Original Article

Oral lesions and periodontal status in diabetics and non-diabetics: A hospital based study

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Abstract

Background: Diabetes mellitus, a chronic hyperglycemic disorder has been associated with several manifestations in the body including the oral cavity. The oral manifestations are of importance and may significantly contribute to the detection of undiagnosed diabetes.

Objective: To find out the prevalence of oral diseases in type 1 and type 2 diabetics, to evaluate and compare the periodontal status of diabetics and non-diabetics.

Materials and Methods: This was a cross sectional study conducted on 110 diabetic subjects of >5 years duration at the M.V. Hospital for Diabetes, Diabetes Research Centre, Chennai. One hundred apparently healthy non-diabetic subjects in the age group of 40-60 years were studied as control. The oral cavity was examined by using a mouth mirror and periodontal status was assessed by utilizing oral hygiene index - simplified (Greene and Vermillion), Miller's mobility index and Ramjford's periodontal disease index.

Results: Subjects with diabetes were found to have significant association with xerostomia ($P < 0.003$) and chronic periodontitis ($P < 0.026$). However, no significant association was found forleukoplakia, traumatic ulcer, frictional keratosis, smoker's melanosis, mucocele, aphthous ulcer, fissured tongue, lichen planus, parotid enlargement, parulis, chronic gingivitis, fibroma, and periodontal abscess.

Conclusion: The study reaffirmed higher prevalence of xerostomia and periodontal disease among diabetic subjects. However, contrary to previous studies, no significant differences were found in the prevalence of traumatic ulcer, fissured tongue, lichen planus, and parotid enlargement.

Keywords: Chronic periodontitis, diabetes, glycosylated hemoglobin, hyperglycemia, oral lesions, xerostomia

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Submitted: 03-Feb-2021, **Accepted:** 22-Mar-2022, **Published:** 17-Oct-2022

INTRODUCTION

Diabetes mellitus (DM) is not a single disease entity, but rather a group of metabolic disorders sharing the common underlying feature of hyperglycemia.[1] People living with DM are expected to rise from 171 million in 2000 to 366 million in 2030[2] or to 642 million in 2040.[3] DM is mainly of two types- type 1 and type 2 DM. Type 1 DM, characterised by an absolute deficiency of insulin caused by pancreatic β cell destruction, accounts for approximately 10% of all cases. Type 2 DM, caused by a combination of peripheral resistance to insulin action and an inadequate secretory response by the pancreatic β cells, approximates 80% to 90% of all cases.

Access this article online

Quick Response Code:

Website: www.jomfp.in

DOI: 10.4103/jomfp.jomfp_37_21

How to cite this article: Sanjeeta N, Sivapathasundharam B, Nandini DB. Oral lesions and periodontal status in diabetics and non-diabetics: A hospital based study. J Oral Maxillofac Pathol 2022;26:419.

DM has been associated with a number of changes in the oral cavity. Benign migratory glossitis (geographic tongue), fissured tongue, median rhomboid glossitis, hyperplastic gingivitis, lichen planus, parotid gland enlargement, higher prevalence and severity of periodontal disease, fungal infections, alterations in salivary flow rates and composition or dental caries, increased tendency to infections, and defective wound healing are some of the oral findings that have been associated with diabetes.[4] Potentially malignant disorders such as leukoplakia, erythroplakia, or oral manifestations of candidiasis, including denture stomatitis, or angular cheilitis have also been reported in diabetics. Both type 1 and type 2 DM have more severe periodontal disease than subjects without diabetes.

The aim of the study is to find out the prevalence of oral diseases in type 1 and type 2 diabetics, to evaluate and compare the periodontal status of diabetics and non-diabetics and also to determine the possible association of the oral diseases and periodontal status with age, type of DM, duration, metabolic control, and hygiene status in diabetics.

MATERIALS AND METHODS

The study consisted of 210 individuals in the age group of 40-60 years, out of which 110 were diabetic and 100 were non-diabetic. Before the commencement of the study, ethical clearance was obtained from the Institutional Ethical committee. The study group consisted of 10 subjects with type 1 diabetes mellitus (Group A), 3 females and 7 males and 100 subjects with type 2 diabetes mellitus (Group B), 45 males and 55 females. All the diabetics represented the disease for a duration of not less than one year. The diabetics were from M.V. Hospital for Diabetics, Diabetics Research Center, Royapuram, Chennai (a referral hospital for diabetics). The control group (Group C) consisted of 100 apparently healthy subjects, 50 males and 50 females.

Diabetics with any severe illness other than diabetes, those with diabetic ketoacidosis, and edentulous patients were excluded from the study. Third molars were excluded during assessment of periodontal status.

Data concerning the type of diabetes, level of metabolic control, duration, and complications were retrieved from the medical records of the subjects.

The level of metabolic control of the diabetics was based on the measurement of glycosylated haemoglobin. Diabetics with glycosylated haemoglobin below 7% is considered well controlled, those with 7% and above and below 9% are considered moderately controlled and those with 9% or more is considered poorly controlled.

All the subjects who were selected for the study were questioned regarding their oral hygiene habit, other habits like smoking, betel-nut chewing and the like. These data were recorded in the proforma.

Clinical examination for all the patients was conducted by the same examiner, using a mouth mirror, dental explorer and William's graduated periodontal probe, and recorded.

Periodontal examination was done using oral hygiene index-simplified (Greene and Vermillion), Miller's mobility index and Ramfjord's periodontal disease index, and recorded in the proforma.

The data obtained from the study were tabulated and subjected to statistical analysis. The test of significance between groups was performed by Student's Independent t- test and the test of significance for proportions were done by z-test.

RESULTS

The data obtained from the study are presented in tabular columns. Table 1 shows the distribution of subjects, comprising a total of 102 males and 108 females. The glycemic status [Table 1] of the diabetics was retrieved from the case records of the subjects, in order to determine the possible association of the prevalence of oral mucosal pathosis with the level of glycemic status in diabetics. The glycemic status of the majority of the diabetics was moderately controlled. All the diabetics were also questioned about the duration of diabetes [Table 1] in order to find out whether the duration of diabetes has an influence on the prevalence of oral diseases. 38% of type 2 diabetics had diabetes for more than 5 years and 27% had diabetes for more than 10 years. The habits of all the subjects who were examined [Table 1] were also questioned and recorded. The significance of habits cannot be overemphasized because certain habits produce well-known lesions of the oral mucosa. Table 2 shows the prevalence of oral diseases in diabetics and non-diabetics. Diabetics had significant association with xerostomia ($P < 0.003$) and chronic periodontitis ($P < 0.026$). Table 3 shows the mean and standard deviation of the parameters for periodontal assessment.

DISCUSSION

Diabetes mellitus has been found to be associated with a number of oral changes. These oral changes are not always

Table 1: Distribution of Gender, Duration, Metabolic Status and Habit

	Type 1/Group A(*n*=10)	Type 2/Group B(*n*=100)	Control/Group C(*n*=100)
Gender			
Male	7 (70%)	45 (45%)	50 (50%)
Female	3 (30%)	55 (55%)	50 (50%)
Duration			
<2 yrs	-	16 (16%)	NA
2-5 yrs	-	19 (19%)	
6-10 yrs	1 (10%)	38 (38%)	
>10 yrs	9 (90%)	27 (27%)	
Controlled status			
Well	1 (10%)	15 (15%)	NA
Moderate	8 (80%)	65 (65%)	
Poorly	1 (10%)	20 (20%)	
Habit			
Smoking	1 (10%)	12 (12%)	22 (22%)
Alcohol	1 (10%)	5 (5%)	14 (14%)
Betel nut chewing	2 (20%)	7 (7%)	11 (11%)
Tobacco chewing	-	2 (2%)	8 (8%)
Others	1 (10%)	9 (9%)	2 (2%)

Table 2: Prevalence of Oral Mucosal Pathoses

Lesion	Group A/Type 1(*n*=10)	Group B/Type 2(*n*=100)	Group C/ Control (*n*=100)	*P*
Leukoplakia	10% (*n*=1)	3% (*n*=3)	8% (*n*=8)	0.121
Traumatic ulcer	10% (*n*=1)	4% (*n*=4)	1% (*n*=1)	0.173
Frictional keratosis	-	1% (*n*=1)	1% (*n*=1)	1.00
Smoker's melanosis	10% (*n*=1)	4% (*n*=4)	6% (*n*=6)	0.517
Mucocele	-	-	1% (*n*=1)	0.751
Apthous ulcer	-	3% (*n*=3)	3% (*n*=3)	1.00
Fissured tongue	-	2% (*n*=2)	-	0.89
Lichen planus	-	1% (*n*=1)	-	0.751
Parotid enlargement	-	2% (*n*=2)	-	0.89
Parulis	-	1% (*n*=1)	1% (*n*=1)	1.00
Chronic gingivitis	80% (*n*=8)	89% (*n*=89)	96% (*n*=96)	0.938
Chronic periodontitis	20% (*n*=2)	11% (*n*=11)	3% (*n*=3)	0.026
Fibroma	-	-	1% (*n*=1)	0.751
Periodontal abscess	-	5% (*n*=5)	1% (*n*=1)	0.09
Xerostomia	20% (*n*=2)	13% (*n*=13)	2% (*n*=2)	0.003 (Significant)

Table 3: Results of Periodontal Assessment (Mean±SD)

	Type I/ Group A (*n*=10)	Type 2/ Group B (*n*=100)	Control/ Group C (*n*=100)	*P*
OHI-S	2.96±0.8	3.16±0.75	2.43±0.95	0.000(S)
Miller's index	0.05±0.05	0.09±0.06	0.03±0.05	0.000(S)
Ramfjord's PDI	2.79±0.48	3.11±0.58	2.46±0.83	0.000(S)

present, are non-specific and are not pathognomonic for diabetes. The oral changes are less likely to be seen in well-controlled diabetics. In a systematic review by José González-Serrano *et al*.,[5] a higher prevalence of oral mucosal disorders was found in patients with DM compared to non-DM patients. This prevalence ranged from 45–88% in T2DM patients to 38.3–45% in non-DM groups and from 44.7% in T1DM patients to 25% in the non-DM population.

Specific oral lesions that have been consistently detected in DM include benign migratory glossitis (geographic tongue), fissured tongue, median rhomboid glossitis, hyperplastic gingivitis, lichen planus, and parotid gland enlargement.

Fissured tongue,[6] including generalized plication, and a double fissure running longitudinally along the dorsum of the tongue, has been reported to be more prevalent in diabetic patients. The pathogenesis of fissured tongue is considered to be a genetically determined developmental variant, a manifestation of aging or changes in the oral environment. In a study of 405 diabetics,[6] the prevalence rate of the fissured tongue was found to be 5.4% and its presence was significantly related to the older subjects who had a longer duration of diabetes. Our study found a prevalence of 2%. The lesser prevalence rate in our study could be attributed to the narrow age range of our subjects as compared to the wide age range of the previous studies done on diverse populations worldwide. Farman[7] conducted a study on 175 diabetics, whose ages ranged from 13 to 79 years, having diabetes ranging from less than 1 week upto 35 years. Fissuring of the dorsal surface of the tongue was present in 49 patients, a prevalence of 28.0%.

The prevalence rates of fissured tongue in a normal population vary, ranging from 0.8% in Nigerians, and Saudi Arabians to 30.5% in Israelis.[8]

In a study on 10,000 healthy Indian subjects in the age group of 5 to 20 years, to assess the prevalence of developmental anomalies of the tongue, fissured tongue was found in 2.71% of the population.[8] Our study conducted on the same population but of the older age group (40-60 years), and having DM gave a prevalence rate of 2%. The similar prevalence rates suggest that age may not be an etiologic factor in the pathogenesis of fissured tongue in this part of the population, contrary to suggestions that aging may be a causative factor for the fissured tongue. Based on the findings of our study, it is quite apparent that fissured tongue may not be a manifestation of aging or of DM.

Xerostomia,[9,10] a subjective sensation of dry mouth has been consistently found to be increased in patients with diabetes. In this study, two type 1 diabetics, 13 type 2 diabetics, and two controls have been found to have xerostomia, giving a prevalence of 13% in type 2 diabetes, and a prevalence of 2% in controls (*P* value = 0.003) which is a significant finding.

All the subjects who presented with xerostomia were ruled out of other causes by history and examination. Of the diabetics with xerostomia, only 1 subject smoked. In

the subject who smoked, we cannot ascertain whether xerostomia is due to smoking, medications or DM. However, the prevalence of xerostomia in our diabetic population is a significant finding.

Studies conducted on diabetics to determine salivary secretion have reported either a decreased salivary flow rate or an increased salivary flow rate when compared to controls, and still, others have reported normal salivary flow rates.[11,12] The discrepancy in the results could be due to differences in the method of saliva collection, and stimulation. Although autonomic neuropathy[13] may be a cause, in part of decreased salivation, it appears that diabetic xerostomia is most often a side effect of the patient's medication.

In the present study, we did not find any cases of median rhomboid glossitis and migratory glossitis that have previously been reported to be more prevalent in persons with diabetes. Guggenheimer *et al*.[6,14] conducted a study on 405 Type 1 diabetic subjects and found median rhomboid glossitis in 29 diabetics giving a prevalence rate of 7.2%, and 5.4% for benign migratory glossitis. Whether median rhomboid glossitis and benign migratory glossitis have a higher prevalence with type 1 diabetes cannot precisely be opined based on our study because we examined only 10 cases of type 1DM.

Lichen planus[15] is an immunologically mediated mucocutaneous disorder, first described by Erasmus Wilson in 1869. In the present study, we identified only a single case of oral lichen planus (OLP) in a 60-year-old female subject, diagnosed with type 2 diabetes and hypertension for about 22 years, giving a prevalence of 1%. The lesion was present on both the right and left buccal mucosa.

Our prevalence rate of 1% is consistent with that of previous studies. In 1981, Martinez Pena failed to find a single case of OLP (1%) in 100 diabetic patients, and in 1985, Lozada-Nur *et al*.[16] reported only two cases of OLP in 99 diabetic subjects, giving a prevalence of 1.68%. Borghelli *et al*.conducted three separate studies on 240, 584 and 729 diabetics (in 1986, 1987 & 1993), and found a prevalence of 0.42% (1 subject), 0.17% (1 subject) and 0.55% (four subjects), respectively.[17]

Grinspan *et al*.[18] (1965) reported a prevalence of 5.71% of OLP cases in 70 diabetics.

The relationship between lichen planus, and diabetes has been extensively studied, but the findings vary. Grinspan's syndrome comprises a triad of oral lichen planus, diabetes mellitus, and hypertension but may be simply a coincidental association of three common disorders. Alternatively, what is diagnosed clinically as lichen planus may be a lichenoid reaction, possibly a side effect of medication, as some oral hypoglycemic agents (such as chlorpropamide and tolbutamide) and some antihypertensive drugs (such as methyldopa and thiazide diuretics) are known to provoke lichenoid reactions. Another theory suggests that, because type 1 diabetes is considered to be an autoimmune disorder and patients with other autoimmune diseases, such as systemic lupus erythematosus or graft-Vs-host disease, may manifest lichen planus-like lesions, a similar effect could occur with diabetes.[6] In addition, 'stress' which could aggravate and unmask subclinical diabetes, and hypertension may also be associated with the development of lichen planus.

Sialosis, which is defined as an asymptomatic, non-inflammatory, and non-neoplastic enlargement of the salivary glands due to metabolic causes, has been observed frequently in diabetic subjects. Sialosis has been reported to affect up to 24% of the diabetic population. Both parotid salivary glands are usually affected although the submandibular salivary glands may also be involved. Sialosis is caused by fatty infiltration of the interstitium, and enlargement of acinar cells.[13] In the present study, two subjects with type 2 diabetes for more than 10 years were found to have parotid gland enlargement, giving a prevalence of 2%. None of the control subjects showed parotid enlargement (*P* value-0.89, not significant).

Leukoplakia, according to WHO,[19] is defined as a whitish patch or plaque that cannot be characterized clinically or pathologically as any other disease, and which is not associated with any physical or chemical causative agent except the use of tobacco. Based on a study by Ujpál *et al*.,[20] the incidence of oral leukoplakia was found to be 6.2% among diabetics, and 11.5% among diabetics who smoke. In our diabetic subjects, oral leukoplakia is not a significant finding.

The prevalence traumatic ulcers has been reported of upto 3.5% in a population of 405 type 1 diabetics.[6] The investigators have attributed the traumatic ulcers to habits such as smoking, having consumed larger amounts of alcohol, and having overt nephropathy. Also, several mechanisms like microangiopathy of the oral vasculature or a defect in the function of the polymorphonuclear leukocytes may also be responsible. In our study, the traumatic ulcer is not a significant finding (*P* value- 0.1738).

We also detected frictional keratosis and smoker's melanosis which were equally distributed among the diabetics and controls.

Periodontal abscesses showed a prevalence of 5% in type 2 diabetics and 1% in controls ($P = 0.09$, not significant).

Periodontal disease has been recognised as the sixth complication of DM.[21-23] Plaque-induced gingivitis is the most common form of the gingival disease and is the result of plaque bacteria and defense cells of the host.[24] Systemic factors contributing to gingivitis, such as the endocrine changes associated with puberty, the menstrual cycle, pregnancy, and diabetes, may be exacerbated because of alterations in the gingival inflammatory response to plaque.[23]

Chronic periodontitis is the most common of all periodontal diseases and is associated with the accumulation of plaque, and calculus, and generally has a slow to moderate rate of disease progression. Increases in the rate of disease progression may be caused by the impact of local, systemic, or environmental factors that may influence the normal host-bacterial interaction. Systemic diseases such as DM may influence the host defenses.[21]

Several studies have confirmed that subjects with diabetes have a higher prevalence and severity of periodontal disease than non-diabetics in the same population.

In the present study, diabetics showed higher plaque and calculus index (OHI-S) values than the controls. Diabetics showed a mean OHI-S value of 3.16 which is significantly higher ($P = 0.000$) than the mean OHI-S value of 2.43 for the controls. This finding can be explained by the fact that excess glucose enters into the oral cavity through the saliva and gingival crevicular fluid in patients with diabetes, and a sugar-rich biofilm will enhance plaque growth in general. Lack of knowledge about oral health and oral hygiene management may be related factors in higher plaque index values.

In type 2 DM, the mean mobility index was 0.09 and the mean periodontal disease index score was 3.11 which is significantly higher than that of controls whose mean mobility index was 0.03, and periodontal disease index score was 2.46 (P value = 0.000, significant).

About 89% of type 2 diabetics and 96% of controls had plaque-induced gingivitis. Because of alterations in the gingival inflammatory response to plaque in DM, plaque-induced gingivitis in DM have more propensity of progressing to chronic periodontitis. Chronic periodontitis was seen in 11% of type 2 DM, and 3% of controls (P value = 0.026, statistically significant).

From the periodontal status assessment conducted with the parameters mentioned above, and based on the findings of this study, it is apparent that periodontal disease in diabetics is of higher prevalence as compared to nondiabetics, which is consistent with previous studies.

In the present study on diabetic subjects, we could not determine the possible association of the other oral mucosal diseases with age, duration of diabetes, metabolic control, and complication due to the insignificant and lesser prevalence of the diseases.

The association of specific oral lesions and diabetes is of great significance both in the detection of undiagnosed diabetes and finding out the pathogenesis of various oro-facial diseases in diabetes. The correlation of certain oral diseases of the oral mucosa, and DM is still a matter of debate.

In recent years, in view of the ever-increasing sedentary lifestyles, and poor eating habits that have contributed to the escalation of DM worldwide, which some have termed as diabesity, DM-related oral diseases deserve adequate recognition and further investigation.

CONCLUSION

Diabetes mellitus comprises a group of metabolic disorders characterised by chronic hyperglycemia. Sustained hyperglycemia in DM affects almost all tissues in the body and is associated with significant complications of multiple organ systems. The oral cavity also frequently undergoes changes that are related to the diabetic condition. A cross-sectional epidemiological study was conducted on diabetics, and an attempt was made to find out the prevalence of oral mucosal pathosis. The study also included a periodontal status assessment to determine the prevalence of the periodontal disease. Based on the results we obtained from our diabetic population, we conclude that diabetics have a greater prevalence of xerostomia as compared to controls. Periodontal disease is more prevalent in diabetics than non-diabetics. No significant differences were found in the prevalence of traumatic ulcer, fissured tongue, lichen planus and parotid enlargement, contrary to previous studies which found a greater prevalence in diabetics.

Financial support and sponsorship
Nil.

Conflicts of interest

There are no conflicts of interest.

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