**

| | Timeline | Mean | Standard Deviation | F Stat | P value |
|---|---|---|---|---|---|
| | GBI Score Base Line | 50 | 15.65 | | |
| **Control Group** | After 6 weeks | 41.04 | 13.65 | 81.7 | **0.0001*** ** |
| | After 12 weeks | 33.92 | 13.69 | | |
| | GBI score base line | 54.18 | 10.07 | | |
| **Test group** | After 6 weeks | 44.42 | 10.11 | 657.6 | **0.0001*** ** |
| | After 12 weeks | 36.72 | 11.46 | | |

**Table 6:**

| | Timeline | Mean | Standard Deviation | F Stat | P value |
|---|---|---|---|---|---|
| | FBS base line | 112.9 | 7.88 | | |
| **Control Group** | After 6 weeks | 111.8 | 7.12 | 1.26 | 0.41 |
| | After 12 weeks | 111.2 | 6.87 | | |
| | FBS base line | 113.2 | 8.61 | | |
| **Test group** | After 6 weeks | 112.1 | 8.09 | 3.21 | 0.29 |
| | After 12 weeks | 110.4 | 7.87 | | |

weeks and 12 weeks.

Table 7 On intergroup comparison of Post prandial blood sugar between Control Group and Test group, a highly statistically significant difference ($p<0.016^{**}$) was noted in the test group from baseline to 6 weeks and 12 weeks. However, control group did not show a statistically significant difference ($p<0.12$) from baseline to 6 weeks and 12 weeks.

Table 8 On intragroup comparison of HOMA-IR values a highly statistically significant difference ($p<0.0001^{*}$) was noted from baseline to 6 weeks and 12 weeks.

Table 9 On comparison of HOMA- IR between Control Group and Test group a highly statistically significant difference ($p<0.0001$) was noted between both the groups from baseline to 6 weeks and 12 weeks

Table 10 On intragroup comparison of HOMA-β values a statistically significant difference ($p<0.001^{**}$) was noted from baseline to 6 weeks and 12 weeks.

Table 11 On comparison of HOMA-β between Control Group and Test group a highly statistically significant difference ($p<0.001$) was noted between both the groups from baseline to 6 weeks and 12 weeks.

## 4. Discussion

In the present study, the association of periodontitis and pre-diabetes was evaluated clinically and microbiologically at the baseline, and postoperatively at 6 weeks and 12 weeks of intervals. The microbial load in pre-diabetes patients was assessed using samples collected from the periodontal pockets ≤5mm by gram staining. The effect of periodontal therapy in pre-diabetes conditions was assessed using Homeostasis Model assessments of IR and β-cell function (HOMA-IR and HOMA-β), FBS, post prandial blood sugar and Fasting insulin.

In the present study, the clinical parameters PPD, clinical attachment level, Bleeding on probing were correlated with HOMA-IR, HOMA-β, BMR, FBS, post prandial blood sugar, Fasting insulin at regular intervals to learn about the association of periodontitis and pre-diabetes and also to know whether if the periodontal therapy (non-surgical) could aid in preventing the progression of pre-diabetic condition to diabetes. The nonsurgical periodontal treatment remains the gold standard for managing the periodontal patients. It can result in the reduction of inflammation, pocket depth reduction and clinical attachment gain. There is no certain magnitude of initial probing pocket depth where nonsurgical periodontal therapy is no longer effective. However, it needs to be emphasized that the root instrumentation is only indicated for sites with probing depth 4mm and above as instrumenting shallow sites will potentially develop the loss of attachment. Yet, no other therapeutic modality can be routinely utilized for the nonsurgical periodontal treatment than the scaling and root debridement or planing or instrumentation. The periodontal treatment is comprised of a bidirectional effort between the clinician and the patient to achieve the best therapeutic outcome. Therefore, the role of a high quality root debridement along with the implementation of a risk factor modification approach (oral hygiene habits, patient's motivation and education, smoking cessation, diabetes control, healthy lifestyle changes) in the management of periodontitis is paramount. Obesity is also a key factor in the occurrence of metabolic syndrome and is strongly linked with insulin resistance (Eckel et al. 2005, Grundy 2005).[14,15] Based on an experimental, controlled, animal study, Amar et al.(2007)[16] have suggested that obesity may disturb immune host defence to periodontal pathogens such as Porphyromonas gingivalis, and thus promote periodontitis. In human studies, a high BMI and a high waist– hip ratio have been related to periodontitis or deep pockets in various populations including young and older subjects, men and women (Saito et al. 2001, 2005).[17,18] A dose dependent relationship between the BMI and deep periodontal pockets has also been described in low-risk middle aged subjects such as non-diabetic, non-smoking people (Ylostalo et al. 2008).

In the present study on comparison of BMI between Control Group and Test group a highly statistically significant difference ($p<0.0001^{***}$) was noted in the test group from baseline to 6 weeks and 12 weeks. However, control group showed no statistically significant difference ($p<0.14$) from baseline to 6 weeks and 12 weeks. Therefore, we can infer that periodontal intervention is helpful for glucose control and insulin sensitivity improvement, which may be associated with decreased serum inflammatory cytokines and increased serum adiponectin levels.

**Table 7:**

| | Timeline | Mean | Standard Deviation | F Stat | P value |
|---|---|---|---|---|---|
| | PPBS base line | 125.9 | 6.92 | | |
| **Control Group** | After 6 weeks | 125.4 | 6.12 | 3.1 | 0.12 |
| | After 12 weeks | 124.9 | 5.32 | | |
| | PPBS base line | 126.7 | 7.00 | | |
| **Test group** | After 6 weeks | 125.7 | 6.07 | 4.2 | **0.016**** |
| | After 12 weeks | 124.2 | 5.21 | | |

**Table 8:**

| Timeline | Mean | Standard Deviation | F stat | P value |
|---|---|---|---|---|
| HOMA -IR base line | 2.90 | 1.54 | | |
| HOMA- IR 6 weeks | 1.71 | 1.21 | 52.3 | **0.0001*** |
| HOMA- IR 12 weeks | 0.72 | 0.54 | | |

**Table 9:**

| | Timeline | Mean | Sd | F | P Value |
|---|---|---|---|---|---|
| | HOMA -IR at baseline | 0.68 | 0.2 | | |
| **Control group** | HOMA -IR After 6 Weeks | 0.68 | 0.2 | 80 | **0.0001***** |
| | HOMA -IR After 12 Weeks | 0.64 | 0.19 | | |
| | HOMA -IR at baseline | 3.43 | 1.74 | | |
| **Test Group** | HOMA -IR After 6 Weeks | 3.28 | 1.56 | 52.3 | **0.0001***** |
| | HOMA -IR After 12 Weeks | 3.1 | 1.43 | | |

**Table 10:**

| Timeline | Mean | Standard Deviation | F stat | P value |
|---|---|---|---|---|
| HOMA -β Base Line | 45.114 | 8.312 | | |
| HOMA-β 6 weeks | 57.654 | 4.364 | 45.04 | **0.001**** |
| HOMA -β12 weeks | 88.068 | 3.147 | | |

**Table 11:**

| | Timeline | Mean | Sd | F | P value |
|---|---|---|---|---|---|
| **Control group** | HOMA- β at baseline | 42.410 | 3.121 | | |
| | HOMA- β After 6 Weeks | 54.125 | 3.245 | 42.31 | **0.001**** |
| | HOMA -β After 12 Weeks | 86.147 | 4.165 | | |
| | HOMA- β At Baseline | 44.272 | 4.321 | | |
| **Test Group** | HOMA -β After 6 Weeks | 58.198 | 5.177 | 43.04 | **0.0001*** |
| | HOMA β After 12 Weeks | 88.724 | 6.144 | | |

A thorough review by Cobb (1996) [19] has been often sited regarding the expected outcomes of nonsurgical periodontal treatment in probing depth reduction and clinical attachment gain at sites that initially were 4 to 6 mm in depth or greater than 7mm. He reported mean pocket depth reduction of 1.29 mm and 2.16 mm respectively and mean gain of clinical attachment of 0.55 and 1.29 mm respectively. So these results are similar to our present study. In the present study on comparison of Periodontal probing depth highly statistically significant difference (p<0.0001***) was noted in the test group from baseline to 6 weeks and 12 weeks. Similarly, control group too showed a statistically significant difference (p<0.002*) from baseline to 6 weeks and 12 weeks. On pairwise comparison a statistically significant difference (p<0.045*) was noted between 6 weeks and 12 weeks. There was a comparable difference in pocket probing depths from baseline to 12 weeks. Non-surgical treatment showed a decrease in probing pocket depth in these subjects. This could be attributed to the decrease in the disease activity caused by the treatment protocol.

In the present study on comparison of Clinical Attachment Loss between Control Group and Test group a highly statistically significant difference (p<0.0001***) was noted from baseline to 6 weeks and 12 weeks. This could be due to the reduction in bacterial load following non-surgical therapy (Haffajee et al 1997). [20]

Bleeding on probing (BOP) is an indicator of tissue inflammatory response to bacterial pathogens. The bleeding on probing (BOP) is a widely used clinical sign as indicator of the periodontal condition and disease progression.A study on this topic, published in 1990 by Lang and colleagues, demonstrates how the absence of BOP represents a reliable indicator of periodontal stability.We know, from the work by Löe et al. (1965), that the onset of gingivitis requires a presence of bacterial plaque for some length of time. Relative Risk analysis by L Checchi et al (2009)demonstrated that the incidence of BOP could be up to 6 times with calculus and up to 8 times with plaque. In the present study on comparison of Gingival bleeding Index between Control Group and Test group a highly statistically significant difference ($p<0.0001$) was noted between both the groups from baseline to 6 weeks and 12 weeks. This could be attributed to the decrease in the plaque and calculus caused by the treatment protocol and improvement in oral hygiene

In the present study comparison of Fasting blood sugar values between Control Group and Test group no significant difference was noted in both the groups from baseline to 6 weeks and 12 weeks. On intergroup comparison of Post prandial blood sugar between Control Group and Test group a highly statistically significant difference ($p<0.016$**) was noted in the test group from baseline to 6 weeks and 12 weeks. However, control group did not show a statistically significant difference ($p<0.12$) from baseline to 6 weeks and 12 weeks. This decrease in PPBS can be attributed to the decrease in inflammation and inflammatory markers (that causes insulin resistance) following non-surgical periodontal therapy.

Insulin resistance is central in the development of the metabolic disturbances encountered in the metabolic syndrome and is linked closely with glucose disorders (Matthews et al. 1985).[21] Given that poor glycemic control is likely involved in the development of periodontitis (Tsai et al. 2002),[22] insulin resistance may also play a central role. Many studies have clearly shown that inflammatory markers levels are higher in cases of periodontitis and increased levels of inflammatory markers causes insulin resistance (Chandrasekhar H et al. 2017).[23] Insulin sensitivity can be measured using different methods. In this study, we used the homeostasis model assessment (HOMA) method to yield an estimate of insulin sensitivity, because it has been shown to be a feasible method in large epidemiological studies for subjects of various ethnicities and varying degrees of glucose tolerance (Wallace et al. 2004).[24] The HOMA method derives an estimate of insulin sensitivity from a mathematical model of fasting plasma glucose and insulin concentrations (Matthews et al. 1985).[21] Periodontitis and insulin resistance (IR) show bidirectional relationship. The homeostatic model assessment (HOMA) is a method used to quantify insulin resistance and beta-cell function. It was first described under the name HOMA by Matthews et al. in 1985.[21]

A recent study reported evidence of a close relationship of both HOMA-IR and HOMA-β in development of periodontal disease in subjects without diabetes (Timonen P et al, 2013).[25] Oelisoa M. Andriankaja et al (2018)[26] concluded that the baseline IR is associated with increased risk of gingival/periodontal inflammation at follow-up in the population of overweight/obese Hispanic adults. These findings have public health relevance, as prevention of IR through a healthy lifestyle may help reduce periodontal inflammation as well as diabetes. Nishioka S et al (2019)[27] concluded that among individuals diagnosed with borderline diabetes, those who had <37% of a lower BOP (%) showed potential improvements in BMI, fasting serum insulin, HOMA-IR, HOMA-β and Matsuda Index following non-surgical periodontal therapy. Similar to the above mention studies in this present study, also we can observe a highly statistically significant difference ($p<0.0001$*) was noted from baseline to 6 weeks and 12 weeks especially after nonsurgical periodontal therapy in test group for HOMA IR and HOMA-β values. This significant decrease in HOMA IR and HOMA-β values might be due to the decrease in circulation of inflammatory mediators (which are provoking for the development of insulin resistance) following nonsurgical periodontal therapy.

## 5. Conclusion

In the present study the clinical parameters PPD, clinical attachment level were evaluated and correlated with HOMA-IR, HOMA-β, BMR, FBS, post prandial blood sugar, fasting insulin at regular intervals to learn about the association of periodontitis and pre-diabetes and also to know whether if the periodontal therapy (non-surgical) could aid in preventing the progression of pre-diabetic condition to diabetes.

The conclusions from this study are

1. There is significant association between prediabetes states and periodontitis.
2. Early diagnosis of periodontitis and a proper treatment in prediabetic group can prevent the progression of prediabetic condition to diabetes and vise versa.
3. Despite of extensive adjustment, remaining confounders may exist, such as other inflammatory biomarkers (interleukins for instance), poor tooth brushing habits (oral hygiene), or periodontal pathogens, habits (smoking, alcohol), gender etc.
4. Smoking is a major risk factor for periodontitis but its relationships with metabolic syndrome and insulin resistance are more complex. So further research can be done to estimate the association between smoking, prediabetes, periodontitis, and their treatment outcomes.

5. Further research can be done to assess association of prediabetic condition, periodontitis and the treatment outcomes in different genders and different age groups.

## 6. Source of Funding

None.

## 7. Conflict of Interest

There are no conflicts of interest.

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