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ORIGINAL RESEARCH

Assessment of Periodontal Inflamed Surface Area and Its Relationship with Glycemic Control in Type 2 Diabetes

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ABSTRACT

Introduction: The objective of this study is to assess the periodontal inflamed surface area (PISA) and its relationship with glycemic control in type 2 diabetes with and without periodontitis.

Materials and methods: A study was performed on 60 outpatients (males and females) reporting to the Department of Periodontics, Rajah Muthiah Dental College and Hospital and the Diabetic Clinic, Rajah Muthiah Medical College, Annamalai Nagar, Chidambaram, Tamil Nadu. The age of the study subjects ranged from 40 to 60 years. Patients having HbA1c levels >7 mg/dl were diagnosed as type 2 diabetes mellitus and were enrolled in the study. The selected patients were divided into two groups of 30 patients each, with at least eight remaining teeth present: Group I – Diabetic patients with periodontitis (test) with probing pocket depth (PPD) of 3 to 10 mm and bleeding on probing (BOP), and Group II – Diabetic patients without periodontitis (periodontally healthy as control) with PPD not exceeding 3 mm and limited BOP. Subjects were excluded if they were under systemic antibiotics 3 months prior and during the study and if they have undergone periodontal treatment 6 months prior to the study.

Results: When HbA1c increased, the PISA values also increased in type 2 diabetic patients with and without periodontitis.

Conclusion: The cross-sectional clinical study reveals that there is a linear association between diabetes and PISA in type 2 diabetic patients with and without periodontitis.

Keywords: Glycosylated hemoglobin A, Periodontal inflamed surface area, Periodontitis, Type 2 diabetes mellitus.

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INTRODUCTION

Periodontal disease is a chronic inflammatory condition affecting the supporting tissues of the teeth and results in destruction of periodontal tissues, leading to connective tissue loss, alveolar bone loss, and formation of pathological pockets around diseased teeth.[1]

Diabetes is a metabolic disorder, i.e., characterized by high blood glucose in the context of insulin resistance and relative insulin deficiency.[2] Studies could simultaneously elucidate the potential causal nature of the association between periodontitis and glycemic control.[3] Gingivitis and periodontitis are two diseases that are initiated by microbial plaque, and these are associated with periodontal diseases.[4] Diabetes mellitus is also a clinical syndrome characterized by hyperglycemia due to deficiency or diminished effectiveness of insulin.[5] The relationship between diabetes and periodontitis deepens; increased patient awareness of the link between diabetes mellitus and oral health and collaboration among dental and medical professionals for the management of affected individuals became increasingly important.[6] There were increased calculus deposition and increased periodontal disease in diabetics than in nondiabetics.[7] It was also reported that there was a high incidence of periodontal disease among diabetic patients.[8] Oral changes observed clinically were actually the result of diabetes or may be due to the preexisting or coexisting metabolic changes.[9] Dose–response relationship in the prediabetic state also indicated that the level of glucose intolerance in nondiabetic individuals also correlated with the severity of periodontal disease.[10] Glycemic control can easily be estimated by measuring hemoglobin A1c (HbA1c) levels in the blood. There exists a two-way relationship between glycemic control of diabetic patients and periodontal disease. Neither probing pocket depth (PPD) nor clinical attachment level (CAL) is appropriate to assess the dose–response relationship between periodontitis and glycemic control (HbA1c levels) in type 2 diabetic patients. Therefore, a new measure of periodontitis, namely the periodontal inflamed surface area (PISA), as a risk factor was developed by Nesse et al.[11] Periodontal inflamed surface area quantifies the inflammatory burden posed by periodontitis, and thus, it can be easily applied. It reflects the surface area of bleeding pocket epithelium in square millimeters.

MATERIALS AND METHODS

Outpatients reporting to the Department of Periodontics, Rajah Muthiah Dental College and Hospital and Diabetic Clinic, Rajah Muthiah Medical College, Annamalai Nagar, Chidambaram, Tamil Nadu, for treatment were selected. An invitation to participate in the study was extended to the patients. Each patient was given a brief explanation of the study, and an informed consent was obtained from each participant before being recruited for the study. The research protocol was approved by the Institutional Human Ethical Committee before the commencement of study. A total of 60 patients, aged 40 to 60 years of both genders, were included in the study. Patients having HbA1c levels >7 mg/dL were diagnosed as type 2 diabetes mellitus and were enrolled in the study. The selected patients were divided into two groups of 30 patients each, with at least eight remaining teeth present: Group I – diabetic patients with periodontitis (test) with PPD of 3 to 10 mm and group II – diabetic patients without periodontitis with PPD not exceeding 3 mm. Subjects were excluded if they were under systemic antibiotics 3 months prior and during the study and if they have undergone periodontal treatment 6 months prior to the study.

Clinical Assessment

Data on PPD[12] (Fig. 1) and Bleeding on probing (BOP)[13] (Fig. 2) at six sites per tooth were entered in a spreadsheet to calculate PISA for each patient.[11] This spreadsheet can be accessed via http://www.parsprototo.info/docs/PISA-CAL.Xls[14] (Fig. 3) and is free for use. Periodontal inflamed surface area was calculated in four steps:

1. After filling PPD measurements at six sites per tooth, the computer calculates the mean PPD for each particular tooth.
2. The mean PPD around a particular tooth is entered into a formula that translates this linear mean PPD into a periodontal epithelial surface area (PESA) for that specific tooth.[15] The PESA for a particular tooth is the root surface area of that tooth (in mm^2), i.e., covered by a pocket epithelium.
3. The PESA may consist of an uninflamed pocket epithelium that does not pose an inflammatory burden. Therefore, for a particular tooth, it is subsequently multiplied by the proportion of sites around that tooth that was affected by BOP. For example, if three out of six sites were affected by BOP, the PESA of that particular tooth was multiplied by 3/6, and this gives the PISA of that specific tooth.
4. The sum of PISAs around each individual tooth is calculated, and this results in total PISA of a patient.

Collection of Blood

Blood samples were collected in the morning between 8:30 and 9:30, following an overnight fasting. About 2 mL of blood was collected using SST II Advance BD Vacutainer Flashback Blood Collection Needle (BD, Franklin Lakes, NJ, USA) (Fig. 4). The HbA1c level was assessed by Glycosylated Haemoglobin *kit* (*Diatek*, Kolkata, India) (Fig. 5). The weight (Fig. 6) and height (Fig. 7) of each patient were measured.

Statistical Analysis

Data thus obtained were analyzed using Statistical Package for the Social Sciences software (version 15.0). The following statistical tools were used to analyze the data. Descriptive statistics was performed for all the parameters. Paired t-test was done to analyze the significance between different groups. Analysis of variance (ANOVA) was applied to compare the mean values of all the parameters among the two groups. Multiple linear regression analysis was applied to correlate the PISA and HBA1c with clinical parameters.

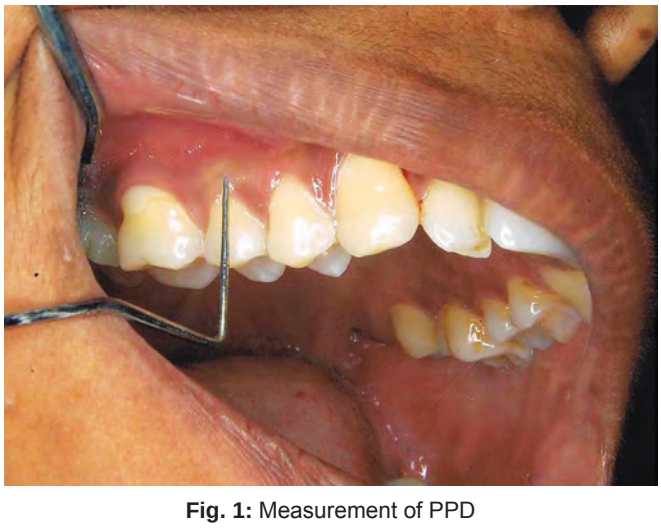


Fig. 1: Measurement of PPD

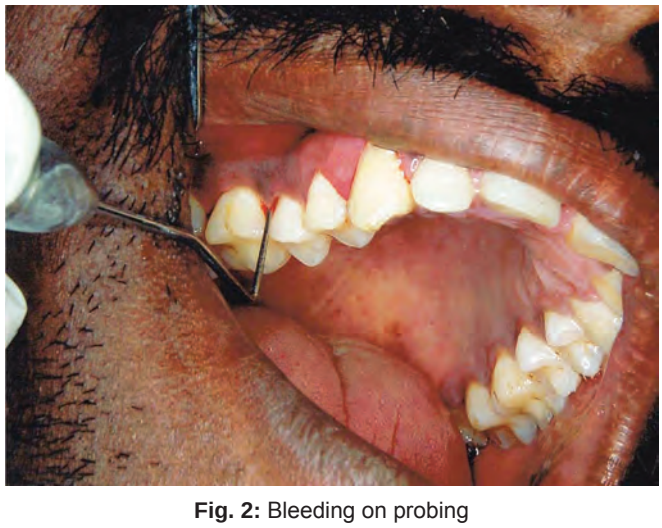


Fig. 2: Bleeding on probing

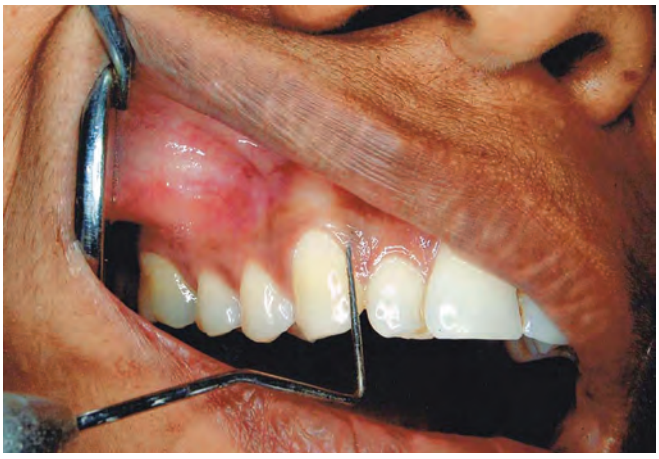


Fig. 3: Measurement of CAL

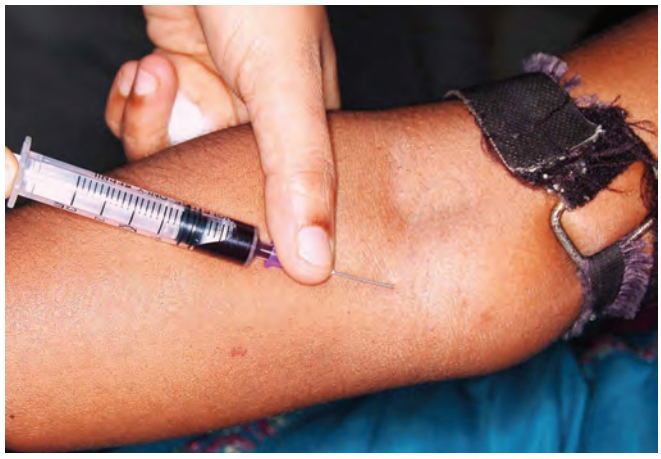


Fig. 4: Collection of blood

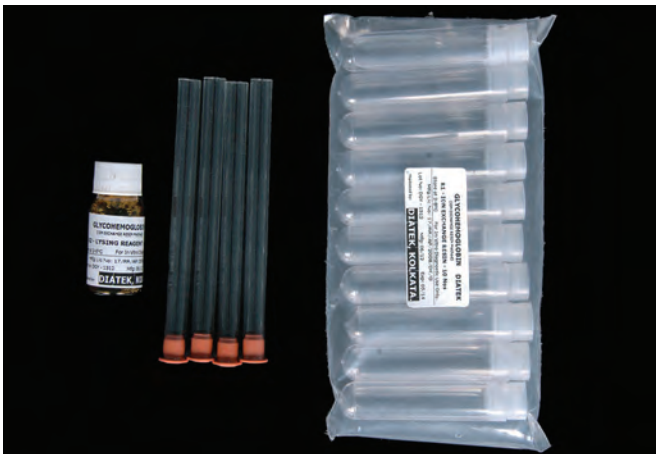


Fig. 5: Glycohemoglobin: Diatek kit

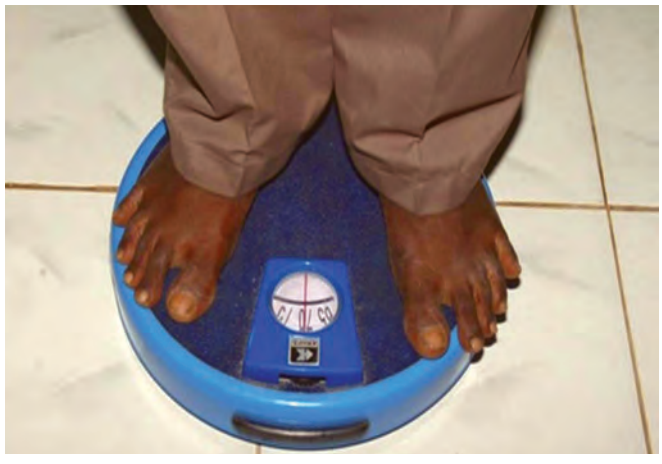


Fig. 6: Assessment of body mass index

Fig. 7: Measurement of height

RESULTS

Based on the statistical analysis, the following conclusions were drawn:

- The regression analysis showed that the body mass index has a significant association as the majority of patients were obese (58.3%) and overweight (41.7%) (Table 1).
- Uncontrolled diabetic patients with periodontitis had greater PISA compared with uncontrolled diabetic patients without periodontitis. Graph 1 shows the mean and standard deviation for HbA1c levels for both (Groups I and II).
- The correlation between PISA and HbA1c level was statistically significant as PISA increased; HbA1c also increased showing a linear association between PISA and glycemic control (Table 2). Periodontal inflamed surface area and HbA1c in groups I and II patients are depicted in a scatter plot (Graphs 2 and 3).

Table 1: Percentage of body mass index in group I (diabetic patients with periodontitis) and group II (diabetic patients without periodontitis)

		Frequency	*Percent*	*Valid percentage*	*Cumulative percentage*
Valid	Healthy	7	11.7	11.7	11.7
	Overweight	18	30.0	30.0	41.7
	Obese	35	58.3	58.3	100.0
	Total	60	100.0	100.0	100.0

Table 2: Descriptive analysis showing mean and standard deviation of PESA, PISA, and HbA1c in diabetic patients with and without periodontitis

Distribution of group		*Periodontal epithelium surface area*	*Periodontal inflamed surface area*	*HbA1c*
Diabetic with periodontitis	Mean	2795.8172	2337.8706	9.9724
	N	30	30	30
	Standard deviation	1093.0668	1365.5563	0.6964
Diabetic without periodontitis	Mean	1229.1984	26.72	8.0323
	N	30	30	30
	Standard deviation	495.8580	660.9852	0.8211
Total	Mean	1986.3975	1205.2654	8.9700
	N	60	60	60
	Standard deviation	1146.8571	1525.6112	1.2365

DISCUSSION

This study evaluated PISA and its relationship with glycemic control in type 2 diabetic patients with and without periodontitis. Type 2 diabetes mellitus is one of the most prevalent metabolic diseases. It is caused by peripheral resistance to insulin action and impaired insulin secretion that results in elevated levels of blood glucose. Establishing the potential causal nature of the association between periodontitis and diabetes requires the assessment of dose–response relationship between inflammatory burden posed by periodontitis and glycemic control. Hence, the study assessed PISA and its relationship with glycemic control in type 2 diabetic patients with and without periodontitis, and it also attempted to correlate PISA with periodontal parameters and glycemic status.

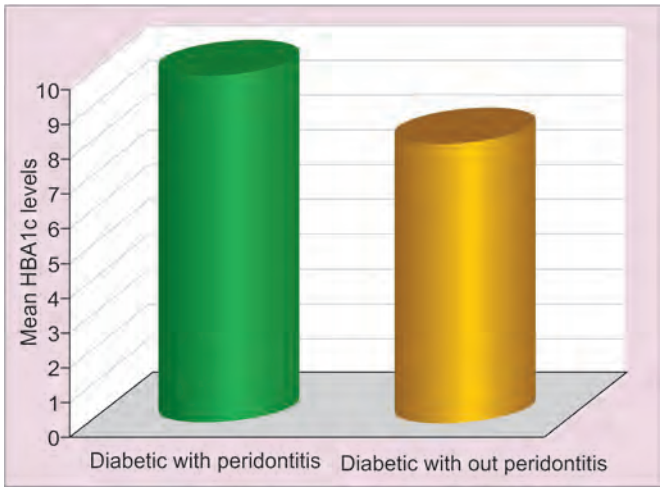
The first and foremost demand was that the new classification should adequately quantify the amount of inflamed periodontal tissue. Second, the classification should be easy to use and broadly applicable. This means that the classification should make use of clinical measurements commonly used to establish periodontitis, i.e., CAL, PPD, gingival recession, and BOP measurements. A literature search was performed to look for a classification of periodontitis to meet these demands. Periodontitis is classified mainly based on PPD and CAL. However, neither PPD nor CAL is appropriate to assess dose–response relationship between periodontitis and HbA1c, because PPD and CAL are linear measures that do not quantify the amount of inflamed periodontal tissue.

Periodontal inflamed surface area reflects the surface area of bleeding pocket epithelium in square millimeters. The most common method for calculating PISA was described by Hujoel et al[15] using a Microsoft Excel spreadsheet. Periodontal inflamed surface area can be calculated using a freely downloadable spreadsheet. Spread sheets are freely available from the website:www.parsprototo.info. It calculates PISA in accordance with CAL, gingival recession, and BOP at six sites per tooth by entering in the freely downloadable spreadsheet. The broader classification of PISA may provide decisive conclusions on periodontitis as a risk factor for other diseases. In this study, PISA and HbA1c were assessed in type 2 diabetic patients with and without periodontitis.

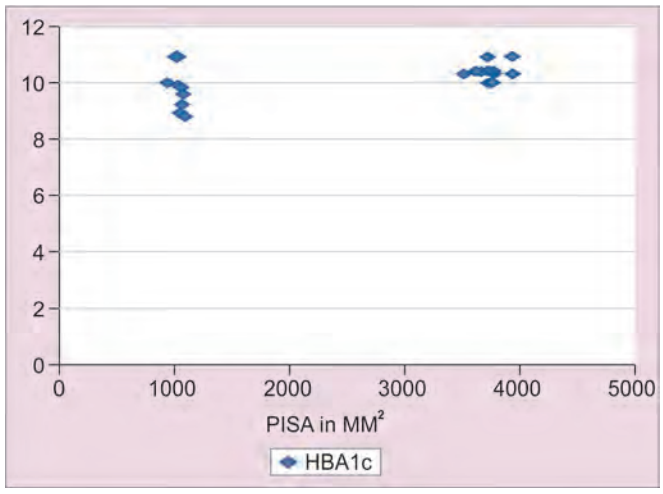
In addition to research purpose, the spreadsheet might also be used to show patients their surface area of bleeding pocket epithelium in square millimeters, illustrating the inflammatory burden posed by periodontitis in their body. An additional advantage of PISA is that it can be retrospectively calculated using existing research data. Periodontal inflamed surface area is calculated in a spreadsheet by four steps. The normal HbA1c level is <7 mg/dL.[16]

In this study, the clinical periodontal parameters that reflect the health of the periodontium, such as PPD, CALs, and BOP for diabetic patients with periodontitis were found to be maximum (8.78, 8.29, and 1.8571, respectively), and for diabetic patients without periodontitis, their values were found to be minimum (2.87, 1.91, and 0.1428, respectively). And then, the periodontal status was compared with that of the glycemic status in type 2 diabetic patients. The results showed that in periodontitis patients, the glycemic level (HbA1c) was increased.

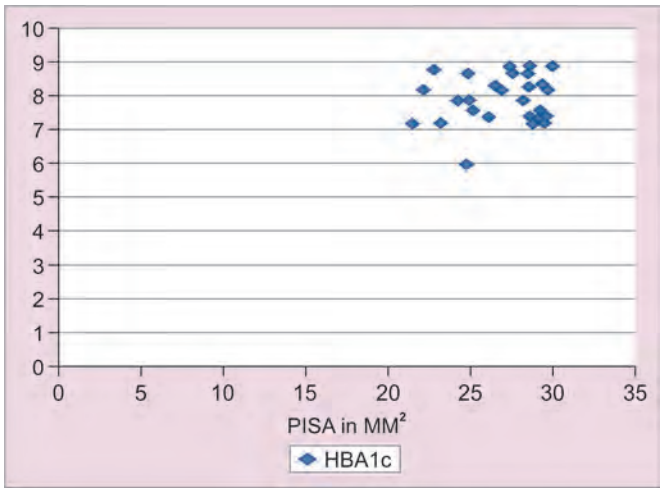
This can be explained by the two-way relationship that exists between periodontitis and diabetes. In diabetes, the adherence, chemotaxis, and phagocytosis of neutrophils are impaired, which prevents neutrophil-mediated destruction of bacteria in the periodontal pocket, thereby increasing periodontal destruction. Advanced glycation end products and their interaction with R (receptor) are believed to induce an oxidative stress that may contribute to chronic monocytic upregulation, activation of nuclear factor κB, and subsequent secretion of proinflammatory cytokines, such as tumor necrosis factor (TNF)-α, interleukin (IL)-1β, and IL-6 involved in periodontal tissue inflammation and destruction. Results similar to this study have been reported by Taylor et al.[17]



Graph 1:



Graph 2:



Graph 3:

In another study in 2002, it was found that patients with poorly controlled diabetes had severe periodontal destruction. Saito et al[10] also reported similar results.

Nesse et al[11] suggested that PISA predicts the probability of periodontitis to cause or deteriorate other diseases by quantifying the inflammatory burden posted by periodontitis. This could be done by assessing PISA and blood levels of inflammatory mediators as TNF-α, IL-6, and IL-1β. This suggested that the PISA values are low in diabetic patients without periodontitis, which coincides with our study results.

This study reported that the values of PISA when compared with diabetic patients with and without periodontitis were significant as suggested by Hujoel et al.[15] Individuals without periodontitis had a typical dentogingival epithelial surface area (DGES) of 5 cm^2. Among individuals with periodontitis, the mean DGES in the three samples ranged from 8 cm^2 (ranging from 1 to 29 cm^2) to 20 cm^2 (ranging from 2 to 44 cm^2). It was concluded that the mean DGES among individuals with periodontitis ranges from 8 to 20 cm^2, considerably smaller than the range of 50 to 200 cm^2 currently assumed.[5] The present study reported that PISA and HbA1c has a close association in diabetic patients with and without periodontitis suggested by Kinane and Bouchard 2008.[18] They also suggested that the effect of periodontal treatment on diabetic control and systemic inflammation is not proven, so there is a need to perform large well-designed randomized clinical trials to establish the benefit of periodontal treatment to glycemic control in type 2 diabetic mellitus patients.

This study reported that when PISA and HbA1c were compared between type 2 diabetic patients with and without periodontitis, the increase in PISA also increases HbA1c levels. Nesse et al suggested that on a group level, an increase in PISA of 333 cm^2 is associated with an increase in HbA1c of 1.0%. Similarly, a decrease in PISA of 333 cm^2 is associated with a decrease in HBA1c of 1.0%. Susanto et al[19] conducted a study to analyze whether periodontitis severity as measured with PISA and CRP predicted HbA1c levels in a group of healthy Indonesians and a group of Indonesians treated for type 2 diabetes mellitus. This implies that periodontitis may contribute to insulin resistance in Indonesians treated for type 2 diabetes mellitus; PISA was not able to predict HbA1c.

The overall observations showed that the PESA values were lesser in diabetic patients without periodontitis than those in periodontally diseased groups. The PESA was found to be higher in diabetic patients with periodontitis compared with periodontally healthy groups. In type 2 diabetic patients with and without periodontitis, when the periodontal surface area increases, the HbA1c also increases. This study can further be improved by increasing the sample size. Moreover, because it is a cross-sectional study, a longitudinal study can be done by recording the values for periodontal inflamed surface before and after surgical treatment in type 2 diabetic

patients with and without periodontitis. Periodontal inflamed surface area, theoretically, appears to be a better classification of periodontitis as a risk factor for other diseases than any other classification currently used (face validity). The upcoming next step is to perform studies to analyze construct validity by means of correlating PISA with the measures of activity, severity, or presence of other diseases that can also be pursued in the future.[11]

CONCLUSION

The study results suggest that an increase in PISA is associated with an increase in HbA1c levels. This relationship might be an indication of causal relationship between PISA and HbA1c. However, PISA can be utilized as a useful tool to quantify the inflammatory burden posed by periodontitis. The overall observations showed that PESA was lesser in diabetic patients without periodontitis than that in diabetic patients with periodontitis.

Tex/Latex Supplementary File

TLS 1: Mean HbA1c levels in both groups I and II subjects.
TLS 2: Assessment of PISA and HbA1c in group I (Diabetic patients with periodontitis).
TLS 3: Assessment of PISA and HbA1c in group II (Diabetic patients without periodontitis).

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