Periodontal Disease as a Complication in Type 2 Diabetes Mellitus—A Hospital Based Study in Uttarakhand, India

Authors

Dr Aviral Shah¹, Dr Rupa Kumari², Dr Abhishek Khandwal³
¹MD Medicine, ²DNB Second year Obstetrics and Gynecology, ³MDS
Email: aviralshahh@gmail.com

Abstract

Objectives: To study the prevalence, association and correlation of periodontal disease in patients with type 2 diabetes mellitus (T2DM).

Methods: One hundred consecutive patients with T2DM and an equal number of age- and sex-matched non-diabetic controls were studied. Diabetes was diagnosed as per American Diabetic Association criteria. All subjects were evaluated for presence and severity of periodontal disease activity clinically and radiologically.

Results: Prevalence of periodontitis in cases and controls was not significantly different as per clinical attachment loss (CAL) (97% versus 90%, p=0.085) and radiological assessment (97% versus 89%, p=0.052) but prevalence of severe periodontitis was higher among cases (p=0.001). Gingival index and periodontal pocket depth were significantly higher among cases (p=0.001 each). Diabetes had a significant association with severity of CAL (p=0.001) and radiological damage (p=0.001) but not with CAL (p=0.085) and radiological damage (p=0.052). Severity of periodontitis had a significant association with glycosylated haemoglobin (HbA1c) level (p=0.001), fasting blood sugar (FBS) (p=0.001) and post-prandial blood sugar (PPBS) (p=0.011) while radiological severity of periodontitis had a significant association with HbA1c (p=0.001) and FBS (p=0.01). CAL showed a significant correlation with HbA1c (r=0.708, p=0.0001), FBS (r=0.476, p=0.001), PPBS (r=0.425, p=0.001) and random blood sugar (r=0.476, p=0.001) but not with age (r=0.061, p=0.393), and duration of diabetes (r= -0.024, p=0.811). Both CAL and radiological severity were independently associated with HbA1c (p=0.000 each).

Conclusions: Periodontitis is an important complication in T2DM. Severity of periodontitis is associated with diabetes and the related parameters, while HbA1c is independently associated with periodontitis.

Significant findings of the study

• Diabetes increases the severity of periodontitis, assessed by the clinical as well as radiological examination.

• Severity of periodontitis has significant correlation with the blood sugar and glycosylated haemoglobin levels.

What this study adds?

• Glycosylated haemoglobin is independently associated with the severity of periodontitis ascertained by clinical attachment loss and radiological examination.

Keywords: Clinical attachment loss; Diabetic complication; India; Periodontitis; Radiological evaluation; Type 2 diabetes mellitus.
Introduction
Diabetes has emerged as a major health problem in India. According to International Diabetes Federation every fifth diabetic in world would be an Indian by the year 2025. Periodontitis has been considered as the sixth complication of diabetes, which puts Indian population at a greater risk of developing it. The relation between periodontal destruction and diabetes mellitus make diabetic screening essential in periodontitis. Diabetes is a metabolic disorder affecting cellular and biochemical processes within the body. Patients with diabetes often have decreased immunity, exhibiting increased susceptibility to infection. Diabetes mellitus has multiple effects on oral tissues and it influences the prevalence and severity of periodontal disease. A study done on the Pirna Indians, a community with the world's highest reported prevalence of non-insulin dependent diabetes mellitus (NIDDM), demonstrated a clear relationship between the prevalence, severity, and incidence of periodontal disease. Likewise, a 50% increase in messenger RNA for the receptor of advanced glycation end-products was recently identified in the gingival tissues of type 2 diabetic subjects compared to non-diabetic controls. Matrix metalloproteinases are critical components of tissue homeostasis and wound healing, and are produced by all of the major cell types in the periodontium. Production of matrix metalloproteinases such as collagenase increases in many diabetic patients, resulting in altered collagen homeostasis and wound healing within the periodontium. This study was done to assess the prevalence of periodontal disease in patients with type 2 diabetes mellitus and the association and correlation of periodontal disease with type 2 diabetes mellitus.

Methods
This cross-sectional study was conducted in the Department of Medicine, Himalayan Institute of Medical Sciences (HIMS), Dehradun, India from July 2013 to June 2014. The protocol for the research project was approved by a suitably constituted Ethics Committee of the institution within which the work was undertaken and it conforms to the provisions of the Declaration of Helsinki as revised in Seoul, 2008. One hundred consecutive patients with type 2 diabetes mellitus were recruited from patients presenting in Medicine OPD, after obtaining written informed consent from the patients. Diagnosis of type 2 diabetes mellitus was made as per American Diabetic Association criteria. An equal number of non-diabetic patients of same age and sex were taken as controls.

Inclusion criteria:
• Patients with type 2 diabetes mellitus.
• Patients of age group 35-65 years.

Exclusion criteria:
• Patients who had undergone periodontal treatment in the 6 months’ period prior to the study.
• History of antibiotic administration within the last 3 months.
• Pregnant women.
• Post-menopausal women

Same exclusion criteria were applicable to controls as well. All enrolled patients were subjected to detailed history and thorough physical examination. Investigations done were complete hemogram, urinalysis, blood sugar-fasting, post-prandial and random, HbA1c (Glycosylated haemoglobin), blood urea nitrogen, serum creatinine, sodium, potassium, bilirubin (total/direct/indirect), alanine aminotransferase (ALT), aspartate aminotransferase (AST), total proteins, albumin, globulin, A/G ratio, lipid profile, electrocardiography (ECG) and fundus examination. All diabetic patients underwent dental examination and were evaluated for presence and severity of periodontal disease activity. Periodontitis was graded clinically on the basis of criteria given below.

Clinical Attachment Loss. Healthy = Clinical attachment loss of 0 mm
Slight = Clinical attachment loss of 1.0 to 2.0 mm
Moderate = Clinical attachment loss of 3.0 to 4.0 mm
Severe = Clinical attachment loss equal to or greater than 5.0 mm

Orthopantomogram was done for radiological evaluation of all periodontitis patients and was graded as given below. A single P score for the horizontal and/or vertical periodontal bone loss, determined from the orthopantomogram, was given for each subject.

P0 = no marginal bone loss
P1 = mild marginal bone loss throughout the dentition or at several sites, but not exceeding 30% of the root length anywhere in the dentition
P2 = moderate marginal alveolar bone loss, involving at least 30% of the root length throughout the dentition or at several sites, but not exceeding 50% bone loss anywhere in the dentition.
P3 = advanced marginal alveolar bone loss, involving at least 50% of the root length throughout the dentition, or at several sites.

Statistical analysis
All data were analyzed with SPSS software version 17.0. The data were expressed as mean ± S.D. for continuous variables and as frequency or percentage for categorical variables. Chi-square test was used for statistical comparison of qualitative variables and for determining their association. Student’s t-test was used for comparison of parametric variables. Correlation between different parameters was assessed by Pearson product moment correlation coefficient. Multivariate analysis using multiple linear regression was done to determine the diabetes related parameters having independent association with clinical and radiological disease severity. 'P' values of less than 0.05 were considered statistically significant.

Results
Baseline characteristics of diabetic patients and controls are shown in table 1. Mean age of patients was 44.73±6.27 years and of controls 43.23±6.26 years. Majority of patients belonged to 35-45 years of age group and were predominantly males. Body weight (p=0.007), height (p=0.001) and body mass index (0.005) were significantly more in cases than in controls. Majority of patients (68%) had diabetes for 1 – 5 years. (Table 1)

Most common symptoms among diabetic patients were unexplained weight loss (46%) followed by blurring of vision (37%), increased thirst (32%), foot pain and numbness (31%), frequent micturition (28%) and increased hunger (22%).

Most common signs were skin lesions (18%) followed by postural hypotension (16%), loss of ankle jerk (14%), macular edema (12%), muscle wasting (8%) and neuropathic foot ulcer (4%).

Diabetic retinopathy (34%) was the commonest complication in diabetic patients followed by diabetic neuropathy (18%) and nephropathy (13%). (Table 2).

Most common symptoms of periodontitis in diabetic cases were bleeding gums (26%), halitosis (16%) and mobility of teeth (12%). Most common signs were gingival inflammation (27%), gum recession (21%) and pus formation (18%). Blood urea nitrogen, serum creatinine, fasting blood sugar, post prandial blood sugar, random blood sugar and glycosylated haemoglobin were significantly higher among cases than controls (p>0.05 each) (Table 3).

Gingival index and periodontal pocket depth were significantly higher among cases than controls (p=0.001 each). Mean clinical attachment losses of molar (p=0.001) and non-molar (p=0.001) teeth were significantly higher among cases than controls. Mean clinical attachment losses of upper jaw (p=0.001) and lower jaw (p=0.001) were significantly higher among cases than controls.

There was no significant difference between cases and controls regarding prevalence of periodontitis
(p=0.713), but prevalence of severe periodontitis was higher among diabetic patients than controls (p=0.001) (Table 4).

Periodontitis did not show a significant association with age, BMI, HbA1c, duration of diabetes and fasting, post-prandial and random blood sugar levels (p>0.05 each). Severity of periodontitis did not have a significant association with age (p=0.614), gender (p=0.928) and body mass index (p=0.975). A statistically significant association was seen between severity of periodontitis and glycosylated haemoglobin level (p=0.001). Duration of diabetes was not significantly associated with severity of periodontitis (p=0.697). Radiological severity of periodontitis showed a significant association with HbA1c (p=0.001) and fasting blood sugar (p=0.01) (Table 5). Diabetes had a significant association with severity of clinical attachment loss (p=0.001) but not with clinical attachment loss (p=0.085). Diabetes did not show a significant association with radiological damage (p=0.052) but had a highly significant association with radiological severity of periodontitis (p=0.001) (Table 6).

Severity of periodontitis showed a highly significant correlation with fasting blood sugar (r=0.476, p=0.001), post-prandial blood sugar (r=0.425, p=0.001), random blood sugar (r=0.476, p=0.001) and glycosylated haemoglobin (r=0.708, p=0.001) but not with duration of diabetes (r=-0.024, p=0.811) (Table 7).

Clinical attachment loss HbA1c showed an independent association with HbA1c (p=0.000) but not with FBS (p=0.141) and post-prandial blood sugar (p=0.645) (Table 8). Radiological severity showed an independent association with HbA1c (p=0.000) but not with fasting blood sugar (p=0.214) (Table 9).

Table 1: Baseline characteristics of diabetic cases (n=100) and controls (n=100)

| Parameters                  | No. of cases (%) / Mean ± SD | No. of controls (%) / Mean ± SD | P value |
|-----------------------------|------------------------------|---------------------------------|---------|
| Mean age (years)            | 44.73±6.27                   | 43.23±6.26                      | 0.092   |
| Age group (years)           |                              |                                 |         |
| 35 – 45                     | 59 (59%)                     | 59 (59%)                        |         |
| 46 – 55                     | 35 (35%)                     | 35 (35%)                        |         |
| 56 – 65                     | 6 (6%)                       | 6 (6%)                          |         |
| Gender                      |                              |                                 |         |
| Males                       | 61 (61%)                     | 61 (61%)                        |         |
| Females                     | 39 (39%)                     | 39 (39%)                        |         |
| Residence                   |                              |                                 |         |
| Rural                       | 68 (68%)                     | 73 (73%)                        | 0.747   |
| Urban                       | 32 (32%)                     | 27 (27%)                        | 0.567   |
| Body Weight (kg)            | 65.22±12.47                  | 61.24±7.89                      | 0.007   |
| Height (cm)                 | 165.38±9.65                  | 160.8±6.74                      | 0.001   |
| BMI† (kg/m²)                | 25.15±4.40                   | 23.67±2.97                      | 0.005   |
| Duration of diabetes (years)|                              |                                 |         |
| 1-5                         | 68 (68%)                     | 0                               | -       |
| 6-10                        | 23 (23%)                     | 0                               | -       |
| > 10                        | 9 (9%)                       | 0                               | -       |
| Mean duration of T2DM‡ (years)| 4.92±3.79                  | 0                               | -       |

†-Body mass index; ‡-Type 2 diabetes mellitus
Table 2: Complications of type 2 diabetes mellitus

| Complications                        | No. of Cases (n=100) | Percentage |
|--------------------------------------|----------------------|------------|
| Retinopathy                          | 34                   | 34%        |
| Neuropathy                           | 18                   | 18%        |
| Nephropathy                          | 13                   | 13%        |
| Coronary circulation                 |                      |            |
| Myocardial infarction                | 8                    | 8%         |
| Cerebral circulation                 |                      |            |
| Transient ischemic attack            | 4                    | 4%         |
| Stroke                               | 2                    | 2%         |
| Peripheral circulation               |                      |            |
| Claudication                         | 12                   | 12%        |
| Ischemia                             | 3                    | 3%         |

Table 3: Showing Clinical profile of periodontitis and laboratory parameters among cases (n=100) and controls (n=100)

| Clinical Features | Number of Cases (%) | Number of Controls (%) | P value |
|-------------------|---------------------|------------------------|---------|
| Bleeding gums     | 26(26%)             | 20(20%)                | 0.425   |
| Halitosis         | 16(16%)             | 26(26%)                | 0.161   |
| Mobility of teeth | 12(12%)             | 14(14%)                | 0.712   |
| Gingival inflammation | 27(27%)          | 8(8%)                  | 0.003   |
| Gum recession     | 21(21%)             | 19(19%)                | 0.773   |
| Pus formation     | 18(18%)             | 15(15%)                | 0.629   |
| Dull gnawing pain | 13(13%)             | 19(19%)                | 0.325   |
| Bleeding on probing | 10(10%)         | 18(18%)                | 0.157   |

| Laboratory Parameters | Number of Cases (gm/dl) | Number of Controls (gm/dl) | P value |
|-----------------------|-------------------------|---------------------------|---------|
| Hb†                   | 12.79±1.53              | 13.86±10.91               | 0.332   |
| TLC‡                  | 1311.88±3373.82         | 1394.59±3069.86           | 0.856   |
| Platelet count        | 207.82±60.20            | 279.42±748.95             | 0.341   |
| Serum creatinine      | 1.34±1.69               | 0.84±0.34                 | 0.004   |
| Blood Urea Nitrogen   | 24.40±17.77             | 17.78±11.48               | 0.002   |
| FBS§                  | 163.09±45.87            | 90.26±16.66               | 0.001   |
| PPBS¶                 | 219.72±75.16            | 122.97±29.17              | 0.001   |
| RBS#                  | 177.41±37.99            | 98.45±9.57                | 0.001   |
| HbA1c††                | 8.83±2.31               | 4.91±0.61                 | 0.001   |
| Total bilirubin       | 1.04±0.29               | 1.01±0.36                 | 0.001   |
| Direct bilirubin      | 0.65±0.23               | 0.65±0.22                 | 1.00    |
| Indirect bilirubin    | 0.56±1.77               | 0.35±0.22                 | 0.240   |
| ALT‡‡                 | 18.45±16.58             | 22.66±21.20               | 0.119   |
| AST||                  | 25.63±33.35             | 28.89±42.69               | 0.548   |
| Serum Total Protein   | 7.12±0.83               | 7.26±0.54                 | 0.159   |
| Serum Albumin         | 4.19±0.67               | 4.35±0.53                 | 0.062   |
| Serum Globulin        | 3.20±2.28               | 3.23±3.17                 | 0.938   |

†- Hemoglobin; ‡-Total leucocyte count; § - Fasting blood sugar; ¶ - Post-prandial blood sugar; # - Random blood sugar; †† -Glycosylated haemoglobin; ‡‡ - Alanine transaminase; || - Aspartate aminotransferase
Table 4: Showing prevalence of periodontitis related parameters in cases (n=100) and controls (n=100)

| Parameters               | No. of Cases (%)/ mean±SD | No. of Controls (%)/ mean±SD | P value |
|--------------------------|----------------------------|------------------------------|---------|
| Healthy                  | 3 (3%) / 6.47±2.32         | 10 (10%) / 4.17±1.94        | 0.591   |
| Periodontitis            | 97 (97%) / 4.96±2.32       | 90 (90%) / 4.17±1.94        | 0.713   |
| Mild                     | 16 (16%) / 6.47±2.32       | 40 (40%) / 4.17±1.94        | 0.004   |
| Moderate                 | 27 (27%) / 5.46±2.46       | 35 (35%) / 4.17±1.94        | 0.375   |
| Severe                   | 54 (54%) / 5.75±2.32       | 15 (15%) / 4.17±1.94        | 0.001   |
| Gingival index           | 2.07±0.80 / 4.03±1.92      | 1.41±0.75 / 4.17±1.94       | 0.001   |
| Periodontal pocket depth |                           |                             |         |
| (mm)                     | Mean clinical attachment loss of molar teeth | 5.46±2.46 | 3.55±2.04 | 0.001 |
|                          | Mean clinical attachment loss of non-molar teeth | 4.96±2.36 | 3.12±1.95 | 0.001 |
|                          | Mean clinical attachment loss of upper jaw | 5.84±2.38 | 4.17±1.94 | 0.001 |
|                          | Mean clinical attachment loss of lower jaw | 5.75±2.32 | 4.03±1.92 | 0.001 |

Table 5: Association of diabetes related parameters with periodontitis and its severity

| Parameters               | No. of patients | P value | No. of patients | P value | No. of patients | Radiological severity | P value |
|--------------------------|-----------------|---------|-----------------|---------|-----------------|------------------------|---------|
| Age Group                | Present | Absent | Mild | Moderate | Severe | Present | Absent | Mild | Moderate | Severe | P1 | P2 | P3 | P value |
| 35 – 45                  | 57      | 2      | 11   | 14       | 32     | 0.614    | 12      | 12   | 33       | 0.906  |
| 46 – 55                  | 34      | 1      | 5    | 10       | 19     | 0.643    | 9       | 14   | 35       | 0.637  |
| 56 – 65                  | 6       | 0      | 0    | 3        | 3      | 0.769    | 6       | 10   | 21       | 0.552  |
| Males                    | 58      | 3      | 8    | 16       | 34     | 0.643    | 9       | 14   | 35       | 0.552  |
| Females                  | 39      | 0      | 8    | 11       | 20     | 0.906    | 9       | 9    | 21       | 0.702  |
| BMI (kg/m²)†             | Present | Absent | Mild | Moderate | Severe | Present | Absent | Mild | Moderate | Severe | P1 | P2 | P3 | P value |
| <18.5                    | 13      | 0      | 4    | 3        | 6      | 0.614    | 16      | 1    | 8        | 0.702  |
| 18.5 – 23.9              | 37      | 2      | 6    | 10       | 21     | 0.769    | 3       | 8    | 12       | 0.702  |
| 24 – 26.9                | 23      | 1      | 2    | 9        | 12     | 0.643    | 5       | 5    | 14       | 0.702  |
| > 27                     | 24      | 0      | 4    | 6        | 14     | 0.643    | 5       | 5    | 14       | 0.702  |
| HbA1c‡ (%)               | Good control | 17    | 1    | 0.619   | 16      | 1    | 0     | 0.001 | 16 | 1     | 0  | 0.001 |
|                          | Fair control | 19    | 1    | 0.598   | 16      | 1    | 0     | 0.001 | 16 | 1     | 0  | 0.001 |
|                          | Poor control | 61    | 1    | 0.809   | 16      | 1    | 0     | 0.001 | 16 | 1     | 0  | 0.001 |
| Duration of T2DM         | Present | Absent | Mild | Moderate | Severe | Present | Absent | Mild | Moderate | Severe | P1 | P2 | P3 | P value |
| 1 – 5 years              | 66      | 2      | 8    | 23       | 35     | 0.697    | 11      | 19   | 36       | 0.552  |
| 6 – 10 years             | 22      | 1      | 4    | 5        | 13     | 0.697    | 5       | 3    | 14       | 0.552  |
| > 10 years               | 9       | 0      | 2    | 1        | 6      | 0.697    | 2       | 1    | 6        | 0.552  |
| FBS§                     | < 126   | 16     | 1    | 0.813   | 7       | 6      | 0.004   | 7     | 6     | 3  | 0.01 |
|                          | 126 - 200 | 66    | 2    | 8       | 20     | 38      | 0.697   | 10    | 16    | 40 | 0.05 |
|                          | 201 - 300 | 13    | 0    | 1       | 11     | 11      | 0.697   | 1     | 1     | 11 | 0.05 |
|                          | > 300   | 2      | 0    | 0       | 2      | 0.697   | 0       | 0    | 0        | 0.05 |
| PPBS¶                    | < 140   | 7      | 1    | 0.162   | 4       | 2      | 0.011   | 4     | 2     | 1  | 0.05 |

Dr Aviral Shah et al JMSCR Volume 04 Issue 04 April
141 - 200  43  0  5  16  22  7  13  23
201- 300  33  2  8  5  20  5  7  21
> 300     14  0  2  1  11  2  1  11

RBS\
≤ 200    76  2  0.529 14  21  41  0.612 16  17  43  0.465
> 200    21  1  2  6  13  2  6  13

†-Body mass index; ‡-Glycosylated haemoglobin; T2DM-type 2 diabetes mellitus; § - Fasting blood sugar; ¶ - Post-prandial blood sugar; # - Random blood sugar

Table 6: Association of diabetes with clinical attachment loss and radiological damage and their severity

| Parameters                      | Number of cases (%) | Number of controls (%) | P value |
|---------------------------------|---------------------|------------------------|---------|
| **Clinical Attachment Loss (mm)** |                     |                        |         |
| Absent                          | 3 (3%)              | 10 (10%)               | 0.085   |
| Present                         | 97 (97%)            | 90 (90%)               |         |
| **Severity of Clinical Attachment Loss (mm)** | | | |
| Slight (1-2mm)                  | 16 (16%)            | 40 (40%)               | 0.001   |
| Moderate (3-4mm)                | 27 (27%)            | 35 (35%)               |         |
| Severe (>5mm)                   | 54 (54%)            | 15 (15%)               |         |
| **Radiological damage**         |                     |                        |         |
| Healthy                         | 3 (3%)              | 11 (11%)               | 0.052   |
| Diseased                        | 97 (97%)            | 89 (89%)               |         |
| **Severity of Radiological damage** |                   |                         |         |
| P1                              | 18 (16%)            | 38 (40%)               | 0.001   |
| P2                              | 23 (27%)            | 37 (35%)               |         |
| P3                              | 56 (54%)            | 14 (15%)               |         |

Table 7: Univariate analysis showing correlation between diabetes related parameters and clinical attachment loss

| Predictor variable     | Dependent variable | R    | p value |
|------------------------|--------------------|------|---------|
| Age                    | Clinical Attachment Loss | 0.061| 0.393   |
| Fasting blood sugar    |                     | 0.476| 0.001   |
| Post-prandial blood sugar |                 | 0.425| 0.001   |
| Random blood sugar     |                     | 0.476| 0.0001  |
| HbA1c level†           |                     | 0.708| 0.0001  |
| Duration of diabetes   |                     | -0.024| 0.811  |

†-Glycosylated hemoglobin
Table 8: Multivariate analysis showing diabetes related parameters having independent association with clinical attachment loss (CAL) Coefficients

| Model   | Unstandardized Coefficients | Standardized Coefficients | 95.0% Confidence Interval for B |
|---------|----------------------------|---------------------------|--------------------------------|
|         | B                          | Std. Error                | Beta              | t     | Sig. | Lower Bound | Upper Bound |
| 1       | 1.08                       | .308                      | .352              | .726  | -5.03 | .720        |
| FBS† (mg/dl) | .002                  | .002                      | .109              | 1.250 | .214  | -.001       | .005        |
| PPBS‡ (mg/dl) | -.002                 | .044                      | -.050             | -.463 | .645  | -.009       | .005        |
| HBA1c§ (%) | .771                    | .075                      | .720              | 10.265| .000  | .622        | .920        |

Dependent Variable: Clinical attachment loss
† - Fasting blood sugar; ‡ - Post-prandial blood sugar; § - Glycosylated hemoglobin

Table 9: Multivariate analysis showing diabetes related parameters having independent association with radiological severity Coefficients

| Model   | Unstandardized Coefficients | Standardized Coefficients | 95.0% Confidence Interval for B |
|---------|----------------------------|---------------------------|--------------------------------|
|         | B                          | Std. Error                | Beta              | t     | Sig. | Lower Bound | Upper Bound |
| 1       | 1.08                       | .308                      | .352              | .726  | -5.03 | .720        |
| FBS† (mg/dl) | .002                  | .002                      | .109              | 1.250 | .214  | -.001       | .005        |
| HBA1c§ (%) | .214                    | .033                      | .571              | 6.547 | .000  | .149        | .278        |

a. Dependent Variable: Radiological severity
† - Fasting blood sugar; ‡ - Glycosylated hemoglobin

Discussion

Periodontitis is a multifactorial disease which primarily progresses due to plaque accumulation and many other risk factors such as environmental, genetic, microbial and host immunity over a period of time. Glucose content of gingival crevicular fluid and blood increases in diabetes mellitus as compared to non-diabetic patients. This changes microflora of periodontal pockets.

A study conducted by Shlossman et al showed a relationship between type 2 diabetes mellitus and periodontal disease. Emrich et al found a significant correlation between diabetic status, age, presence of sub-gingival calculus and increased prevalence and greater severity of destructive periodontal disease and that diabetes increases the risk of developing destructive periodontal disease threefold. On the other hand, diabetic subjects with severe periodontitis at baseline had a six-fold increased risk of worsening of glycaemic control over time compared to diabetic subjects without periodontitis. The age range of the patients in present study was 35-65 years with mean age 44.73 ± 6.27 years for cases and 43.23±6.26 years for controls. Periodontal disease and its relationship with age has been a debatable question. Landmark studies of Schurch et al, Beck et al, and Papapanou et al have reported worsening of periodontal scores with age. However in our study, age matched controls were enrolled to eliminate any confounding effect of same and age was not
significantly associated with the severity of periodontitis. Similar results were reported by a study in Pima Indians which showed type 2 diabetes was associated with severity of periodontitis independent of age.  

Gender is considered as one of the confounding factors in periodontitis. A study reported higher prevalence of periodontitis in males due to tobacco use and higher incidence in females due to effect of progesterone and oestrogen interplay on periodontium. In present study, gender matched individuals were enrolled as controls to eliminate aforementioned bias. There was no effect of gender on the relationship of type 2 diabetes with periodontitis. Similar results were reported by a study on Pima Indians which showed type 2 diabetes was associated with severity of periodontitis independent of gender.

Genco et al. reported that BMI positively correlated with the severity of periodontal attachment loss and this relationship is modulated by insulin resistance. In present study, cases had significantly higher BMI score as compared to controls which could be an aggravating factor for diabetes as well as periodontitis. However, no significant association was found between severity of periodontitis and BMI scores in T2DM.

Duration of diabetes and severity of periodontitis have been studied and contradictory results have been reported. Briggs et al conducted a cross-sectional study and compared the periodontal status of 118 diabetic men and found that duration of diabetes was not significantly related to periodontal status. Cerda et al reported that patients with more than 5 years of diagnosed diabetes had higher CAL scores as compared to those with diabetes for less than 5 years. However, in our study, duration of diabetes was not significantly associated with severity of periodontitis. Since majority of patient in our study belonged to rural population so the date of diagnosis and onset of disease may have wide time lag. This delay in diagnosis could be mainly attributed to low health awareness among population.

The results of present study suggested that 97% of the diabetic subjects were having at least some amount of periodontal destruction and 90% of cases had some amount of periodontal disease. Similar to our findings, Kumar et al reported prevalence of periodontitis as 91.7% while Mansour et al and Zang et al reported prevalence of periodontitis as 96.7% and 95.9% respectively in type 2 diabetic population. A high prevalence of periodontitis in diabetic cases supports the view that periodontitis is the sixth complication of diabetes mellitus and both diseases are linked bi-directionally. Whereas, one study reported lesser prevalence as it only recorded periodontitis when clinical attachment loss exceeded 3 or 4 mm. Similarly, Tsai et al recorded only severe cases defined by clinical attachment loss > 6 mm, not taking in account the amount of moderate attachment loss and hence reported lower prevalence of periodontitis. Our study more precisely recorded even the slight attachment loss as less as one mm thus identifying the actual periodontal disease burden in diabetic population. However, no significant difference was observed between prevalence of periodontitis among diabetic cases and controls.

A high prevalence of periodontitis was also observed for controls, as they were exclusively taken from periodontal practice of dental department which reported higher number of patients with periodontitis who were otherwise healthy.

Clinical attachment loss is considered yard stick to measure effective periodontal status. Diabetes is associated with 6 fold increase in periodontitis status as measured by clinical attachment loss scores. In our study, majority of cases (54%) had severe periodontitis while majority of controls had mild to moderate periodontitis (75%). A significant association was seen between diabetes and severity of clinical attachment loss. Similar findings have been reported by Novak et al.
Orthopantomogram has been used to assess the severity of periodontitis. Ours was the first study which confirmed the clinical attachment loss by demonstrating radiographic attachment loss or radiographic alveolar bone loss to verify the status of periodontitis and the radiographic scores corroborated the clinical attachment scores.

Type 2 diabetes and decreased insulin sensitivity are associated with the production of advanced glycation end products (AGE), which trigger inflammatory cytokine production, thus predisposing to inflammatory diseases such as periodontitis.4

In present study with worsening of glycosylated haemoglobin scores, higher clinical attachment loss scores were reported. Among cases, 54 % had severe periodontitis with poor glycaemic control while in controls only 15 % of individuals had severe periodontitis. This shows that progression of periodontitis is higher in diabetic than in non-diabetic patients. Similar findings were reported by Nelson et al 8 and Zambon et al.17

Periodontal pocket depth is also used as a measuring tool to classify periodontal disease though it is less accurate as compared to clinical attachment loss. Deeper periodontal pockets are seen in diabetic patients. Campus et al26 studied 212 patients and found significantly increased periodontal probing depth among cases. Our study also showed that with worsening of glycaemic control, increase in pocket depth was seen.

Very few studies have evaluated the differences in molar and non-molar teeth for attachment loss while evaluating for periodontitis and diabetes. In our study, we observed that molar teeth had higher values of clinical attachment loss and periodontal probing depth scores as compared to non-molars in diabetic cases. Similar findings were seen by Bandyopadhyay et al, in Gullah African Americans population.27 This could be explained as molar teeth are multi-rooted and have furcation area. This anatomical difference from non-molar teeth provides additional points for clinical attachment loss and progression of periodontitis and hence higher values of clinical attachment loss and periodontal probing depth scores.

Gingival Bleeding is an indicator of inflammation and is one of the first clinical sign of periodontitis. It may also be possible that vascular changes in diabetes mellitus result in increased gingival bleeding. The relation of the control of diabetes to development of vascular changes has been studied by Tchobroutsky 28, who observed less vascular changes in well controlled diabetes as compared to poorly controlled diabetes. In our study also, diabetic cases had higher mean gingival score as compared to controls. This could be explained as among cases, 54 % individuals had severe periodontitis while among controls, only 15 % of individuals had severe periodontitis. Similar findings were seen by Sznajder et al29, who showed a higher level of gingival bleeding among diabetic cases than controls in their study.

Apoorva et al30 reported that severity of periodontal disease increases with fasting blood sugar level. To best of our knowledge, our study was the first study to evaluate correlation of fasting, post-prandial and random blood sugars and glycosylated haemoglobin with periodontitis and showed that fasting, post-prandial and random blood sugars and glycosylated hemoglobin correlated significantly with severity of periodontitis. However, out of fasting and post-prandial blood sugars and glycosylated haemoglobin, only glycosylated haemoglobin was found to have independent association with severity of clinical attachment loss. This is supported by our finding that out of factors having significant association with radiological severity of periodontitis, only glycosylated haemoglobin was independently associated with radiological severity.

Thus diabetes appears to have a direct impact on development and/or increase in severity of periodontitis.
Conclusions
Periodontitis was a common finding in type 2 diabetic patients and severe periodontitis was more frequent in diabetic patients than in controls. Clinical attachment loss and radiological severity of periodontitis had significant association with diabetes. Severity of periodontitis had significant correlation with blood sugar and glycosylated haemoglobin while glycosylated haemoglobin was independently associated with severity of periodontitis. Hence, periodontal examination should be included as an integral part of examination in type 2 diabetic patients as detection of periodontitis at an early stage can help in preventing its progression and also ensure a better control of diabetes.

Acknowledgments
We are thankful to the Himalayan Institute Hospital Trust (Dehradun, Uttarakhand, India) for funding this project.

Disclosure: None declared

References
1. Kumar A, Pandey MK, Singh A, et al. Prevalence and severity of periodontal diseases in type 2 diabetes mellitus of Bareilly region (India). Int J Med Sci Public Health. 2013; 2: 77-83.
2. Anil S, Remani P, Vijayakumar FT, et al. Total Hemolytic Complement (CH50) and Its Fractions (C3 and C4) in the Sera of Diabetic Patients With Periodontitis. J Periodontol 1990;61:27-29.
3. Cerda J, Torre CV, Malacara JM, et al. Periodontal disease in non-insulin dependent diabetes mellitus (NIDDM). The effect of age and time since diagnosis. J Periodontol. 1994; 65: 991-5.
4. Yan SF, Ramaswamy R, Schimdt AM, et al. Receptors for AGE (RAGE) and its ligands—cast into leading roles in diabetic and the inflammatory response. J Mol Med (Berl). 2009; 87: 235-47.
5. Yan SF, Ramaswamy R, Schimdt AM, et al. Mechanisms of disease: advanced glycation end-product and their receptor in inflammation and diabetes complications. Nat Clin Pract Endocrinol & Metab. 2008; 4: 285-93.
6. Longo DL, Fauci AS, Kasper DL, et al. Editors. Harrison’s Principles of Internal Medicine. 18th ed. New York: McGraw-Hill; 2012; 2: 2970-71.
7. Armitage GC. Periodontal diagnosis and classification of periodontal diseases. Periodontol 2000. 2004; 34:9-21.
8. Sewón L, Parvinen T. The prevalence of periodontal bone loss in Finnish adults measured using simplified radiographic criteria. Proc Finn Dent Soc. 1988; 84:79-83.
9. Nelson RG, Shlossman M, Budding LM, et al. Periodontal disease and NIDDM in Pima Indians. Diabetes care. 1990; 13:836-40.
10. Ficara AI, Levin MP, Grover ME, et al. A comparison of the glucose and protein content of gingival fluid from diabetics and non-diabetics. Periodontal Res. 1975; 10:71-4.
11. Shlossman M, Knowler WC, Pettitt DJ, et al. Type 2 diabetes mellitus and periodontal disease. J Am Dent Assoc. 1990; 121:532-6.
12. Emrich LJ, Shlossman M, Genco RJ, et al. Periodontal disease in non-insulin-dependent diabetes mellitus. J Periodontol. 1991; 52:123-31.
13. Taylor GW, Burt BA, Becker MP, et al. Non-insulin dependent diabetes mellitus and alveolar bone loss progression over 2 years. J Periodontal. 1998; 69: 76-83.
14. Schurch E, Minder CE, Lang NP, et al. Periodontal conditions in a randomly
selected population in Switzerland. Comm Dent Oral Epidemiol. 1988; 16: 181-6.
15. Beck J, Garcia RI, Heiss G, et al. Periodontal disease and cardiovascular disease. J Periodontol. 1996; 16: 170-8.
16. Papapanou PN, Wennström JL. Periodontal status in relation to age and tooth type. Clin Periodontol. 1988; 1: 23-6.
17. Zambon JJ, Reynolds H, Fisher JH, et al. Microbiological and immunological studies of adult periodontitis in patients with noninsulin-dependent diabetes mellitus. J Periodontol. 1988; 59; 23-31.
18. Genco RJ. Current view of risk factors for periodontal disease. J Periodontal. 1996; 67:1041-9.
19. Genco RJ, Grossi SG, Ho A, et al. A proposed model linking inflammation to obesity, diabetes, and periodontal infections. J Periodontol. 2005; 76:2075–84.
20. Bridges RB, Anderson JW, Saxe SR, et al. Periodontal status of diabetic and non-diabetic men: Effects of smoking, glycemic control, and socioeconomic factors. J Periodontol. 1996; 67:1185-92.
21. Saremi A, Nelson RG, Tulloch-Reid M, et al. Periodontal Disease and Mortality in Type 2 Diabetes. Diabetes Care. 2005; 28:27-32.
22. Zhang JQ, Pan YP, Ma L, et al. A survey on the periodontal status in type 2 diabetic patients. Chinese journal of Stomatology. 2009; 44:668-71.
23. Novak MJ, Potter RM, Blodgett J, et al. Periodontal disease in Hispanic Americans with type 2 diabetes. J Periodontol. 2008; 79:629-36.
24. Tsai C, Hayes C, Taylor GW. Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population. Community Dent Oral Epidemiol. 2002;30:182–92.
25. Demmer RT, Jacobs DR, Desvarieux M. Periodontal disease and incident type 2 diabetes mellitus: Results from the first national health and nutrition examination survey and its epidemiologic follow-up study. Diabetes Care. 2008; 31: 1373-9.
26. Jansson H, Lindholm E, Lindh C, et al. Type 2 diabetes and risk for periodontal disease: A role for dental health awareness. J Clin Periodontol. 2006; 33: 408–14.
27. Bandyopadhyay D, Marlow NM, Fernandes JK, et al. Periodontal disease progression and glycaemic control among Gullah African Americans with type-2 diabetes. J Clin Periodontol. 2010; 37:501–9.
28. Tchobroutsky G. Relation of diabetic control to development of microvascular complications. Diabetologia. 1978; 15: 143- 52.
29. Sznajder N, Carraro JJ, Rugna S, et al. Periodontal findings in diabetic and non-diabetic patients. J Periodontol. 1978; 49:445-8.
30. Apoorva SM, Sridhar N, Suchetha A. Prevalence and severity of periodontal disease in type 2 diabetes mellitus (non–insulin-dependent diabetes mellitus) patients in Bangalore city: An epidemiological study. J Indian Soc Periodontol. 2013; 17: 25–9.