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Prevalence And Severity of Periodontal Disease in Type 2 Diabetes Mellitus (Non–Insulin Dependent Diabetes Mellitus) Patients In Srinagar City

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Abstract
Background: *Periodontal disease has been labeled as the sixth complication of the diabetes. Abnormal collagen metabolism, abnormal polymorphonuclear cell function and altered sulcular microbial flora progresses periodontitis among diabetic patients.*
Aim: *To find out the prevalence and severity of periodontal disease among Non-insulin dependent Diabetes Mellitus Patients in srinagar city jammu and kashmir India.*
Materials and Methods: *50 patients with type 2 diabetics (mean age: 49.3 ± 8.97) and 50 patients with non-diabetics (mean age: 47.6 ± 8.85) were examined. . Plaque component of ramfjord's Periodontal Disease Index (PBI) (Schick and Ash Modification 1961), Papillary Bleeding Index (PBI) (Muhlemann H.R 1977), Periodontal Disease Index (PDI) (Sigurd P. Ramfjord 1967), Relative Attachment level (RAL) were recorded. Data was analyzed using SPSS version 15. P ≤ 0.05 and ≤0.01 was considered as statistically significant and highly significant respectively*
Results: *Diabetics had significantly higher mean PBI (p<0.01), PDI (p<0.05) than non-diabetics. The mean P1 (p >0.05) and RAL (p> 0.05) although were higher in diabetics than non-diabetics but not significantly different between the two groups.*
Conclusion: *Type‐II diabetic patients have a higher prevalence of periodontal disease than non diabetic individuals. More emphasis is to be put on the co-managemnt of oral and overall health in patients with diabetes by dental and general practitioners.*
Keywords: *Diabetes mellitus, severity, Srinagar, periodontitis*

I. Introduction

Periodontal diseases are one of the more prevalent oral diseases affecting more than 50% of Indian community. Untreated chronic periodontitis is responsible for tooth loss in majority of the cases. Constant presence of chronic inflammation and inflammatory mediators has also been proved to be a significant risk factor of systemic disease like diabetes mellitus (DM).[1] Epidemiological research indicates that periodontal diseases are widespread Throughout the world and evidence exists to show that their extent and severity increases with age.[2] This view of a particularly high prevalence of periodontal diseases appears to have originated from early epidemiological studies using an index system that gave weight to gingivitis and moderate periodontitis resulting from poor oral hygiene and calculus deposition.[3] Albandar and Rams[4] in an overview concluded that subjects of Asian ethnicity had the third highest prevalence of periodontitis. DM, a chronic metabolic diseases characterized by hyperglycemia, is often attributed to environmental and genetic factors.[5] The main types of diabetes are classified primarily on the basis of their underlying pathophysiology. Type-1 diabetes, which constitute 5-10% of all the cases results from autoimmune destruction of insulin producing β- cells in the pancreas leading to total absence of insulin secretion. Individuals with type 1 idiopathic DM lack immunologic markers indicative of an autoimmune destructive process of the beta cells. However, they develop insulin deficiency by unknown mechanisms and are prone to ketosis. Type-2 diabetes which constitutes about 85-90% of all cases comprises of a heterogeneous group of disorders characterized by variable degrees of insulin resistance, impaired insulin secretion, and increased glucose production. Distinct genetic and metabolic defects in insulin action and secretion gives rise to the common phenotype of hyperglycemia in type 2 DM. Type 2 DM is preceded by a period of abnormal glucose homeostasis classified as impaired fasting glucose (IFG) or impaired glucose tolerance (IGT).[6] Asia in particular has the highest prevalence of diabetes in the

world. Countries exhibiting the fastest rise in diabetic population growth include India and China, among many other developing countries.[7] Diabetes is an alarming public health problem, affecting 245 million people worldwide. Each year seven million individuals develop diabetes and the projection for the year 2030 expects that 366 million people will have the disease worldwide.[8] India leads the world today with the largest number of diabetic patients in any given country. Prevalence of diabetics has increased from 2.1-12.1% since 1970. WHO has issued a warning that India will be the "Diabetes capital of the world".[9] Gingival and periodontal diseases occur in childhood, adolescent and early adulthood, but the prevalence of periodontal disease, tissue destruction, and tooth loss increases with age.[10] Periodontal disease has been labeled as the "sixth complication of the diabetes".[11] There are two different hypotheses. According to one angiopathy, abnormal collagen metabolism, abnormal polymorphonuclear cell function and altered sulcular microbial flora are found in close association with the severity of periodontitis among diabetic patients.[12,13] Another hypotheses is that no relationship exists between DM and periodontal disease when two conditions exist together. It is a coincidence rather than a specific cause and effect relationship. Severity and distribution of local irritant affects the severity of periodontal diseases among diabetic patients.[14,15] No literature exist which depicts the prevalence and severity of periodontal diseases among patients with type 2 diabetes in hilly state of Himachal Pradesh. Hence, this study has been taken up to test the hypothesis that diabetic patients have more severe periodontal disease experience.

II. Materials And Methods

Persons attending at govt. Dental College and Hospital Srinagar with a medical history of type 2 diabetes aged 35 years or older comprised the study population. Information concerning diabetic status was retrieved from the patients' medical records. A nurse who had been trained on the aspects of this study recruited participants into the study. The Controls were considered non-diabetic by self-reported negative history of diabetes. Persons were excluded from the study if they did not have at least three Ramfjord's teeth or their substitutes. In addition, individuals who were on antibiotic treatment one month prior to examination or pregnant, were also excluded. A dentist conducted the intra-oral examination with the patient seated on a dental chair. A standard overhead light was used in a well-ventilated room with good natural light. Demographic information, medical, dental and social histories were recorded by a dental assistant who prior to the study had been trained for this purpose. The examiner was blinded on the medical history of the person being examined for periodontal disease. It was originally planned that some of the persons examined would be recalled for confirmation of diagnosis. However, the number of people reexamined was not enough for statistical computation. Plaque component of ramfjord's Periodontal Disease Index (PBI) (Schick and Ash Modification 1961)[16], Papillary Bleeding Index (PBI) (Muhlemann H.R 1977)[17,18],Periodontal Disease Index (PDI) (Sigurd P. Ramfjord 1967)[18,19], Relative Attachment level (RAL) i.e. distance between base of sulcus or pocket and a fixed reference point (horizontal notch) on the stent [20] were recorded. Plaque was assessed on the facial and lingual surface of all the teeth after using disclosing agent (fig 1). A Periodontal probe was gently moved along the gingival sulcus, on the mesial and distal aspects of the base of each interdental papilla (fig 2). Periodontal Disease Index (PDI), which combines the assessment of gingival inflammatory status and sulcular depth, was used to measure the presence and severity of periodontal disease. Evolution of index score was carried out for all the teeth. Gingivitis component of Periodontal Disease Index (PDI) was assessed first. After gently drying the gingiva, the extent of changes in color, contour, and form of the gingiva was evaluated. Evidence of ulceration and bleeding on gentle touching with the side of a periodontal probe were noted, as signs of severe gingivitis. Scoring was done as per the criteria. (fig 3)

The next step in the scoring procedure was recording of crevice depth in relation to the cement-enamel junction. A UNC 15 probe was used for the purpose. The relative Attachment level (RAL) is the distance between the base of the sulcus or pocket and a fixed reference point (horizontal notch) on the stent. This was later on used for assessment of clinical attachment gain or loss Determination of the level of attachment was done using UNC-15 probe.(fig 4)

Data were analyzed using SPSS (version 15). Means of variables were compared using t-test. Statistical significance was set at p-value < 0.05. The research proposal was approved by the Committee for Research on Human Subjects of the University of Kashmir. Permission to conduct the research was granted by the Dean/Principal of GDC Srinagar. All participants were requested to consent in writing before being included in the study.

III. Results

One-hundred subjects of whom 50 were diabetics and 50 non- diabetics were included in the study. The mean age of diabetic group was 49.3 years (SD ± 8.97) and that of non diabetics was 47.6 years (SD ± 8.85). There was no difference in gender distribution and socioeconomic status between the two groups. They also did not differ significantly in terms of their oral health status and perception for need of periodontal treatment.

While describing the involvement of periodontal tissues by gingivitis or periodontitis, it is customary required that both the severity and extent of these diseases be assessed. In this study, we used the mean Plaque component of ramfjord's Periodontal Disease Index (PI) and Papillary Bleeding Index (PBI). On the other hand, mean Periodontal Disease Index (PDI) and mean clinical attachment loss (CAL) were used to determine the severity of periodontitis.

3.1, Plaque Component of Periodontal Disease Index (Schick and Ash Modification) Table - 1, Graph - 1)

The mean plaque index score obtained for the diabetic group was 1.58 ± 0.150 and for the non-diabetic group was 1.56±0.13. 't' value was 0.762 which was statistically non- significant ($p > 0.05$).

On comparison of mean plaque index score for the diabetic and non-diabetic groups, even though the mean plaque index score was observed to be slightly higher in the diabetic group, the difference was statistically non-significant ($p > 0.05$).

3.2, Papillary Bleeding Index (PBI) (Muhlemann H.R 1977): (Table - 2, Graph - 2)

The mean of PBI score obtained for the diabetic group was 2.72±0.396 and for the non-diabetic group was 2.51±0.425. t-value was 2.540, which was statistically significant ($p < 0.01$).

On comparison, higher mean PBI score was found in diabetic group, which was statistically significant, when compared to non- diabetic group.

3.3, Periodontal Disease Index (PDI) (Sigurd P. Ramjord 1967) (Table -3, Graph - 3)

The mean of PDI score obtained for the diabetic group was 3.69 ±0.745 and for the non-diabetic group was 3.19±0.928. 't' value was 2.981 which showed statistically significant difference of PDI between the two groups ($p < 0.05$)

On comparison, higher mean PDI scorer was found in diabetic group, which was statistically significant, when compared to the non-diabetic group.

3.4, Relative Attachment Level (RAL) (Table - 4, Graph - 4)

The mean of relative clinical attachment level score obtained for the diabetic group was 11.80±0.68 and for the non-diabetic group was 11.68±0.683. 't' value was 0.868 which was statistically non- significant between the two groups ($p > 0.05$).

Even though, on comparison mean loss of clinical attachment was observed to be higher in the diabetic group, but the difference was statistically non-significant.

Table 1: Plaque Component of Periodontal Disease Index (Schick and Ash Modification, 1961)

Group	Mean	SD	t-value	P-value	Remarks
Diabetic	1.58	0.150	0.762	0.448	Not Sig.
Non-Diabetic	1.56	0.139			

Table 2: Papillary Bleeding Index (PBI) (Muhlemann H.R 1977)

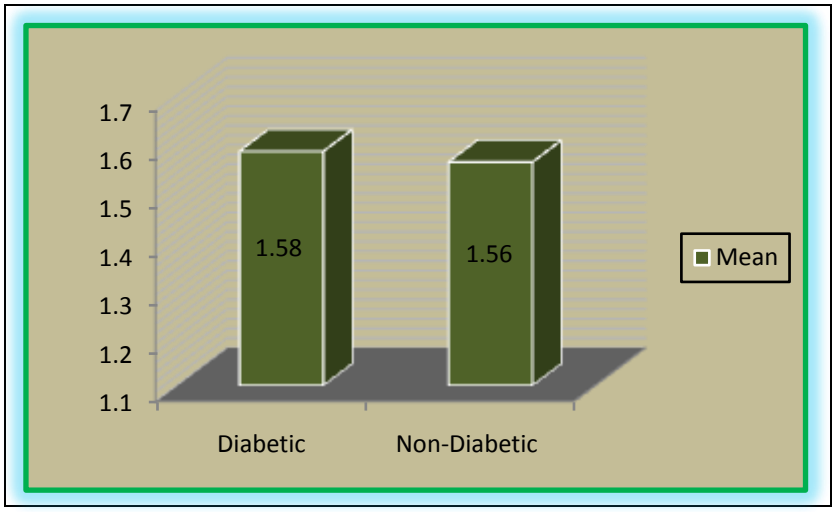
Group	Mean	SD	t-value	P-value	Remarks
Diabetic	2.72	0.396	2.540	0.013	Sig.
Non-Diabetic	2.51	0.425			

Table 3: Periodontal Disease Index (Pdi) (Sigurd P. Ramfjord, 1959)

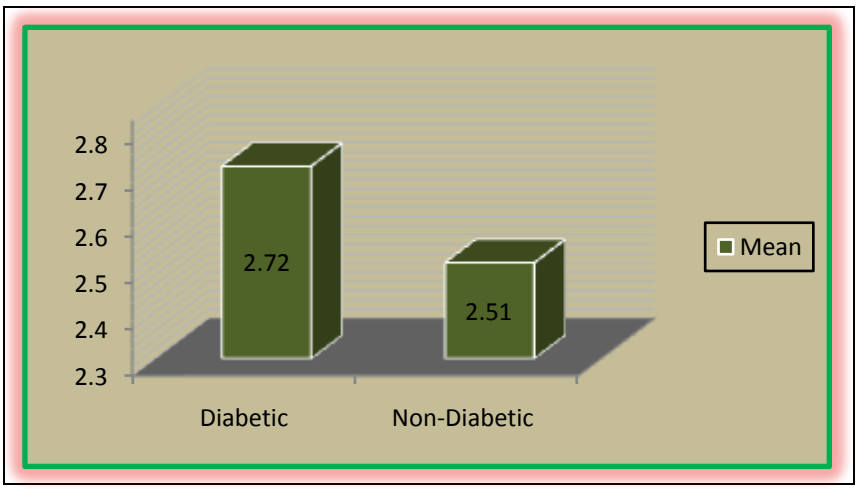
Group	Mean	SD	t-value	P-value	Remarks
Diabetic	3.69	0.745	2.981	0.004	Sig.
Non-Diabetic	3.19	0.928			

Table 4: Relative Attachment level (RAL)

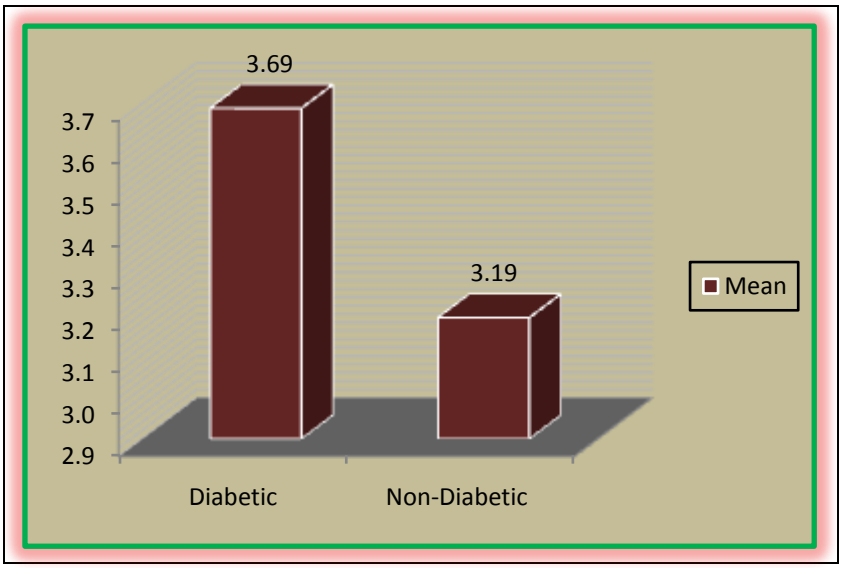
Group	Mean	SD	t-value	P-value	Remarks
Diabetic	11.80	0.700	0.868	0.388	Not Sig.
Non-Diabetic	11.68	0.683			



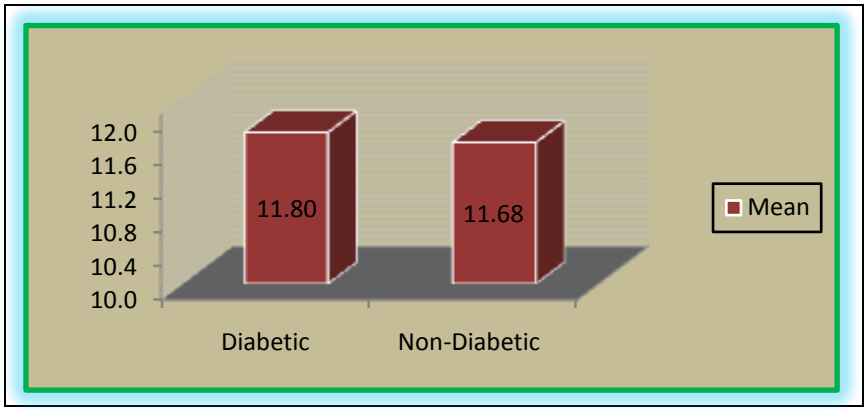
Graph 1: Plaque Component of Periodontal Disease Index (Schick and Ash Modification, 1961)



Graph 2: Papillary Bleeding Index (PBI) (Muhlemann H.R 1977)



Graph 3: Periodontal Disease Index (PDI) (Sigurd P. Ramfjord, 1959)



Graph 4: Relative Attachment level (RAL)

Fig. 1:- Plaque component of ramfjord's periodontal disease index (schick & ash)

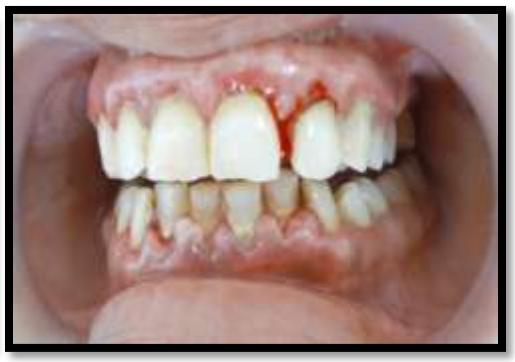


Fig. 2:- Gingival bleeding using papillary bleeding index

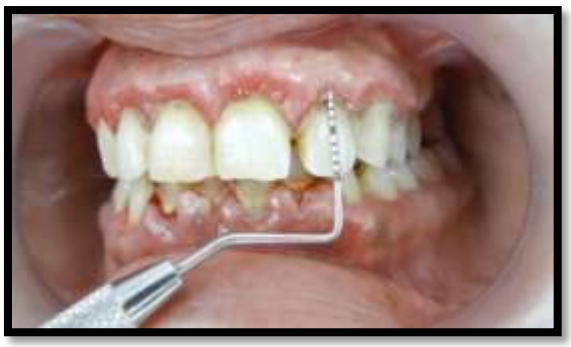


Fig. 3:- Periodontal condition Ramfjord's periodontal disease index

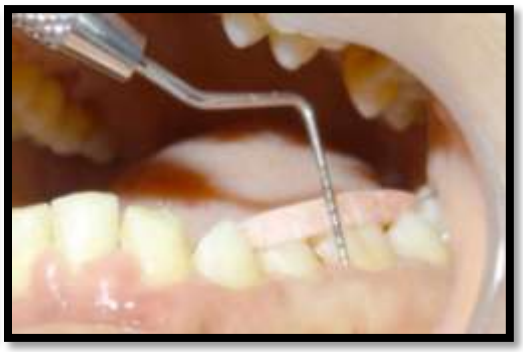


Fig. 4:- Relative attachment level using UNC – 15

IV. Discussion

The aim of this study was to investigate the prevalence and severity of periodontal disease among type 2 diabetics and compare them with non- diabetics controls. Several studies have shown that periodontal disease prevalence is higher and more severe in diabetics than non- diabetics.[21,22,23,24,25] In this study, the means of PD, CAL and GI were significantly higher in the diabetics compared to the non- diabetics. These parameters are the main determinant measures of periodontal disease; it implies that diabetics in this study suffered from more advanced periodontal disease than non- diabetics. This is consistent with the findings of other studies.[21,22,23,24,25] In addition, the mean number of teeth present was significantly lower in diabetics than in non- diabetics. Since advanced periodontal disease ultimately leads to tooth loss, lower number of teeth in diabetics than non diabetics is an expected finding.

On comparison of mean plaque index score for the diabetic and non-diabetic groups, even though the mean plaque index score was observed to be slightly higher in the diabetic group, the difference was statistically non-significant ($p > 0.05$).

The results of this study are in agreement with the studies conducted by Sbordone et al[26], Oliver and Tervonen[27], Sznajder N in 1978[28], Kawamura et al in 1998[29], Salvi et al in 2005[30], Jolanta Siudikiene et al 2005[31], Y.S.Khadir et al 2008[32]

In our study, the difference of mean plaque index score between the diabetic and non- diabetic groups was non significant. This may be because the diabetic subjects considered for present study were undergoing treatment for diabetes and they were following all most similar oral hygiene procedures.

On comparison, higher mean PBI score was found in diabetic group, which was statistically significant, when compared to non- diabetic group.

Similar observations were made by Ervasti et al in 1984[14], Emrich L.J et al 1991[21], De Pommereau V et al in 1992[33], Firatli et al in 1996[34], Sbordone et al in 1998[26], Guglielmo et al in 2005.[35] Y. S. Khader et al 2008[32], Ajita Meenawat et al 2013.[36]

In our study, a significantly higher mean PBI score was observed in diabetics as compared to non-diabetics. This could depict an intensified inflammatory response of the gingiva in the diabetic group, because of shift of subgingival microbiota from predominantly of Grame-positive microflora to predominantly Grame-negative rods and filaments[37]. Also alterations in the hostimmuno-inflammatory response to potential pathogens result in impairment of neutrophil adherence, chemotaxis, and phagocytosis, which facilitate significantly increased production of pro-inflammatory cytokines and mediators[38,39]The net effect of these host defense alterations in diabetes is an increase in periodontal inflammation and bleeding on probing.[40]

On comparison, higher mean PDI scorer was found in diabetic group, which was statistically significant, when compared to the non-diabetic group.

Similar observations were made by Cohen et al in 1970[41], Rylander et al 1987[42], Emrich L.J et al 1991[21], Ngakinya et al in 1997[43], Collin et al in 1998[44], Campus et al in 2005[35], Yousef S Khader et al 2006[45], Raymond B et al 1996[46], Eun-Kyong KIM et al 2013[47] , Ajita Meenawat et al 2013[36].

In our study, a significant difference was found between the means of Periodontal Disease Index score of the diabetics and non- diabetics despite comparable plaque accumulation. This may be because Periodontal Disease Index has both gingival and periodontal components. The gingival component expresses the severity of gingivitis, and the periodontal component expresses the destruction of the underlying tissues. In our study, the mean of Papillary Bleeding Index score was significantly higher in the diabetic group, which indicates the severity of gingivitis in this group. Thus, the gingival component of Periodontal Disease Index may be responsible for significant difference in Periodontal Disease Index between the two groups.

Even though, on comparison mean loss of clinical attachment was observed to be higher in the diabetic group, but the difference was statistically non-significant.

The results of our study are similar to those of Sznajder N et al in 1978[28], Pinson M et al in 1995[48], Sbordone L et al in 1998[26], Yousef S Khader et al 2006[45], which have found no significant difference of relative clinical attachment level between the two groups.

The non-significant difference in means of relative clinical attachment level of diabetic and non-diabetic groups in our study could be because of the multifactorial etiology of periodontal disease and complex nature of diabetes. Even though, there are patients who develop periodontal complications after a short duration of diabetes with a reasonable level of metabolic control, there are some patients who never show periodontal complications even with long-standing poorly controlled disease and a poor oral hygiene. This is probably due to a variation in individual susceptibility assumed to be related to differences in genetic background.[49]

This study has some limitations. Firstly, being a cross-sectional study, the results do not represent a causal association between type 2 diabetes and periodontal disease. However, the findings of this study add to the existing literature. Secondly, the study population was hospital-based and may not be representative of the general population. Thirdly, scoring for periodontal disease was done on index teeth or their substitutes. If this is so, then underestimation of periodontal disease occurred equally to both diabetics and non-diabetics. It therefore did not influence our results.

In conclusion, the results of this study are in agreement with those of other studies in that diabetes mellitus increases the prevalence and severity of periodontal disease. Diabetics and their health care givers should be well informed of this association so that appropriate measures are taken to monitor for early signs of periodontal disease.

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