

## CORRELATION BETWEEN HBA1C LEVEL AND SEVERITY OF PERIODONTAL DISEASE IN TYPE 2 DIABETIC PATIENTS. A CROSS-SECTIONAL STUDY: 89 MILITARY DENTAL CENTER, CMH KOHAT

<sup>1</sup>Muhammad Roman ,<sup>2</sup>Arooj Fatima, <sup>3</sup>Dr. Farhat Amin, <sup>4</sup>Dr. Ahmad Bin Zaheer , <sup>5</sup>Dr. Hamna Mahmood , <sup>6</sup>Owais Khan, <sup>7</sup>Muhammad Shoaib Khan ,<sup>8</sup>Sheikh Ramish Alam, <sup>9</sup>Zeemal Nazir ,  
<sup>\*10</sup>Atta Ur Rehman

<sup>1</sup>KMU IHS, Kohat

<sup>2</sup>Department of Chemistry, QAU, Islamabad, 45320, Pakistan

<sup>3</sup>Rahbar College of Dentistry, Lahore

<sup>4</sup>Services Institute of Medical Sciences

<sup>5</sup>Shahida Islam Medical and Dental College, Lodhran

<sup>6</sup>Dental Department, Faculty of Allied Health Sciences, Gomal University Dera Ismail Khan, KPK, Pakistan

<sup>7</sup>Dental Department, Faculty of Allied Health Sciences, Gomal University Dera Ismail Khan, KPK, Pakistan

<sup>8</sup>Dental Department, Faculty of Allied Health Sciences, Gomal University Dera Ismail Khan, KPK, Pakistan

<sup>9</sup>Dental Department, Faculty of Allied Health Sciences, Gomal University Dera Ismail Khan, KPK, Pakistan

<sup>\*10</sup>Dental Department, Faculty of Allied Health Sciences, Gomal University Dera Ismail Khan, KPK, Pakistan

<sup>1</sup>[romanktk148@gmail.com](mailto:romanktk148@gmail.com), <sup>2</sup>[afatima@chem.qau.edu.pk](mailto:afatima@chem.qau.edu.pk), <sup>3</sup>[farhatamin008@gmail.com](mailto:farhatamin008@gmail.com), <sup>4</sup>[drahmadbinzaheer@gmail.com](mailto:drahmadbinzaheer@gmail.com),

<sup>5</sup>[hamnamahmood4044@gmail.com](mailto:hamnamahmood4044@gmail.com), <sup>6</sup>[ok714926@gmail.com](mailto:ok714926@gmail.com), <sup>7</sup>[shoibed566@gmail.com](mailto:shoibed566@gmail.com), <sup>8</sup>[pithiisheikh@gmail.com](mailto:pithiisheikh@gmail.com),

<sup>9</sup>[zaimbaloch289@gmail.com](mailto:zaimbaloch289@gmail.com), <sup>\*10</sup>[malakatta11@gmail.com](mailto:malakatta11@gmail.com)

DOI: <https://doi.org/>

### Keywords :

HbA1c, periodontal disease, type 2 diabetes, glycemic control, periodontal parameters

### Article History

Received on 25 Jan, 2026

Accepted on 20 Feb, 2026

Published on 21 Feb, 2026

Copyright @Author

Corresponding Author: \*

Atta Ur Rehman

### Abstract

**Background:** Periodontal disease is a common complication of diabetes, and glycemic control is thought to influence its severity. This study aimed to investigate the correlation between HbA1c levels and the severity of periodontal disease in type 2 diabetic patients. **Methods:** This cross-sectional study was conducted at 89 Military Dental Center, CMH Kohat, involving 100 type 2 diabetic patients. Periodontal parameters (probing depth, clinical attachment loss, and bleeding on probing) were recorded, and HbA1c levels were measured. Patients were categorized into three groups based on HbA1c levels: good control (<7%), fair control (7-8%), and poor control (>8%). **Results:** The mean age of patients was  $52.4 \pm 8.2$  years, with a mean HbA1c level of  $8.2 \pm 1.5\%$ . A significant positive correlation was found between HbA1c levels and periodontal parameters (probing depth:  $r=0.56$ ,  $p<0.001$ ; clinical attachment loss:  $r=0.61$ ,  $p<0.001$ ; bleeding on probing:  $r=0.48$ ,  $p<0.001$ ). Patients with poor glycemic control (HbA1c >8%) had more severe periodontal disease. **Conclusion:** This study suggests a significant correlation between HbA1c levels and the severity of periodontal disease in type 2 diabetic patients. Maintaining good glycemic control may help prevent or reduce the severity of periodontal disease in diabetic patients.

## 1. INTRODUCTION

### 1.1 Overview

Diabetes Mellitus (DM) and periodontitis are two prevalent, complicated, long-term conditions that have a known reciprocal association and affect people's health and quality of life (1). In other words, severe periodontitis is linked to impaired glycaemic control, and DM (especially if there is inadequate glycaemic control) is linked to a rise in the frequency and intensity of periodontitis. Traditional non-surgical periodontal therapy has been linked to benefits in glycaemic control in diabetics; following periodontal therapy, glycated hemoglobin (HbA1c) is reduced by around 0.4% (2).

Despite being thoroughly studied, the relationship between DM and periodontitis is complicated and challenging to fully understand (3). Mealey and Oates (2006) pointed out that evaluating the body of research should be done carefully since the results may be interpreted differently due to limited numbers of study subjects, a substantial diversity of demographics, and varied definitions of the parameters under investigation (4). The pathophysiology of the micro- and macro-vascular consequences of DM, such as retinopathy, nephropathy, and cardiopathy, is said to be comparable to the processes by which DM affects the periodontium (5). DM has a detrimental impact on phagocytosis, chemotaxis, and neutrophil adhesion, which promotes bacterial persistence and accelerates periodontal destruction (6). However, pro-inflammatory cytokines and mediators are formed in greater quantities by hyper-responsive monocytes and macrophages, which also show up in gingival crevices (7). The production of advanced glycosylation end products, which also negatively impact periodontal tissues, is associated with several DM problems (8).

Numerous epidemiological researches have shown that patients with DM have more severe and common periodontal disease than people without the disease (9). Additionally, DM has been proposed as a major risk factor for periodontitis (10). Although, it is still unclear how precisely DM influences the emergence of periodontal disease, it has been demonstrated that the severity of both conditions DM and periodontitis depends on a number of risk factors, including the patient's age, social behaviour, degree of oral hygiene, duration and metabolic control of DM, and certain aggravating factors, such as smoking (3, 9, 11).

### 1.2 Diabetes Mellitus (DM)

DM is a chronic metabolic condition marked by persistent hyperglycemia brought on by decreased insulin production, insulin resistance, or both (12). Systemic issues, including increased atherosclerosis, microvascular damage, and end-organ malfunction, result from this imbalance, which interferes with the metabolism of carbohydrates, proteins, and lipids (13). With 81% of cases occurring in low- and middle-income countries and a projected 537 million cases (10.5% of adults) reported in 2021, the prevalence of DM has reached epidemic proportions worldwide (14, 15). This increase has been caused by sedentary lifestyles, obesogenic environments, and rapid urbanization, especially in regions experiencing dietary and epidemiological changes (16). Early mortality and morbidity are increased by long-term micro and macrovascular problems such as nephropathy, retinopathy, neuropathy, chronic hyperglycemia, and cardiovascular diseases (3, 11). Particularly alarming is the increasing incidence of early-onset diabetes mellitus (EOD; diagnosis  $\leq 35$  years), which poses particular clinical and public health issues (17). Younger patients were more likely to experience micro- and macrovascular consequences, have greater lifetime healthcare needs, and develop diseases more quickly (18, 19). The etiology of EOD is complicated and includes developmental origins (e.g., intrauterine metabolic programming, maternal DM), genetic predisposition (e.g., epigenetic modifications, familial aggregation), and modifiable lifestyle factors (e.g., excessive consumption of sugar-sweetened beverages, physical inactivity) (20-23).

Traditionally, DM manifests as a trio of symptoms: polyphagia, polyuria, and polydipsia. Hyperglycemia and the ensuing osmotic imbalance are directly responsible for these symptoms. There are two potential explanations for the problems. The first is the polyol route, in which the enzyme aldol reductase converts glucose to sorbitol. Sorbitol has been linked to the majority of diabetic problems and is regarded as a tissue toxin (24). The second process is the non-enzymatic addition of hexoses to proteins, which causes advanced glycation end products (AGEs) to develop. The functions of several bodily proteins, including haemoglobin, lipoproteins, collagen, lens proteins, and plasma albumin, are altered (25). Therefore, the goal of managing DM is to keep blood sugar levels within normal values, and there is ample evidence that careful management of hyperglycemia helps avoid problems (26).

The effectiveness of this control is monitored by measuring the levels of glycosylated serum proteins, particularly HbA1c, which provides an indication of the serum glucose levels over the previous two to three months due to its combination with the red blood cells. Different types of DM are recognized by the categorization system (27). Type 1 Diabetes Mellitus or T1DM (formerly known as insulin-dependent DM, or IDDM) and Type 2 Diabetes Mellitus or T2DM (formerly known as non-insulin-dependent DM, or NIDDM) are the two main forms of the illness. The pathophysiology of T1DM, an autoimmune condition that leads to the death of pancreatic  $\beta$  cells and consequent insulin production loss, and T2DM, which is related to insulin resistance, are different (3, 28). This new categorization was created to emphasize that the pathology involved determines the type of DM rather than whether insulin is necessary for the disease's management, as both categories may need insulin treatment at some point. Patients with T1DM have a propensity to develop ketoacidosis if insulin is not given. The loss of the islet cells that make insulin is the cause of this illness. Insulin secretory defects combined with target cell resistance to insulin cause T2DM. Elevated venous blood glucose levels are the basis for the recently updated (27) diagnostic criteria for DM. The following tests are all regarded as positive:

1. Blood sugar levels  $\geq 200$  mg/dl were assessed at random in individuals exhibiting symptoms including polydipsia, polyuria, or weight loss.
2. People with fasting venous blood sugar levels are  $\geq 126$  mg/dl.
3. Venous blood sugar levels  $\geq 200$  mg/dl, two hours following a 75 g glucose load, and at least once more throughout the test (29).

#### 1.2.1 Type 2 Diabetes Mellitus (T2DM)

Dysregulation of the metabolism of fats, proteins, and carbohydrates is a characteristic of T2DM, which may be caused by reduced insulin production, insulin resistance, or both. Compared to T1DM and gestational DM, T2DM is the most prevalent of the three main forms of the disease, making up more than 90% of all instances. Its main cause is a progressive reduction in the amount of insulin produced by pancreatic  $\beta$  -cells, frequently in conjunction with pre-existing insulin resistance in skeletal muscle, adipose tissue, and the liver (30, 31). Prediabetes (30, 32), a high-risk condition for T2DM, is usually followed by overt hyperglycemia. Impaired fasting glucose (IFG), impaired glucose tolerance

(IGT), or high HbA1c are indicators of prediabetes. While IGT is characterized by insulin resistance and decreased insulin production after meals, IFG denotes higher fasting plasma glucose (FPG) levels (32). HbA1c values in pre-diabetic people range from 5.7% to 6.4%, indicating a considerable variation in pathogenesis. Between 3% and 11% of people with prediabetes develop T2DM each year (33).

With 382 million persons afflicted in 2013, mostly in low- and middle-income nations, T2DM is a serious worldwide concern for health (34). This figure is expected to increase to 592 million by 2035 (34). This epidemic is caused by a number of elements, including Asians' greater body fat percentage, abdominal obesity, inadequate early nutrition, and subsequent over nutrition (35). Men are somewhat more likely than women to experience it (34).

#### 1.2.2 T2DM and Glycemic Control

Nearly half of T2DM patients have HbA1c levels higher than the usually advised target of less than 7% (36, 37). However, many individuals whose HbA1c is below the current threshold may experience microvascular problems. Novel disease indicators and therapies to assist in identifying and managing patients before they are diagnosed with T2DMs may be developed as a result of new knowledge of the biochemical pathology underlying microvascular problems (38).

For those with T2DM, macrovascular problems may be the leading cause of morbidity. The late introduction of rigorous glycemic therapy may be harmful to cardiovascular results, according to recent large trials like Veterans Affairs Diabetes Trial (VADT) (39) and Action to Control Cardiovascular Risk in Diabetes (ACCORD) (40). Early in the course of the illness, when stringent glycemic control seems to be most beneficial, underlying atherosclerosis develops. Strong data suggest that the existing treatment guidelines, which call for intervention at a conventional HbA1c target, do not benefit all T2DM patients equally. In order to treat the subset of diabetic patients who are most vulnerable to microvascular and macrovascular problems more vigorously, better markers for faster disease progression and complications are required (38).

#### 1.3 Periodontal Disease

Bacterial inflammatory illnesses that impact the periodontal attachment apparatus are collectively referred to as periodontitis (29). Clinical attachment loss (CAL), gingival bleeding, the development of

periodontal pockets, and gingival recessions that result in the loss of alveolar bone are all signs of periodontitis (1). Advanced stages of periodontitis typically affect a tiny percentage of the population, even though mild forms like gingivitis or initial loss of periodontal attachment are quite frequent in all age cohorts (1, 41). Chronic gingival inflammation does not seem to be directly related to the development of periodontitis, and there is insufficient data on predictive markers of chronic periodontitis in individuals with rapidly developing periodontal symptoms (3). The traditional indicators of inflammation associated with periodontitis are swelling, redness, elevated local intrasulcular temperature, and tenderness (pain) (29).

Six categories have been found in a newly updated taxonomy of periodontal disorders (29, 42).

- I. Gingival diseases.
- II. Necrotizing periodontal diseases.
- III. Aggressive periodontitis.
- IV. Abscesses of the periodontium
- V. Chronic periodontitis.
- VI. Periodontitis as an appearance of systemic diseases (29, 42).

Gingivitis is a common inflammatory illness affecting the gingival tissues, impacting more than half of adults in the U.S., particularly characterized by gingival bleeding (43). While many individuals may have an inflammatory infiltrate without obvious signs, gingivitis can lead to periodontal disease, characterized by the destruction of the dental attachment apparatus. Although probing depth (PD) and CAL are used to diagnose periodontitis, they reflect past damage rather than current attachment loss. The prevalence of moderate to chronic periodontal disease is estimated to be around 14% (44), despite previous research suggesting that the condition may be underestimated in current surveys (29, 45-47).

With a combined incidence of over 62% among dentate individuals, periodontitis remains to be a global public health concern. According to research conducted between 2011 and 2020, the anticipated cumulative prevalence for severe periodontitis is 23.6%, while it is 53.2% for moderate-to-severe instances (48). Estimates from 1990 to 2010 showed that 10.8% of people had severe periodontitis (49). According to a different research, the prevalence has significantly grown over the past three decades (up to 2019), currently impacting 1.1 billion people worldwide (50). Cardiovascular disease, which has a 6.6% global prevalence and is the primary cause of

morbidity and mortality, is less frequent than periodontal disease (51, 52).

#### 1.4 Bidirectional Relationship Between Diabetes and Periodontal Disease

It has been suggested that periodontitis and DM are related in both directions (53). Researchers from around the world have recently shown interest in the connection between DM and chronic periodontitis. Numerous epidemiological studies over the years have shown that adults with type 2 diabetes have a higher prevalence, incidence, and severity of periodontitis (4). In T2DM patients with progressive systemic issues, periodontitis is more prevalent and chronic, and its frequency increases with age (10).

Patients with diabetes mellitus frequently suffer from severe alveolar bone loss as a result of periodontitis, which is brought on by a weakened immune system and slowed tissue repair. The invasion of anaerobic bacteria into periodontal tissues, including the gingival connective tissue, the periodontal ligament, and the alveolar bone, is known as periodontal disease. Periodontal pathogens like *P. gingivalis* lipopolysaccharide (P-LPS) and several cytokines, such as tumour necrosis factor-alpha (TNF-alpha), interleukin-1 (IL-1), and interleukin-6 (IL-6), stimulate osteoclast differentiation in gingival connective tissue. After that, alveolar bone resorption progresses, resulting in tooth loss and a reduction in oral function. It has been shown that the incidence of periodontitis is two to three times higher in individuals with diabetes mellitus than in those without the condition. Recent research has demonstrated that periodontitis affects diabetes mellitus, and that periodontal pathogens such as P-LPS and TNF-alpha may exacerbate insulin resistance by inhibiting smooth muscle cells' ability to incorporate glucose. According to a clinical trial, people with periodontitis had higher blood C-reactive protein (CRP) levels even though periodontal therapy improved the HbA1c levels of diabetic patients. These results imply that periodontal pathogens affect systemic illnesses and that periodontal therapy can somewhat mitigate these conditions. Additionally, periodontal pathogens may encourage the development of atherosclerosis (54).

Increased inflammation is often associated with problems from DM. TNF-alpha, IL-6 (55), and an enhanced Th1/Th2 cell ratio are systemic manifestations of T2DM that are linked to microvascular problems (56). Rats with T2DM have elevated nuclear factor-kappa B and upregulated

TNF-alpha in their large blood arteries, both of which lead to macrovascular problems (57). The beginning and development of diabetic nephropathy are related with higher levels of inflammatory cytokines, including IL-1, IL-6, IL-18, and TNF-alpha (58-60). Early-stage DM is brought on by inflammation-induced endothelial cell and pericyte loss, which causes hypoxia and subsequently promotes angiogenesis (61). Therefore, elevated inflammation is the driving force behind the pathology of a number of DM problems (62).

Periodontitis and alveolar bone loss are more common in those with poorly managed DM, who are also most vulnerable to the other microvascular and macrovascular problems (29, 63). Therefore, enhancing glycemic management can slow the pathological evolution of periodontal disease (64, 65), and T2DM is thought to be among the periodontitis risk factors (66, 67). Insulin resistance may be lessened by periodontal treatment that lowers inflammation. Additionally, long-term periodontal diseases may worsen insulin resistance and impair glycemic management (68, 69). Furthermore, there is evidence that periodontal inflammation resolution can improve metabolic regulation (with reported HbA1c reductions of around 0.4%); however, these results need to be confirmed by large, multi-center, randomized controlled studies (67).

It is unclear exactly how glycemic control affects periodontitis and how treating it may impact glycemic control in individuals with T2DM. A widely accepted theory links the pathophysiology of insulin resistance and periodontitis to inflammatory pathways (67, 70). A model describing how severe periodontal disease aggravates DM and complicates metabolic control is provided. The advanced glycation end product (AGE)-mediated cytokine response that plays a role in DM may be strengthened by an infection-mediated loop of increased cytokine synthesis and release by prolonged stimulation from LPS and products of periodontopathic organisms. According to this view, the increased tissue death observed in diabetic periodontitis may be explained by the interaction of these two pathways: infection and AGE-mediated cytokine overexpression. There is a two-way interaction between DM and periodontal disease/infection, since it shows how periodontal infection may exacerbate the severity of DM and the degree of metabolic control. According to this suggested twofold route of tissue damage, managing

chronic periodontal infection is crucial to managing DM over the long term (68). To completely comprehend the connection between DM and periodontitis, more research is required (54).

Hence, unquestionably, DM is an important risk factor for periodontitis disease (9, 71, 72). In contrast to those without DM, those with DM had a roughly twofold higher chance of developing periodontitis (5). An important factor in identifying elevated risk is the degree of glucose control. For instance, after adjusting for age, sex, education, ethnicity, and smoking, adults with an HbA1c level of >9% in the US National Health and Nutrition Examination Survey (NHANES) III had a significantly higher prevalence of severe periodontitis than those without DM (OR 2.90; 95% CI 1.40, 6.03) (67, 73).

### **1.5 Problem Statement**

According to available studies, T2DM patients in Pakistan currently lack a comparable thorough evaluation of attachment loss and PD in each kind of tooth. While earlier research in other populations has shown that glycemic control significantly affects the severity of periodontal disease in diabetics, these studies have not considered confounding variables like poor oral hygiene and smoking when assessing the influence of poorly controlled DM on periodontium. The majority of studies have examined the connection between periodontitis and DM of a certain type (either T1DM or T2DM), but they have not examined potential variations in the development of periodontitis in relation to the various physiopathologies of DM, especially T2DM. Hence, to assess the degree of periodontal disease in T2DM patients, the current study sought to overcome this deficit.

### **1.6 Objective**

The goal of the study was to evaluate the association between HbA1c levels and the severity of periodontitis in adult patients with T2DM who visited the 89 Military Dental Centre CMH Kohat. Also, to compare periodontal parameters across categories of glycemic control (HbA1c: good, moderate, poor) and to assess the influence of demographic aspects (sex, age, BMI) and lifestyle factors (smoking, oral hygiene practices) on periodontal disease severity in diabetic patients.

### **1.7 Significance of the Study**

With almost 6.2 million people between the ages of 20 and 79 suffering from DM, Pakistan is ranked sixth in the world. A moderate form of periodontitis was reported in 6-10% of Pakistani DM patients

between the ages of 35 and 44. Given the high prevalence of type 2 diabetes in Pakistan and the frequent occurrence of periodontitis, it is imperative to comprehend the relationship between glycemic control and periodontal severity. This knowledge can help diabetic patients receive targeted prevention, prompt interventions, and integrative care strategies. This is especially relevant in regions with limited healthcare resources, where early identification of high-risk patients may improve oral and systemic health outcomes.

## 2. METHODOLOGY

### 2.1 Study Design

This research was designed as a cross-sectional analytical observational study aimed at investigating the correlation between HbA1c levels and periodontal disease severity in T2DM. 250 patients between the ages of 30 and 70 who were registered at the 89 Military Dental Center, CMH Kohat, participated in the cross-sectional study. The research was carried out from October 2025 to January 2026.

### 2.2 Study Setting

The research was conducted at the 89 Military Dental Centre, CMH Kohat. Clinical periodontal assessments were performed in the dental department under standardized conditions, while HbA1c measurements were obtained from the hospital's accredited laboratory.

The study duration is 5 months, from 2 October 2025 to 31 January 2026.

### 2.3 Study Population

Adult T2DM patients using outpatient care are part of the target population.

#### 2.3.1 Inclusion Criteria

- Adults aged 30-70 years
- Diagnosed with T2DM for at least 1 year
- Presence of  $\geq 12$  natural teeth
- Willingness to provide written informed consent

#### 2.3.2 Exclusion Criteria

- Type 1 diabetic patients
- Pregnant or lactating women
- History of systemic conditions affecting periodontal health (e.g., immunodeficiency, malignancy)
- Periodontal treatment within the last 5 months
- Antibiotic or anti-inflammatory therapy within the last 3 months
- Severe cardiovascular, renal, or hepatic disease.

### 2.4 Sample Size Calculation

Calculated sample size:  $n = 250$  patients

### 2.5 Ethical Considerations:

The Gomal University, DI Khan Faculty of Allied Health Science (Dental Department) standard research committee gave its approval to the project. Additionally, the Command of Dental Block and the HOD of Dental OPD both provided formal consent. All procedure was performed according to rules and regulations.

### 2.6 Data Collection Procedure

#### 2.6.1 Clinical Periodontal Examination

A single calibrated periodontist performed examinations using a UNC-15 periodontal probe.

Parameters measured:

1. Probing Depth (PD) - six sites per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, distolingual)
2. Clinical Attachment Loss (CAL) - measured from CEJ to the pocket base
3. Bleeding on Probing (BOP) - recorded as 0 (absent) or 1 (present) per site
4. Plaque Index (PI) - assessed using the Silness-Löe plaque scoring method

Calibration procedure:

- Examiner measured 10 patients twice, 48 hours apart
- Intra-class correlation coefficient (ICC) calculated for PD and CAL
- ICC  $\geq 0.85$  considered acceptable

#### 2.6.2 Glycemic Assessment

- HbA1c (%) measured using High-Performance Liquid Chromatography (HPLC)
- Classification:
  - Good control:  $< 7\%$
  - Moderate control: 7-8.9%
  - Poor control:  $\geq 9\%$

#### 2.6.3 Demographic and Lifestyle Data

- Sex, age, body mass index (BMI), duration of DM, smoking habits, and oral hygiene practices
- Collected *via* structured interviewer-administered questionnaire

### 2.7 Data Management and Quality Control

- Data entered in Microsoft Excel, double-checked by a second researcher
- Outliers checked using box plots
- Missing data handled using pairwise deletion
- All patients received standard dental care after examination

### 2.8 Statistical Analysis

Software: SPSS version 26.0

#### 1. Descriptive Statistics

- Mean  $\pm$  SD for continuous variables

- Frequency and percentage for categorical variables
2. Normality Testing
    - Shapiro-Wilk test for PD, CAL, HbA1c
  3. Correlation Analysis
    - Pearson correlation for normally distributed variables
    - Spearman correlation for non-normal variables
  4. Multivariate Regression
    - Main outcome: Correlation between HbA1c and PD, CAL, BOP, PI
    - Linear regression with PD and CAL as dependent variables
    - Independent variables: HbA1c, age, duration of DM

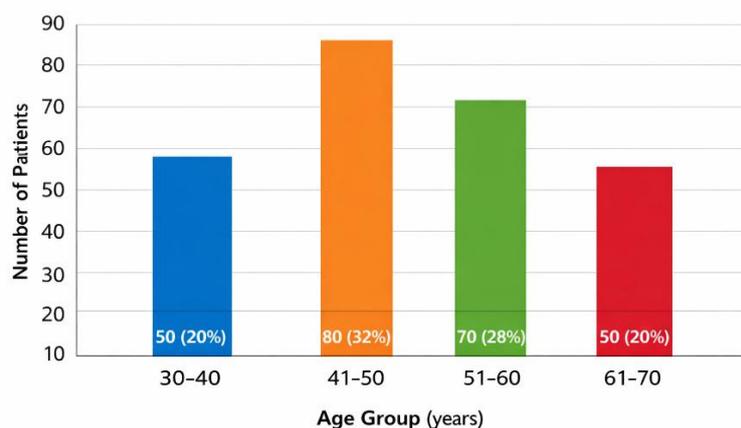
### 3. DATA ANALYSIS AND RESULTS

#### 3.1 Clinical Examination and Data Collection

The operator used a mouth mirror, disposable gloves, and a blunt-ended periodontal probe during the clinic examination of an adult under headlight illumination. Following the clinic examination, information was gathered through questionnaires

**Table 3.1:** *Age Distribution*

Variable (Age)	Frequency	Percentage (%)	Mean	Standard deviation
30-40	50	20%	49.8	10.4
41-50	80	32%		
51-60	70	28%		
61-70	50	20%		
Total	250	100%		



**Figure 3.1.** *Age Distribution of Study Population*

#### Gender

In the overall patients, the number of males was n=142(56.8%), and females were n=108(43.2%)

**Table 3.2:** *Gender Distribution*

Sex	Frequency	Percentage (%)
Male	142	56.8
Female	108	43.2

and conversations with guardians regarding demographics, dental hygiene, eating habits, and dietary practices.

#### 3.1.1 Statistical Analysis

To determine the correlation between HbA1c level and the severity of periodontal disease in T2DM patients, descriptive statistics were used. To ascertain whether the change was substantial in the severity of periodontal disease in T2DM diabetics among the various exposure variables, the chi-square test was used.

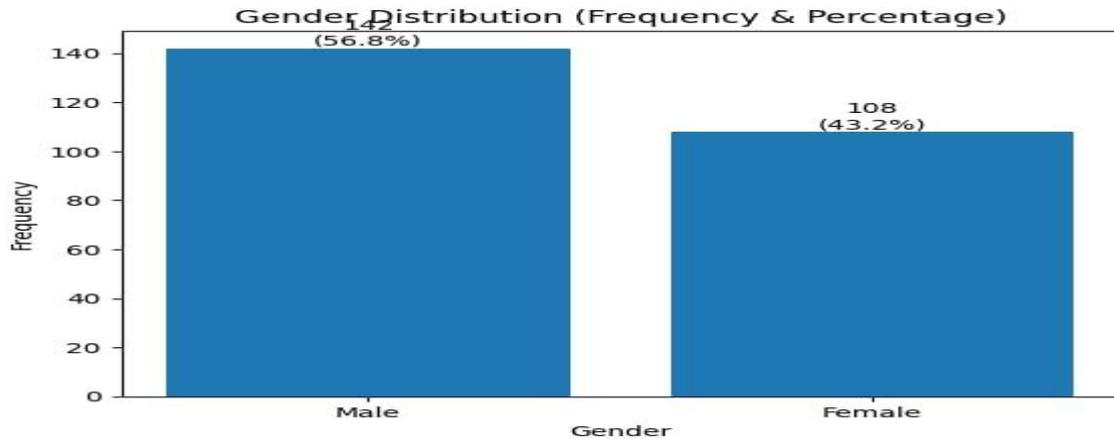
#### 3.1.2 Results

A total of 250 patients were included, with a mean age recorded of 49.8 ± 10.4, with a maximum age limit of 70 years and a minimum of 30 years; all patients were diabetic.

Severity of periodontitis among the age group 30-40 years, n=50 (20%), the second group, age 41-50 years, was n=80 (32%), the third group, age 51-60 years, n=70 (28%), and the fourth group, age 61-70 years, n=50 (20%). Aged 41-50 had a significantly greater severity of periodontal disease in T2DM (32%).

severity of periodontitis in T2DM was more common in males compared to females.

Total	250	100%
-------	-----	------



**Figure 3.2. Gender Distribution (Frequency and Percentage)**

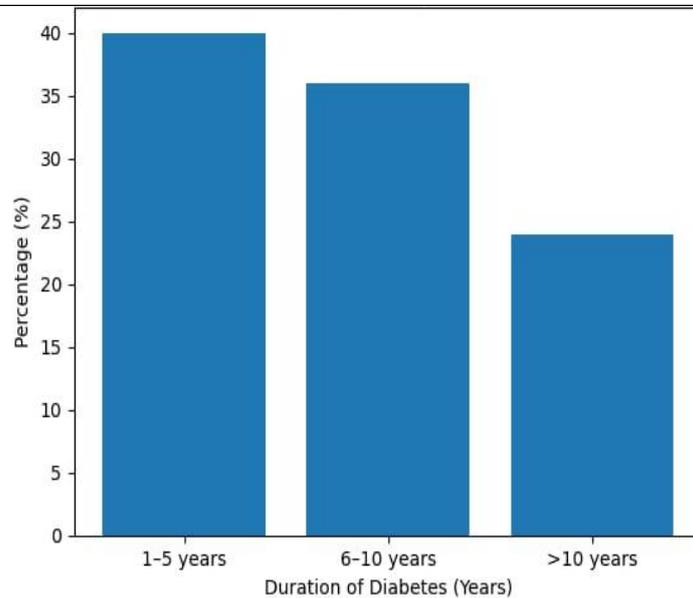
**Durations of Diabetes years**

The durations of DM among the patients revealed that n=100 (40%) had DM for 1-5 years, and approximately n=90 (36%) of the patients had

durations of DM ranging from 6-10 years. Long-standing DM of more than 10 years was observed in n=60 (24%) of the study populations. Overall of the patients 76% had DM for 10 or less.

**Table 3.3: Durations of Diabetes (years) Distribution**

Durations DM (years)	Frequency	Percentage (%)
1-5	100	40%
6-10	90	36%
>10	60	24%



**Figure 3.3. Distribution of Participants by Duration of Diabetes**

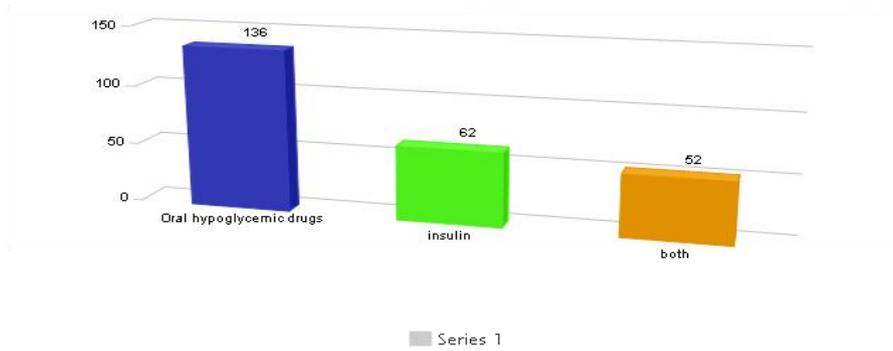
**Diabetic Treatment**

The majority of the patients, n=136(54.4%), were using oral hypoglycemic drugs. Insulin therapy was reported by n=62(24.8%) patients, while

n=52(20.8%) patients were managing DM through diet control alone. This was the most commonly used treatment modality among the patients.

**Table 3.4: Diabetic Treatment**

Variable	Frequency	Percentage (%)
Oral hypoglycemic drugs	136	54.4
Insulin	62	24.8
Both	52	20.8



**Figure 3.4. Diabetic Treatment**

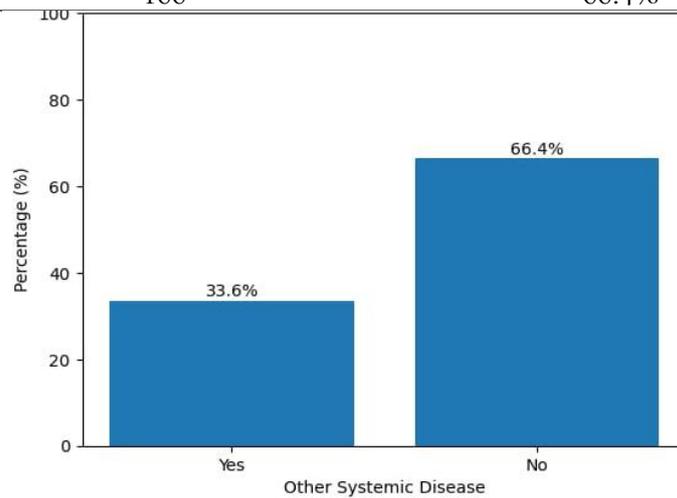
**Other Systemic Disease**

The distribution of other systemic diseases among the study patients. Out of the sample, n=84(33.6%) patients reported the presence of other systemic

diseases, whereas n=166(66.4%) patients had no history of any other systemic disease. The majority of the study population did not suffer from additional systemic conditions.

**Table 3.5: Other Systemic Disease**

Variable	Frequency	Percentage (%)
Yes	84	33.6%
No	166	66.4%



**Figure 3.5. Percentage Distribution of Other Systemic Diseases**

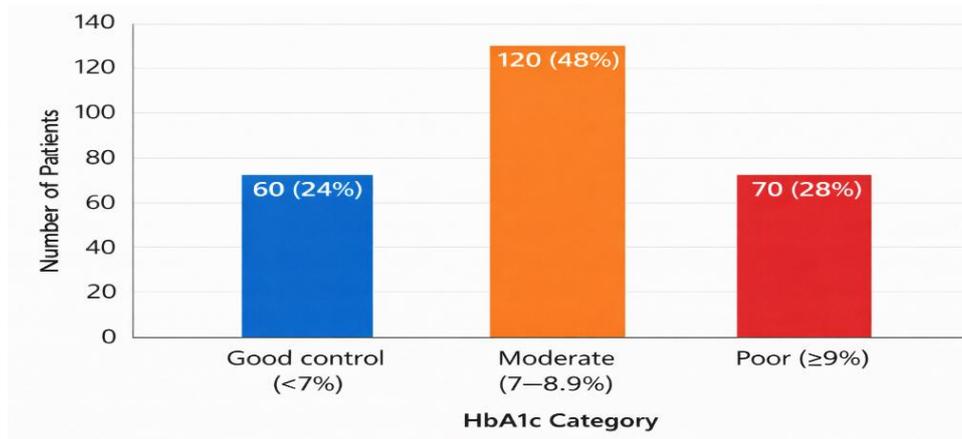
**HbA1c Category**

The HbA1c category distribution showed that n=60 (24%) of the patients had good glycemic control (HbA1c,7%), moderate glycemic control (HbA1c 7-

8.95%) was observed in n=120 (48%) of the patients, and poor glycemic control was observed in n=70 (28%) of the study population. Overall of the patients had 76% moderate to poor glycemic control.

**Table 3.6: HbA1c Category Distribution**

HbA1c category	Frequency	Percentage (%)
Good control (<7%)	60	24%
Moderate (7-8.9%)	120	48%
Poor (>9%)	70	28%



**Figure 3.6. HbA1c Category Distribution**

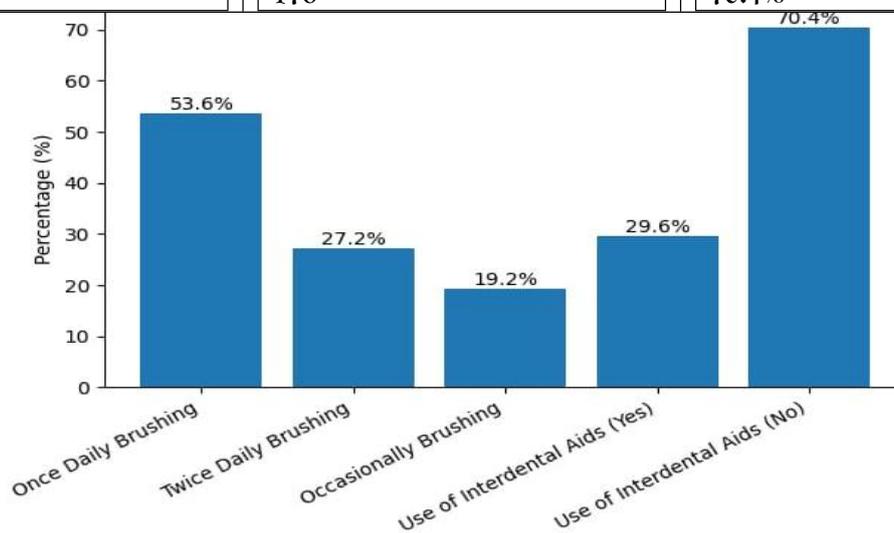
**Oral Hygiene Practices**

Oral hygiene practices of the patients are shown in **Figure 3.8.**, more than half of the patients brushed their teeth once daily, n=134 (53.6%), while n=68 (27.2%) brushed twice daily. A smaller proportion

reported brushing occasionally, n=48 (19.2%). Regarding the use of interdental aids, only n=74 (29.6%) patients reported using them, whereas the majority, n=176 (70.4%), did not use interdental aids.

**Table 3.7: Use Tooth brushing and interdental Aids**

Variable	Frequency	Percentage (%)
<b>Tooth Brushing</b>		
Once Daily	134	53.6%
Twice Daily	68	27.2%
Occasionally	48	19.2%
<b>Use of interdental Aids</b>		
Yes	74	29.6%
No	176	70.4%



**Figure 3.7. Oral Hygiene Practices (Percentage Distribution)**

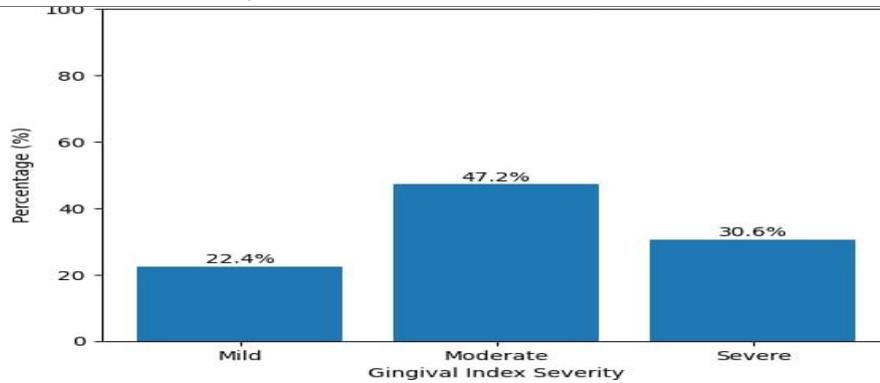
**Gingival Index (GI)**

The distribution of gingival inflammation severity among the study patients based on the GI. The results show that moderate gingivitis was the most prevalent condition, affecting 118 patients (47.2%). This was followed by severe gingivitis, observed in 76

participants (30.6%), while mild gingivitis was present in 56 patients (22.4%). Overall, the results show that a significant percentage of the study population had moderate to severe gingival inflammation, which is indicative of the patients' poor periodontal health.

**Table 3.8:** *Gingival Index (GI)*

Severity	Frequency(n)	Percentage (%)
Mild	56	22.4%
Moderate	118	47.2%
Severe	76	30.6%



**Figure 3.8 Percentage Distribution of Gingival Index (GI)**

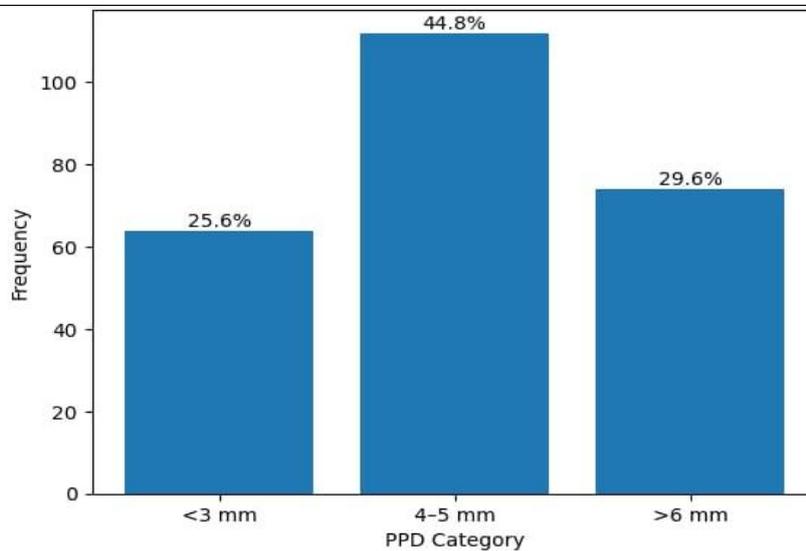
**Probing pocket Depth (PPD)**

*Table 3.8* shows how PPD was distributed among the study participants. The results show that 112 patients (44.8%) had PPD of 4-5 mm. PPD larger than 6 mm, which was found in 74 patients (29.6%),

indicated advanced periodontal involvement. However, 64 participants (25.6%) had PPD of less than 3 mm. A high prevalence of periodontitis was indicated by the percentage of the study population that had moderate to deep periodontal pockets.

**Table 3.9:** *Robing pocket Depth (PPD)*

PPD category	Frequency(n)	Percentage (%)
<3mm	64	25.6%
4-5mm	112	44.8%
>6mm	74	29.6%



**Figure 3.9. Distribution of Probing Pocket Depth (PPD)**

**Clinical Attachment Loss (CAL)**

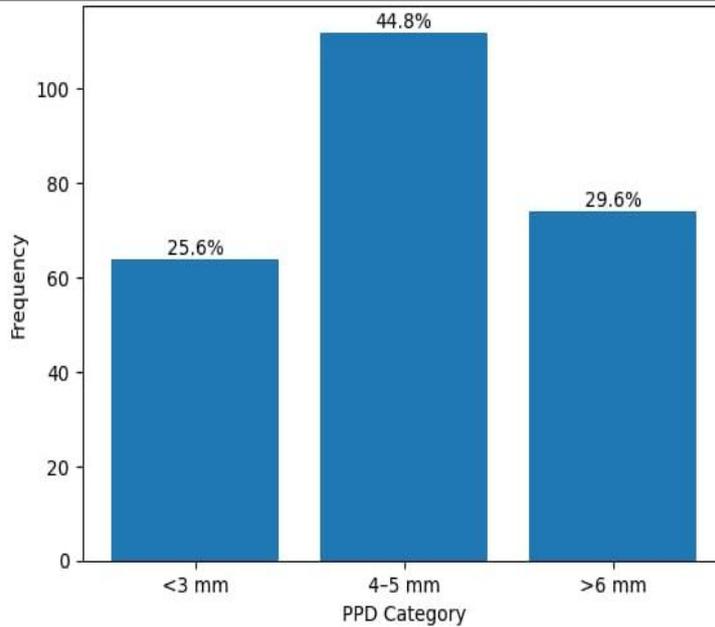
The research patients' CAL severity is displayed in *Table 3.9*. The majority of patients (106, or 42.4%) had moderate attachment loss. 72 patients (28.8%) had mild attachment loss, and 72 patients (28.8%)

had severe attachment loss. These results show that many patients had a discernible loss of periodontal attachment, which is indicative of continuous periodontal tissue degradation.

**Table 3.10:** *Clinical Attachment Loss (CAL)*

Severity	Frequency(n)	Percentage (%)
Mild	72	28.8%

Moderate	106	42.4%
Severe	72	28.8%



**Figure 3.10. Distribution of Probing Pocket Depth (PPD)**

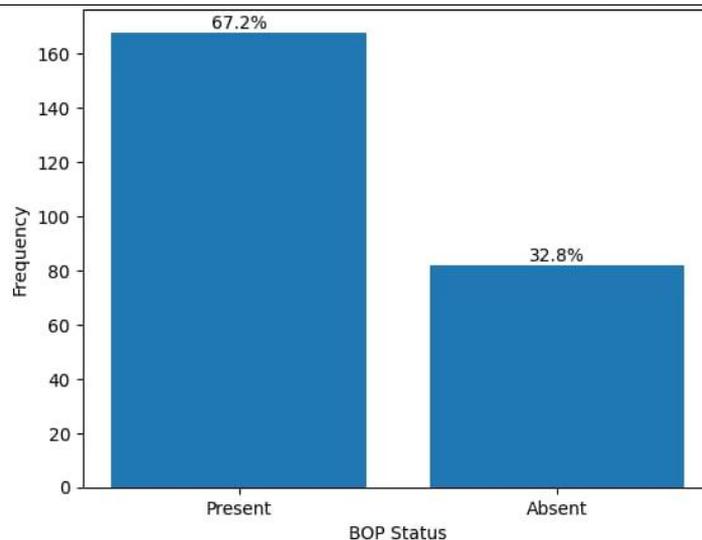
**Bleeding on Probing (BOP)**

*Table 3.10* displays the BOP prevalence among the study participants. BOP was present in most of the patients, observed in 168 (67.2%) patients. In contrast, 82 (32.8%) patients showed no bleeding on

probing. These results indicate that gingival inflammation was common in the study population, suggesting poor periodontal health in a large number of patients.

**Table 3.11: Bleeding on probing (BOP)**

Status	Frequency(n)	Percentage (%)
Present	168	67.2%
Absent	82	32.8%



**Figure 3.11. Distribution of Bleeding on Probing (BOP)**

**Overall Periodontal Disease Severity**

*Table 3.11* lists the study participants' overall periodontal disease severity. Of the participants, 108 (43.2%) had moderate periodontal disease, which was the most prevalent. Of the participants, 78

(31.2%) had severe periodontal disease, and 64 (25.8%) had mild disease. The majority of patients had moderate to severe periodontal disease, according to these results, suggesting that the study

population had a high prevalence of periodontal issues.

**Table 3.12:** *Overall Periodontal Disease Severity*

Severity	Frequency(n)	Percentage (%)
Mild	64	25.8%
Moderate	108	43.2%
Severe	78	31.2%

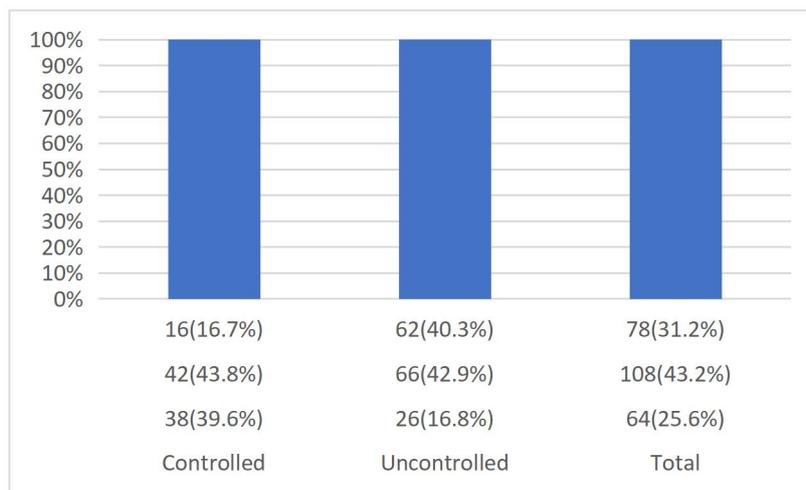
**Table 3.12** shows the association between the study participants' HbA1c status and the degree of periodontitis. The participants were divided into two groups according to their glycemic control, controlled, and uncontrolled HbA1c levels. The distribution of periodontal disease severity within these two groups was then investigated. Of the 96 patients with controlled HbA1c levels, 42 patients (43.8%) had moderate periodontal disease and 38 patients (39.6%) had mild periodontal disease. In this group, only 16 patients (16.7%) had severe periodontal disease. These results imply that mild to moderate periodontal involvement was more common in people with good glycemic control, while a comparatively smaller percentage had severe disease. Conversely, the severity of periodontitis was significantly higher in individuals with uncontrolled HbA1c levels (n = 156). While 66 patients (42.9%) had moderate periodontal disease, only 26 patients (16.8%) had mild periodontal disease. In this group, 62 patients (40.3%) had severe periodontal disease. This implies that poor glycemic control was

associated with a higher frequency of advanced periodontal destruction. Patients with uncontrolled HbA1c levels had much higher rates of severe periodontal disease than those with controlled HbA1c, as can be seen by comparing the two groups. On the other hand, mild periodontal disease was more common in patients with controlled glycemic status. Although periodontal disease was moderate in both groups, there was a noticeable shift toward severe disease in the uncontrolled group.

Of the 250 participants, 64 (25.6%) had mild periodontal disease, 108 (43.2%) had moderate disease, and 78 (31.2%) had severe periodontal disease. The distribution clearly shows that more severe periodontal disease is associated with worsening glycemic control. These findings support the hypothesis that poor glycemic control in people with type 2 diabetes may make periodontal disease worse. The results highlight the importance of maintaining optimal HbA1c levels to reduce the risk of severe periodontal involvement, as well as the close relationship between DM control and periodontal health.

**Table 3.13:** *Association of HbA1c status with periodontal Disease severity*

HbA1c state	Mild n(%)	Moderate n(%)	Severe n(%)	Total n(%)
Controlled	38(39.6%)	42(43.8%)	16(16.7%)	96
Uncontrolled	26(16.8%)	66(42.9%)	62(40.3%)	156
<b>Total</b>	<b>64(25.6%)</b>	<b>108(43.2%)</b>	<b>78(31.2%)</b>	<b>250</b>



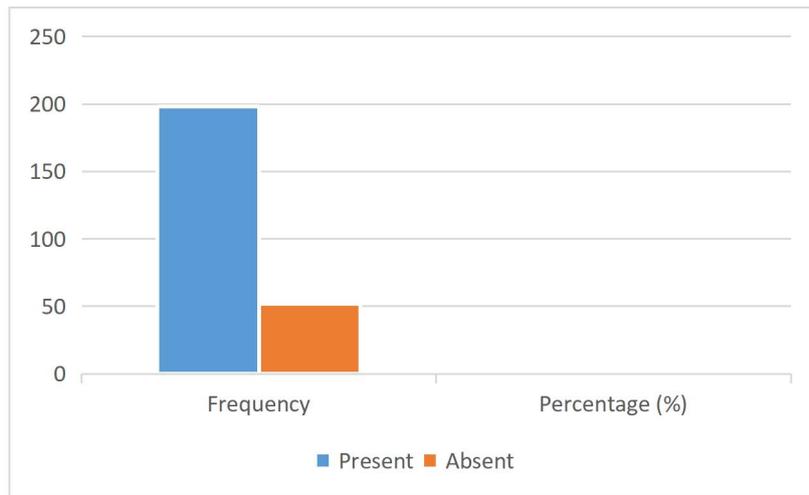
**Figure 3.12.** *Association of HbA1c status with periodontal Disease severity*

**Table 3.14** displays the prevalence of periodontal disease among study participants. 198 participants (79.2%) had periodontal disease, meaning that most of the study participants had the condition. Merely 52 individuals (20.8%) did not exhibit any

indications of periodontal disease. These results imply that the study group had a high prevalence of periodontitis, indicating a significant burden of periodontal issues among the participants.

**Table 3.14:** *Presence of periodontal disease*

Periodontal disease	Frequency	Percentage (%)
Present	198	79.2%
Absent	52	20.8%



**Figure 3.13.** Association of uncontrolled HbA1c with severe periodontal disease

A relationship exists between the presence of severe periodontitis and the HbA1c status of the research participants. The distribution of severe and non-severe forms of periodontal disease was compared between the controlled and uncontrolled HbA1c groups. Eighty patients (83.3%) had non-severe periodontal disease, whereas sixteen patients (16.7%) with controlled HbA1c levels had severe periodontal disease. This result implies that advanced periodontal destruction was less common in patients with good glycemic control. Participants with uncontrolled HbA1c levels, on the other hand, displayed a noticeably different pattern. Sixty-two patients (40.3%) had severe periodontitis, whereas ninety-two patients (59.7%) had non-severe periodontitis. Therefore, compared to the controlled group, the uncontrolled HbA1c group had a much higher percentage of severe periodontitis. When comparing the two groups, it was evident that uncontrolled HbA1c was associated with a higher incidence of severe periodontitis. Patients with poor glycemic control were more than twice as likely to have severe periodontal involvement as those with

controlled HbA1c levels. This illustrates how long-term hyperglycemia damages periodontal tissues. Overall, these results show a strong correlation between the severity of periodontitis and inadequate glycemic control. Uncontrolled HbA1c appears to play a role in the development of periodontal disease, potentially due to immune response compromise and delayed tissue healing associated with diabetes. The findings show how crucial it is for people with type 2 diabetes to maintain appropriate glycemic control in order to reduce their risk of developing severe periodontal disease.

#### **Association of uncontrolled HbA1c with severe periodontal disease**

**Table 3.15** shows the association between severe periodontitis and HbA1c status. Of the 16 patients (16.7%) with controlled HbA1c levels, 16 had severe periodontitis, whereas 80 (83.3%) had non-severe disease. On the other hand, 62 (40.3%) of the patients with uncontrolled HbA1c had severe periodontal disease, while 92 (59.7%) had non-severe disease. These results indicate that a higher risk of severe periodontitis is linked to uncontrolled HbA1c.

**Table 3.15:** *Association of uncontrolled HbA1c with severe periodontal disease*

HbA1c state	Severe PD n(%)	Non -severe PD n(%)
Controlled	16(16.7%)	80(83.3%)
Uncontrolled	62(40.3%)	92(59.7%)

**Chi-square Test Analysis**

A statistical technique for analyzing the association between two categorical variables is the Chi-square ( $\chi^2$ ) test. It was used in this study to ascertain whether the severity of periodontal disease (mild, moderate, or severe) is significantly correlated with HbA1c status (controlled vs. uncontrolled). The test compares each category's observed frequencies to what would be predicted if there were no association between the variables. The distribution of periodontitis severity varies between the controlled and uncontrolled HbA1c groups when the p-value is less than 0.05, representing a statistically significant relationship. The conclusion that poor glycemic control leads to worse periodontal outcomes in T2DM patients is supported by the finding that uncontrolled HbA1c was significantly associated with increased severity of periodontitis using the Chi-square test.

A strong correlation between the GI and HbA1c status. Gingival inflammation was found to be more severe in patients with uncontrolled glycemic levels (HbA1c) than in those with controlled levels. In particular, only 16 out of 96 (16.7%) patients in the controlled group had "Severe" gingival index scores, compared to 60 out of 156 (38.5%) patients in the uncontrolled group. In contrast, the controlled group had a higher prevalence of "mild" gingival inflammation (35.4%) compared to the uncontrolled group (14.1%). A p-value of less than 0.001 was obtained from statistical analysis using the Chi-square test, which produced a value of  $\chi^2 = 26.15$  with two degrees of freedom (df). This confirms that the association between poor glycemic control and increased severity of gingival disease is statistically highly significant.

**Table 3.16:** *Association between HbA1c status and the gingival index (GI)*

GI	Controlled n=96	Uncontrolled n=156	Total	$\chi^2$	df	p-value
Mild	34	22	56	26.15	2	<0.001
Moderate	46	72	118			
Severe	16	60	76			
Total	96	154	250			

A chi-square test of independence was carried out to examine the association between HbA1c status (Controlled vs. Uncontrolled) and the severity of

CAL. The results show a statistically significant relationship between the two variables,  $\chi^2(2, N = 250) = 21.07, p < 0.001$ .

**Table 3.17:** *Association between HbA1c status and clinical attachment loss (chi-square)*

CAL severity	Controlled n=96	Uncontrolled n=156	Total	$\chi^2$	df	p-value
Mild	40	32	72	21.07	2	<0.001
Moderate	40	66	106			
Severe	16	56	72			
Total	96	154	250			

**1. Statistical Significance**

The P-value is 0.04, which is less than the standard threshold of 0.05. This shows a statistically significant correlation between HbA1c status and the presence of periodontal disease. We can reject the null hypothesis that there is no association between these two variables.

**2. Prevalence of Disease**

In the uncontrolled group, 126/154 patients have periodontal disease. In the controlled group, 72/96 patients have periodontal disease. While both groups show a high prevalence, the percentage is higher in those with uncontrolled HbA1c levels.

**Table 3.18:** *Association between HbA1c status and presence of periodontal Disease*

HbA1c status	Disease present	Disease Absent	Total	$\chi^2$	df	p-value
Controlled	72	24	96	4.13	1	0.04
Uncontrolled	126	28	154			

Total	198	52	250
-------	-----	----	-----

#### 4. DISCUSSION

##### 4.1 Overview of the Study

The severity of periodontitis in individuals with T2DM is one of the most common conditions worldwide. It affects people of all ages and socioeconomic statuses. Geographical location, oral hygiene practices, dietary habits, and access to dental care are some of the variables that affect the prevalence. At the 89 Military Dental Center, CMH Kohat, we carried out an observational cross-sectional study to find out how severe periodontal disease was in people with T2DM who were between the ages of 30 and 70. A sampling technique was used to gather data, and the population was divided into four age groups: 30-40, 41-50, 51-60, and 61-70. Of the 250 patients in our data, 56.8 were men and 43.2% (74) were women with severe periodontal disease in T2DM.

##### 4.2 Demographic Characteristics of Study Participants

The average age of the study's participants was  $49.8 \pm 10.4$  years, which is in line with the age range that T2DM usually affects. Grossi *et al.* (75) and Taylor *et al.* (76) reported similar age distributions and discovered that middle-aged and older individuals are more likely to have periodontal destruction associated with diabetes mellitus. Men made up 56.8% of the sample, while women made up 43.2%. The preponderance of men in the study is consistent with research by Soskolne and Klinger (29), who suggested that men often have poorer dental hygiene practices and put off getting medical care.

##### 4.3 Duration of Diabetes and Periodontal Status

The duration of DM is a significant factor influencing periodontal health. In the current study, 40% of patients had DM for more than ten years, and 41.6% had it for one to ten years. Long-term diabetes mellitus is associated with cumulative hyperglycemic exposure, which can lead to microvascular damage, immune system impairment, and delayed wound healing. Consistent with the current findings, patients with DM for more than ten years showed significantly higher CAL and PD.

##### 4.4 Glycemic Control and HbA1c Levels

The mean HbA1c value for the study was  $8.1 \pm 1.4\%$ , suggesting that participants' glycaemic control was generally subpar. Only 38.4% of patients had controlled DM (HbA1c < 7%), whereas 61.6% of patients had uncontrolled glycaemic status. These results are in line with research by Mealey and Oates (4) and Khader *et al.* (77), who discovered that the

majority of diabetic patients with periodontal disease had elevated HbA1c levels. Chronic hyperglycemia alters collagen metabolism and heightens inflammatory responses in periodontal tissues, resulting in the production of AGEs.

##### 4.5 Oral Hygiene Practices and Periodontal Health

Oral hygiene practices are a significant factor in the development of periodontitis. In this study, 53.6% of patients brushed once a day, while only 27.2% brushed twice. Additionally, only 29.6% of participants said they used interdental devices. Poor oral hygiene practices among diabetic patients have been previously documented by Preshaw *et al.* (67), who emphasize that in hyperglycemic conditions, poor plaque control exacerbates periodontal inflammation. The low use of interdental aids may account for the high prevalence of moderate to severe periodontitis observed in this study.

##### 4.6 Gingival Inflammation and Glycemic Status

According to a GI evaluation, 47.2% of patients had moderate gingivitis and 30.4% had severe gingival inflammation. Mild gingivitis was only present in 22.4% of cases. Patients with uncontrolled DM may have more severe gingival inflammation due to decreased neutrophil function and increased production of pro-inflammatory cytokines. Salvi *et al.* (78) reported similar findings, finding that people with DM had increased inflammatory responses.

##### 4.7 Probing Pocket Depth (PPD) and Periodontal Destruction

According to PPD measurements, 29.6% of patients had deep pockets  $\geq 6$  mm, while 44.8% of patients had pockets measuring 4-5 mm. Advanced periodontal destruction is indicated by deep periodontal pockets. The results of Emrich *et al.* (79), who found considerably deeper pockets in patients with poorly controlled DM, are supported by the high prevalence of deep pockets among uncontrolled diabetic patients in this study.

##### 4.8 Clinical Attachment Loss (CAL)

According to CAL analysis, 28.8% of patients had severe attachment loss and 42.4% had moderate CAL. It has been extensively documented that poor glycemic control is linked to elevated CAL. Chronic hyperglycemia has been linked to decreased collagen turnover and increased periodontal breakdown, according to Grossi and Genco (80).

##### 4.9 Bleeding on Probing (BOP)

Widespread periodontal inflammation was indicated by the presence of BOP in 67.2% of patients. The results of Nishimura *et al.* (81), who linked elevated

HbA1c levels to increased gingival bleeding, are consistent with increased BOP among uncontrolled diabetics.

#### 4.10 Overall Periodontal Disease Severity

In the current study, 31.2% of patients had severe periodontal disease, and 43.2% had moderate periodontal disease. The percentage of people with mild disease was only 25.6%. The influence of DM on periodontal health is highlighted by the high prevalence of moderate to severe periodontitis, which also highlights the necessity of integrated medical-dental care.

#### 4.11 Association Between HbA1c and Periodontal Disease Severity

The degree of periodontitis and HbA1c status were discovered to be statistically significantly correlated ( $p < 0.001$ ). Sixty-two patients with uncontrolled diabetes had severe periodontal disease, compared to only sixteen in the control group. These findings support the hypothesis that poor glycemic control is associated with increased periodontal destruction, which is consistent with studies by Taylor (2001) and Preshaw (2012), (82).

#### 4.12 Presence of Periodontal Disease in Study Population

Diabetic patients have a high disease burden, as evidenced by the 79.2% of participants who had periodontal disease. Similar prevalence rates have been reported by South Asian populations, highlighting the regional significance of the issue.

#### 4.13 Biological Mechanisms Linking Diabetes and Periodontal Disease

Numerous mechanisms can account for the association discovered in this study:

- Increased AGEs formation
- Impaired neutrophil chemotaxis
- Altered cytokine profiles
- Microvascular changes
- Delayed tissue repair

People with diabetes mellitus experience increased periodontal deterioration as a result of these processes.

#### 4.14 Clinical Implications

The findings emphasize the importance of:

- Regular periodontal screening in diabetic patients
- Collaborative care between physicians and dentists
- Strict glycemic control to improve periodontal outcomes

#### 4.15 Conclusion of Discussion

The current study clearly demonstrates that poor glycemic control is strongly and statistically significantly linked to more severe periodontal disease in patients with type 2 diabetes. These findings demonstrate the need for integrated management strategies to improve systemic and oral health outcomes.

#### 5. REFERENCES

1. Bascones-Martinez A, Matesanz-Perez P, Escribano-Bermejo M, González-Moles M-Á, Bascones-Ilundain J, Meurman J-H. Periodontal disease and diabetes-Review of the Literature. *Med Oral Patol Oral Cir Bucal*. 2011;16(6):e722-9.
2. Casanova L, Hughes F, Preshaw P. Diabetes and periodontal disease: a two-way relationship. *British dental journal*. 2014;217(8):433-7.
3. Pranckeviciene A, Siudikiene J, Ostrauskas R, Machiulskiene V. Severity of periodontal disease in adult patients with diabetes mellitus in relation to the type of diabetes. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub*. 2014;158(1):117-23.
4. Mealey BL, Oates TW. Diabetes mellitus and periodontal diseases. *Journal of periodontology*. 2006;77(8):1289-303.
5. Mealey BL, Ocampo GL. Diabetes mellitus and periodontal disease. *Periodontology* 2000. 2007;44(1).
6. Grant-Theule D, editor *Periodontal disease, diabetes, and immune response: a review of current concepts*. The Journal of the Western Society of Periodontology/Periodontal Abstracts; 1996.
7. Salvi GE, Collins JG, Yalda B, Arnold RR, Lang NP, Offenbacher S. Monocytic TNF $\alpha$  secretion patterns in IDDM patients with periodontal diseases. *Journal of clinical periodontology*. 1997;24(1):8-16.
8. Schmidt AM, Weidman E, Lalla E, Hori O, Brett JG, Lamster IB. Advanced glycation endproducts (AGEs) induce oxidant stress in the gingiva: a potential mechanism underlying accelerated periodontal disease associated with diabetes. *Journal of periodontal research*. 1996;31(7).
9. Khader YS, Dauod AS, El-Qaderi SS, Alkafajei A, Batayha WQ. Periodontal status of diabetics compared with nondiabetics: a meta-analysis.

- Journal of Diabetes and its Complications. 2006;20(1):59-68.
10. Løe H. Periodontal disease: the sixth complication of diabetes mellitus. *Diabetes care*. 1993;16(1):329-34.
  11. Lalla E, Papapanou PN. Diabetes mellitus and periodontitis: a tale of two common interrelated diseases. *Nature Reviews Endocrinology*. 2011;7(12):738-48.
  12. Cloete L. Diabetes mellitus: an overview of the types, symptoms, complications and management. *Nursing Standard (Royal College of Nursing (Great Britain))*: 1987). 2021;37(1):61-6.
  13. Kumar A, Gangwar R, Ahmad Zargar A, Kumar R, Sharma A. Prevalence of diabetes in India: A review of IDF diabetes atlas 10th edition. *Current diabetes reviews*. 2024;20(1):105-14.
  14. Kengne AP, Ramachandran A. Feasibility of prevention of type 2 diabetes in low-and middle-income countries. *Diabetologia*. 2024;67(5):763-72.
  15. Golovaty I, Ritchie ND, Tuomilehto J, Mohan V, Ali MK, Gregg EW, et al. Two decades of diabetes prevention efforts: a call to innovate and revitalize our approach to lifestyle change. *Diabetes research and clinical practice*. 2023;198:110195.
  16. Hossain MJ, Al-Mamun M, Islam MR. Diabetes mellitus, the fastest growing global public health concern: Early detection should be focused. *Health Science Reports*. 2024;7(3):e2004.
  17. Pan J, Jia W. Early-onset diabetes: an epidemic in China. *Frontiers of medicine*. 2018;12(6):624-33.
  18. Kyrou I, Tsigos C, Mavrogianni C, Cardon G, Van Stappen V, Latomme J, et al. Sociodemographic and lifestyle-related risk factors for identifying vulnerable groups for type 2 diabetes: a narrative review with emphasis on data from Europe. *BMC endocrine disorders*. 2020;20(Suppl 1):134.
  19. Serbis A, Giapros V, Kotanidou EP, Galli-Tsinopoulou A, Siomou E. Diagnosis, treatment and prevention of type 2 diabetes mellitus in children and adolescents. *World journal of diabetes*. 2021;12(4):344.
  20. Schnurr TM, Jakupović H, Carrasquilla GD, Ångquist L, Grarup N, Sørensen TI, et al. Obesity, unfavourable lifestyle and genetic risk of type 2 diabetes: a case-cohort study. *Diabetologia*. 2020;63(7):1324-32.
  21. Negrato CA, Martins RLdM, Louro MD, Medeiros GA, Lanzarin JV, Zajdenverg L, et al. Association between perinatal and obstetric factors and early age at diagnosis of type 1 diabetes mellitus: a cohort study. *Journal of Pediatric Endocrinology and Metabolism*. 2024;37(8):673-9.
  22. Azeem S, Khan U, Liaquat A. The increasing rate of diabetes in Pakistan: A silent killer. *Annals of medicine and surgery*. 2022;79:103901.
  23. Abbas E, Fawwad A, Siddiqui IA, Afzal MS, Ansar M, Saqib MAN, et al. Risk Factors for the Development of Early Onset Diabetes in the Population of Sindh Province, Pakistan. *Biomedicines*. 2025;13(5):1107.
  24. Robison Jr WG, Kador PF, Kinoshita JH. RCapillaries: basement membrane thickening by galactosemia prevented with aldose reductase inhibitor. *Science*. 1983;221(4616):1177-9.
  25. Brownlee M. Glycation products and the pathogenesis of diabetic complications. *Diabetes care*. 1992;15(12):1835-43.
  26. Control D, Group CTR. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *New England journal of medicine*. 1993;329(14):977-86.
  27. Diagnosis ECot, Mellitus CoD. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes care*. 1997;20(7):1183-97.
  28. Association AD. Standards of medical care in diabetes—2007. *Diabetes care*. 2007;30(suppl\_1):S4-S41.
  29. Soskolne WA, Klinger A. The relationship between periodontal diseases and diabetes: an overview. *Annals of periodontology*. 2001;6(1):91-8.
  30. DeFronzo RA. From the triumvirate to the ominous octet: a new paradigm for the treatment of type 2 diabetes mellitus. *diabetes*. 2009;58(4):773-95.
  31. DeFronzo RA, Ferrannini E, Groop L, Henry RR, Herman WH, Holst JJ, et al. Type 2 diabetes mellitus. *Nature reviews Disease primers*. 2015;1(1):1-22.
  32. Abdul-Ghani MA, Tripathy D, DeFronzo RA. Contributions of  $\beta$ -cell dysfunction and insulin

- resistance to the pathogenesis of impaired glucose tolerance and impaired fasting glucose. *Diabetes care*. 2006;29(5):1130-9.
33. Gerstein HC, Santaguida P, Raina P, Morrison KM, Balion C, Hunt D, et al. Annual incidence and relative risk of diabetes in people with various categories of dysglycemia: a systematic overview and meta-analysis of prospective studies. *Diabetes research and clinical practice*. 2007;78(3):305-12.
  34. Federation D. *IDF Diabetes Atlas 6th edn* (Brussels: International Diabetes Federation). 2013.
  35. Chan JC, Malik V, Jia W, Kadowaki T, Yajnik CS, Yoon K-H, et al. Diabetes in Asia: epidemiology, risk factors, and pathophysiology. *Jama*. 2009;301(20):2129-40.
  36. Koro CE, Bowlin SJ, Bourgeois N, Fedder DO. Glycemic control from 1988 to 2000 among US adults diagnosed with type 2 diabetes: a preliminary report. *Diabetes care*. 2004;27(1):17-20.
  37. Ong KL, Cheung BM, Wong LY, Wat NM, Tan KC, Lam KS. Prevalence, treatment, and control of diagnosed diabetes in the US National Health and Nutrition Examination Survey 1999–2004. *Annals of epidemiology*. 2008;18(3):222-9.
  38. Stolar M. Glycemic control and complications in type 2 diabetes mellitus. *The American journal of medicine*. 2010;123(3):S3-S11.
  39. Duckworth W, Abraira C, Moritz T, Reda D, Emanuele N, Reaven PD, et al. Glucose control and vascular complications in veterans with type 2 diabetes. *New England journal of medicine*. 2009;360(2):129-39.
  40. Gerstein H. Action to Control Cardiovascular Risk in Diabetes Study Group, Effects of intensive glucose lowering in type 2 diabetes. *N Engl j Med*. 2008;8:2545-59.
  41. Sheiham A, Netuveli GS. Periodontal diseases in Europe. *Periodontology 2000*. 2002;29(1).
  42. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Annals of periodontology*. 1999;4(1):1-6.
  43. Albandar JM, Kingman A. Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988-1994. *Journal of periodontology*. 1999;70(1):30-43.
  44. Miller A. Oral health of United States adults. The national survey of oral health in US employed adults and seniors, 1985-86. 1987.
  45. Kelly JE, Van Kirk LE, Garst CC. *Oral Hygiene in Adults, United States, 1960-1962: US Department of Health, Education, and Welfare, Public Health Service; 1966.*
  46. Kelly JE, Harvey CR. Basic data on dental examination findings of persons 1-74 years, United States, 1971-1974: Department of Health, Education, and Welfare, Public Health Service, Office ...; 1979.
  47. Brown L, Oliver R, Loe H. Periodontal diseases in the US in 1981: prevalence, severity, extent, and role in tooth mortality. *Journal of periodontology*. 1989;60(7):363-70.
  48. Trindade D, Carvalho R, Machado V, Chambrone L, Mendes JJ, Botelho J. Prevalence of periodontitis in dentate people between 2011 and 2020: a systematic review and meta-analysis of epidemiological studies. *Journal of clinical periodontology*. 2023;50(5):604-26.
  49. Kassebaum N, Bernabé E, Dahiya M, Bhandari B, Murray C, Marcenes W. Global burden of severe periodontitis in 1990-2010: a systematic review and meta-regression. *Journal of dental research*. 2014;93(11):1045-53.
  50. Chen MX, Zhong YJ, Dong QQ, Wong HM, Wen YF. Global, regional, and national burden of severe periodontitis, 1990–2019: An analysis of the Global Burden of Disease Study 2019. *Journal of clinical periodontology*. 2021;48(9):1165-88.
  51. James SL, Abate D, Abate KH, Abay SM, Abbafati C, Abbasi N, et al. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *The lancet*. 2018;392(10159):1789-858.
  52. Villoria GE, Fischer RG, Tinoco EM, Meyle J, Loos BG. Periodontal disease: A systemic condition. *Periodontology 2000*. 2024;96(1):7-19.
  53. Joshipura KJ, Muñoz-Torres FJ, Dye BA, Leroux BG, Ramírez-Vick M, Pérez CM. Longitudinal association between periodontitis and development of diabetes. *Diabetes research and clinical practice*. 2018;141:284-93.

54. Nagata T. Relationship between diabetes and periodontal disease. *Clinical Calcium*. 2009;19(9):1291-8.
55. Daniele G, Guardado Mendoza R, Winnier D, Fiorentino T, Pengou Z, Cornell J, et al. The inflammatory status score including IL-6, TNF- $\alpha$ , osteopontin, fractalkine, MCP-1 and adiponectin underlies whole-body insulin resistance and hyperglycemia in type 2 diabetes mellitus. *Acta diabetologica*. 2014;51(1):123-31.
56. Zhang Q, Fang W, Ma L, Wang Z-D, Yang Y-M, Lu Y-Q. VEGF levels in plasma in relation to metabolic control, inflammation, and microvascular complications in type-2 diabetes: a cohort study. *Medicine*. 2018;97(15):e0415.
57. Bitar MS, Ayed AK, Abdel-Halim SM, Isenovic ER, Al-Mulla F. Inflammation and apoptosis in aortic tissues of aged type II diabetes: amelioration with  $\alpha$ -lipoic acid through phosphatidylinositol 3-kinase/Akt-dependent mechanism. *Life Sciences*. 2010;86(23-24):844-53.
58. Duran-Salgado MB, Rubio-Guerra AF. Diabetic nephropathy and inflammation. *World journal of diabetes*. 2014;5(3):393.
59. Elmarakby AA, Sullivan JC. Relationship between oxidative stress and inflammatory cytokines in diabetic nephropathy. *Cardiovascular therapeutics*. 2012;30(1):49-59.
60. Navarro-González JF, Mora-Fernández C, De Fuentes MM, García-Pérez J. Inflammatory molecules and pathways in the pathogenesis of diabetic nephropathy. *Nature Reviews Nephrology*. 2011;7(6):327-40.
61. Behl Y, Krothapalli P, Desta T, DiPiazza A, Roy S, Graves DT. Diabetes-enhanced tumor necrosis factor- $\alpha$  production promotes apoptosis and the loss of retinal microvascular cells in type 1 and type 2 models of diabetic retinopathy. *The American journal of pathology*. 2008;172(5):1411-8.
62. Graves DT, Ding Z, Yang Y. The impact of diabetes on periodontal diseases. *Periodontology 2000*. 2020;82(1):214-24.
63. SG G. Assessment of risk for periodontal disease II. Risk indicators for alveolar bone loss. *J Periodontol*. 1995;66:23-9.
64. Katagiri S, Nitta H, Nagasawa T, Izumi Y, Kanazawa M, Matsuo A, et al. Effect of glycemic control on periodontitis in type 2 diabetic patients with periodontal disease. *Journal of diabetes investigation*. 2013;4(3):320-5.
65. Goyal L, Gupta N, Bey A. Periodontal therapy: a useful adjunct to improve glycemic control. *Journal of Pharmacy and Bioallied Sciences*. 2012;4(2):173-4.
66. Jimenez M, Hu FB, Marino M, Li Y, Joshipura KJ. Type 2 diabetes mellitus and 20 year incidence of periodontitis and tooth loss. *Diabetes research and clinical practice*. 2012;98(3):494-500.
67. Preshaw PM, Alba AL, Herrera D, Jepsen S, Konstantinidis A, Makrilakis K, et al. Periodontitis and diabetes: a two-way relationship. *Diabetologia*. 2012;55(1):21-31.
68. Mirza B, Syed A, Izhar F, Ali Khan A. Bidirectional relationship between diabetes and periodontal disease: review of evidence. *J Pak Med Assoc*. 2010;60(9):766-8.
69. Genco RJ, Grossi SG, Ho A, Nishimura F, Murayama Y. A proposed model linking inflammation to obesity, diabetes, and periodontal infections. *Journal of periodontology*. 2005;76:2075-84.
70. Stoicescu M, Calniceanu H, Ţig I, Nemeth S, Tent A, Popa A, et al. Significant aspects and correlation between glycemic control and generalized chronic periodontitis in type 2 diabetes mellitus patients. *Experimental and Therapeutic Medicine*. 2021;22(1):671.
71. Salvi GE, Carollo-Bittel B, Lang NP. Effects of diabetes mellitus on periodontal and peri-implant conditions: update on associations and risks. *Journal of clinical periodontology*. 2008;35:398-409.
72. Chávarry N, Vettore MV, Sansone C, Sheiham A. The relationship between diabetes mellitus and destructive periodontal disease: a meta-analysis. *Oral health prev dent*. 2009;7(2):107-27.
73. Tsai C, Hayes C, Taylor GW. Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population. *Community dentistry and oral epidemiology*. 2002;30(3):182-92.
74. Qureshi A, Haque Z, Bokhari SAH, Baloch AA. Evaluation of HbA1c in type-2 diabetes mellitus patients with periodontitis: preliminary findings of three-arm clinical trial. *JPMA*. 2020;2020.
75. Grossi SG, Skrepicki FB, DeCaro T, Robertson DC, Ho AW, Dunford RG, et al. Treatment of periodontal disease in diabetics reduces glycated hemoglobin. *Journal of periodontology*. 1997;68(8):713-9.

76. Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M, Knowler WC, et al. Severe periodontitis and risk for poor glycemic control in patients with non-insulin-dependent diabetes mellitus. *Journal of periodontology*. 1996;67:1085-93.
77. Khader YS, Albashaireh ZS, Alomari MA. Periodontal diseases and the risk of coronary heart and cerebrovascular diseases: A meta-analysis. *Journal of periodontology*. 2004;75(8):1046-53.
78. Salvi GE, Kandylaki M, Troendle A, Persson GR, Lang NP. Experimental gingivitis in type 1 diabetics: a controlled clinical and microbiological study. *Journal of clinical periodontology*. 2005;32(3):310-6.
79. Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in non-insulin-dependent diabetes mellitus. *Journal of periodontology*. 1991;62(2):123-31.
80. Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: a two-way relationship. *Annals of periodontology*. 1998;3(1):51-61.
81. Nishimura F, Murayama Y. CONCISE REVIEW biological: Periodontal inflammation and insulin resistance—lessons from obesity. *Journal of Dental Research*. 2001;80(8):1690-4.