

Evaluation of Prevalence of Dental Caries in Diabetic Tobacco Users in a Sample Indore Population: A Cross Sectional Study.

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Abstract

Aim: The aim of this cross-sectional study was to evaluate and compare the prevalence of dental caries in diabetic tobacco users in a sample Indore population.

Methodology: A total of 200 individuals of age group 20-60 years were enrolled and assessed for their eligibility to participate in the cross sectional study and divided into four groups. Group A: Type II diabetic individuals who used smokeless tobacco, Group B: Type II diabetic individuals with a smoking habit, Group C: Non-diabetic individuals who used smokeless tobacco, Group D: Non-

diabetic individuals with a smoking habit. Random Blood Sugar (RBS) was recorded for all the individuals; only the patients beyond the normal range of RBS, further underwent Fasting Blood Sugar (FBS) estimation for confirmation of presence of Type II diabetes. Their dental caries status was assessed using DMFT/DMFS index.

Results: Intergroup comparison of diabetic and non-diabetic individuals revealed a higher prevalence of dental caries in subjects with type II diabetes mellitus. Intragroup comparison of all the diabetic individuals revealed a significantly higher prevalence of dental caries

in Group A (Type II diabetic individuals who used smokeless tobacco) with DMFT mean value of 7.26 ± 3.829 and DMFS mean value of 13.63 ± 8.539 followed by Group B (Type II diabetic individuals with a smoking habit) where the DMFT mean value were 4.86 ± 2.952 and DMFS mean value were 7.53 ± 3.211 followed by Group C (Non-diabetic individuals who used smokeless tobacco) where DMFT mean value were 3.0 ± 2.952 and DMFS mean value were 5.23 ± 3.721 with least prevalence of dental caries found in Group D (Non-diabetic individuals with a smoking habit) where DMFT mean value were 2.1 ± 1.082 and DMFS mean value were 3.2 ± 1.373 .

Conclusion: Within the limitations of this study, the results indicate that the use of tobacco in any form increases the predisposition to caries development in diabetic as well as nondiabetic individuals. It also indicates that smokeless forms of tobacco are more detrimental than smoked forms of tobacco in caries development.

Keywords: Diabetes, Tobacco, Dental caries

Introduction

Diabetes is one of the most common metabolic disorders and emerges secondary to an interaction between genetic, environmental and lifestyle factors [1]. Diabetic patients are prone to extensive fluid loss due to polyuria, impaired immune response to infections, altered connective tissue metabolism, and various microvascular changes. These factors may lead to various oral diseases like xerostomia, salivary gland dysfunction, lichen planus, periodontal disease, myeloid leukemia, increased susceptibility to infections and also oropharyngeal cancer[2,3,4]. In addition, a higher prevalence of dental caries has been reported in diabetic patients compared to nondiabetics [5]. According to the National Household Survey of Drug and Alcohol Abuse (2002), the overall prevalence of current

tobacco use in India is 55.8% [6] which is consumed in various smokeless and smoked forms. Almost 30% of Indians older than 15 years of age have been reported to use some form of tobacco [7]. Nicotine, present in tobacco, which is a highly addictive component is responsible for tobacco dependence. The various substances present in tobacco smoke trigger free radical processes which has been reported to interfere with vascular homeostasis resulting in increased inflammation/oxidative stresses leading to β -cell dysfunction [8].

Local and systemic effects of tobacco in oral cavity are dependent on the form, frequency, and duration of its use and is also dose dependent [9-14]. The aim of this study, therefore, was to assess the influence of tobacco usage on caries development in type II diabetics in a sample population of Indore region.

Methodology

Two hundred adult volunteers with the habit of smoking or use of smokeless tobacco, belonging to Indore district, within the age group of 20–60 years, who reported to the Department of Conservative Dentistry and Endodontics, were assessed for eligibility to participate in the cross sectional study (data were collected for 4 months from March 2019 to June 2019). Random Blood Sugar (RBS) was recorded for all the individuals; and only the patients with RBS more than 140mg/dl, milligram per decilitre (7.8mmol/l, millimoles per litre) were recalled further for the estimation of Fasting Blood Sugar (FBS) for the confirmation of presence of Type II diabetes. The selection criteria for diabetic individuals included presence of type-II diabetes since at least 3 years, the individuals having FBS limit of 126 mg/dl (7 mmol/L) or higher than that, individuals free of diabetic complications and those who had not undergone any preventive procedure for caries including fluoride exposure. The

nondiabetic individuals who participate in the study had RBS within the normal range of 79-140 mg/dL (4.4-7.8 mmol/l) and with no medical history of diabetes.

The exclusion criteria included individuals over 60 years and less than 20 years of age, those taking drugs which alter salivary parameters, those using both smoked and smokeless tobacco, those who consumed alcohol, those with any systemic disease, history of radiation therapy, salivary gland diseases or disorders and denture wearers.

Sample size calculation using G*power software revealed that minimum of 13 samples per group were required to detect a significant difference in the mean values of DMFT/DMFS scores among the various groups at an alpha of 0.05 and power of 80%. Therefore, in order to have safe representation of samples, it was intended to include more than 13 samples per group. Hence a total of 200 samples were chosen to ensure a safe representative data.

All of the 200 volunteers were evaluated for participation in the study, following the guidelines suggested by the CONSORT (Consolidated Standards of Reporting Trails) group. Ethical clearance was obtained from the Institutional Ethical Committee which was in accordance with the Declaration of Helsinki. The participants were well informed in advance and their written consent was obtained before the start of the study. The individuals who volunteered for study were carefully evaluated. A total 44 volunteers who did not meet the inclusion criteria were excluded from the study. The remaining 156 participants were screened for Type II Diabetes Mellitus, by detailed recording of past medical history, fasting blood sugar (FBS) levels and screening of past medical reports.

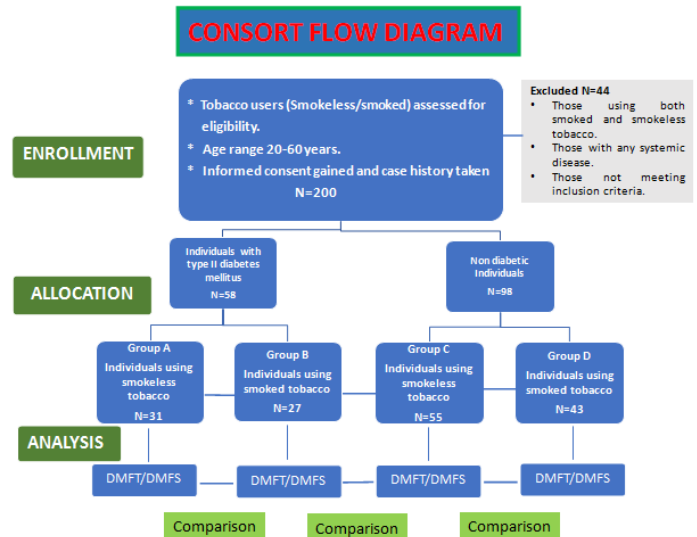
The participants (N=156) were then divided into four study groups.

Group A: Type II diabetic individuals who used smokeless tobacco (N=31)

Group B: Type II diabetic individuals with a smoking habit (N=27)

Group C: Non-diabetic individuals who used smokeless tobacco (N=55)

Group D: Non-diabetic individuals with a smoking habit (N=43)



All cases were examined by a single examiner with assistance from a recorder, who was well versed with case history recording and examination procedure. The dental caries status was assessed in all groups using Decayed-Missing-Filled (DMF) index adopted by the World Health Organization (WHO 1987).

1. DMF teeth index (DMFT) denoting the prevalence of dental caries.
2. DMF surfaces index (DMFS) denoting the severity of dental caries.

Statistical Analysis: The data was entered in an excel sheet and analysed using SPSS software (Statistical Package for Social Sciences), 20.0 version, IBM, Chicago. The probability distribution of data was analysed using Kolmogorov Smirnov test. The p value=0.034 revealed that the data had non-normal distribution. Thus, the non-parametric test of significance was applied. The comparison between the groups was done using Man

Whitney U test. The p value < 0.05 was considered statistically significant. Confidence interval was set at 95%.

Result

Intragroup comparison of all the diabetic individuals revealed a significantly higher prevalence of dental caries in Group A (Type II diabetic individuals who used smokeless tobacco) with DMFT mean value of 7.26 ± 3.829 and DMFS mean value of 13.63 ± 8.539 followed by Group B (Type II diabetic individuals with a smoking habit) where the DMFT mean value were 4.86 ± 2.952 and DMFS mean value were 7.53 ± 3.211 followed by Group C (Non-diabetic individuals who used smokeless tobacco) where DMFT mean value were 3.0 ± 2.952 and DMFS mean value were 5.23 ± 3.721 with least prevalence of dental caries found in Group D (Non-diabetic individuals with a smoking habit) where DMFT mean value were 2.1 ± 1.082 and DMFS mean value were 3.2 ± 1.373.

Table 1: Description of mean & median of DMFT & DMFS score of individuals belonging to different groups.

	Group A		Group B		Group C		Group D	
	DMFT	DMFS	DMFT	DMFS	DMFT	DMFS	DMFT	DMFS
Mean ± SD	7.26 ± 3.829	13.63 ± 8.539	4.86 ± 2.952	7.53 ± 3.211	3.0 ± 2.952	5.23 ± 3.721	2.1 ± 1.082	3.2 ± 1.373
Median	6.0	14.0	3.0	5.0	4.0	7.0	2.0	3.0

SD- Standard deviation DMFT - Decayed-Missing-Filled teeth DMFS - Decayed-Missing-Filled surfaces

Table 2: Description of mean & median of DMFT & DMFS score of individuals belonging to different groups.

	Group A Diabetic Patients Using Smokeless Tobacco		Group B Diabetic Patients Using Smoked Tobacco	
	DMFT	DMFS	DMFT	DMFS
Mean	7.2667	13.6333	4.8600	7.5333
Median(Iq)	6.0000(5-11)	13.0000(6-19)	4.0000(3-6)	7.0000(5-8)
Standard Deviation	3.68782	8.63382	2.99682	3.70071

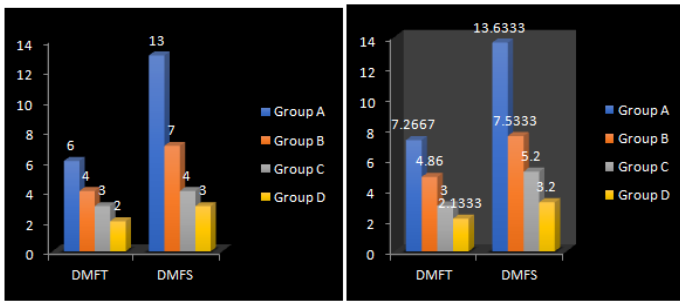
IQ – Inter-Quartile range

	Group C Non Diabetic Patients Using Smokeless Tobacco		Group D Non Diabetic Patients Using Smoked Tobacco	
	DMFT	DMFS	DMFT	DMFS
Mean	3.0000	5.2000	2.1333	3.2000
Median	3.0000(2-4)	4.0000(3-7)	2.0000(1-3)	3.0000(2-4)
Standard Deviation	1.0000	3.27763	1.12546	1.37321

Table 3: Comparison of caries experience of the patients belonging to different groups.

Groups	DMFT P value	DMFS P value
Group B vs group D	0.002*	0.000*
Group A vs Group C	0.000*	0.001*
Group B vs Group C	0.067*	0.030*
Group A vs Group D	0.000*	0.000*
Group B vs Group A	0.051*	0.037*
Group D vs Group C	0.037*	0.055

Graph 1: Showing mean and median DMFT and DMFS of study subjects belonging to 4 groups



On comparing the caries incidence of smokeless and smoked tobacco in diabetic individuals, it was found that those consuming smokeless tobacco reported more caries as compared to smoked tobacco (p value < 0.05). On comparing the caries incidence of smokeless and smoked tobacco users amongst non-diabetic individuals it was found that those consuming smokeless tobacco experience statistically more significant caries as compared to individuals with a smoking history (p value < 0.05).

Maximum DMFS and DMFT was recorded amongst individuals with type II diabetes and in those consuming smokeless tobacco.

Discussion

Diabetes mellitus is a massive, growing, silent epidemic that has the potential to cripple health services in all parts of the world [15]. The prevalence of dental caries and its burden on the general population are of significant public health interest. Therefore, it is important to identify the patients who may be at a high risk of developing dental caries and associated oral diseases. The results of the present study revealed that caries prevalence (as measured by DMFT index) and caries severity (as measured by DMFS index) is significantly higher in tobacco consuming diabetic individuals as compared to tobacco consuming nondiabetic individuals. Previous studies have reported that type II diabetes is an inducing factor for caries development[16].

Furthermore, intragroup comparison revealed that smokeless tobacco users had significantly higher DMFT/DMFS index when compared to smoked tobacco users indicating that smokeless forms of tobacco is more detrimental when compared to smoked forms of tobacco in caries development.

Dental caries is caused by demineralization of tooth structure that is triggered by the accumulation of microbial plaque flora [17]. Saliva is essential for maintaining the oral equilibrium and the effects of saliva and its constituents on the oral microorganisms influences the development of dental caries. Salivary components (immunoglobulins, salivary protein, salivary calcium, and inorganic phosphorous and alkaline phosphatase levels) its flow rate, viscosity, buffering capacity, pH plays a major role in protection against initiation and progression of dental caries [18,19]. A decrease in the flow rate of saliva causes reduction in the cleansing, and buffering capacity leading to diminished levels of calcium that is essential for the repair of decayed tooth [20]. Low salivary pH promotes the growth of acidogenic bacteria which then allows the acidogenic bacteria to proliferate creating an inhospitable environment for the protective microflora. This allows for a shift in the oral environmental balance to favor cariogenic bacteria, which further lowers the salivary pH and thus the cycle continues [21].

Elevated salivary glucose levels associated with DM, may favor the growth of *Streptococcus mutans* and *Lactobacilli*. Xerostomia, another feature associated with DM, responsible for the low buffering capacity of saliva and this may interfere with the remineralization of early carious lesions. These factors also lower the activity of neutrophils which accelerates the microbial accumulation and thus maximizes the risk of tooth decay among diabetics. [22]

Intake of small amounts of carbohydrate rich foods consumed by diabetic patients may prove to be supportive of dental caries when coupled with elevated blood glucose levels. All this have a very negative impact on sympathetic and parasympathetic nervous systems leading to micro-angiopathy. This also causes dehydration and hormonal changes that are responsible for the alteration in the salivary flow rate [23,24]. The results of the present study are in accordance with previous studies conducted by Iqbal Singh et al. [16], Maria Moin *et al.* [25], and Malicka *et al.* [26], who reported a high prevalence of dental caries among diabetic patients. However, the results obtained in the present study were in contrast to previous studies conducted by Qureshi *et al* [27] and others who reported that there is no consistent pattern regarding the relationship between dental caries and diabetes [28].

Sri Kenneth et al.[29] reported that decreased salivary pH and an increased incidence of dental caries was observed in participants with uncontrolled diabetes as compared to the normal individuals.

Mechanistic links suggest that hyperglycaemia often results in altered cellular immunity, proliferation of bacteria, and formation of advanced glycation end-products (AGEs). Altered cellular immunity results in dysfunction of cells, inflammation and degradation of supporting connective tissue. These end-products of bacterial metabolism stimulate endothelial receptors and perpetuate a series of inflammatory events by attracting monocytes which ultimately leads to degradation of the attachment apparatus which promotes root surface caries [30].

Excessive use of smokeless tobacco has been reported to cause degenerative changes in more than 40% of minor salivary glands which are located at the site of chronic tobacco placement [31].

On an average, a wad of tobacco is kept in the oral cavity for 30 min, and hence, this prolonged duration of chewing tobacco also creates an environment conducive to caries activity [32]. The greater risk of caries development with smokeless tobacco may be attributed to prolonged exposure to sugars added to neutralize the bitter taste of tobacco, the levels of which may be from form-to-form, store-to-store, brand-to-brand, and state-to-state[33]. Hellqvist et al. [34] in their study demonstrated that the nicotine-containing tobacco products contained traces of glucose, fructose, sucrose (0.5-1%) and starch (approximately 1.5%).

Major biological effects of smokeless tobacco related to dental caries include:

- High levels of fermentable sugar and sweeteners (4-13% wt.) in smokeless tobacco which stimulate growth of cariogenic bacteria.
- Extracts of smokeless tobacco which may serve as a growth substrate for microbes which are frequently associated with human dental caries i.e., *Streptococcus mutans*, *Streptococcus salivarius* and *Streptococcus sanguis* [35].

Huang et al.[36] in an in vitro study demonstrated that nicotine enhanced the biofilm formation and biofilm metabolism of *Streptococcus mutans*, *Streptococcus sanguis*, and *Lactobacillus* and therefore nicotine may be one of the contributors of caries development. In addition, Nicotine causes reduction in salivary flow, thus promoting caries.

Another factor for which makes the teeth more susceptible to dental caries is the exposure of the more vulnerable root surfaces due to gingival recession associated with tobacco chewing[37,38]. The results of the present study are in accordance to those conducted by Bloom et al (2012) [39], Holmen et al (2013) [40] and Lashkari et al (2016) [41]

which discarded the older perspective of tobacco having a caries protective influence [42].

The caries prevalence in smoked tobacco users may be ascribed to sugars which are also used as cigarette additives to serve as flavor and humectants. Effect of nicotine on gustatory reflex appears to be the initial stimulation followed by depression and the long-term use of tobacco also decreases the sensitivity of taste receptors resulting in depressed salivary reflex [43]. Consequentially, there is altered taste response and decreased salivary flow in smokers [44]. Salivary buffering capacity in smokers is also found to be approximately 20% lower than in nonsmokers leading to acidic pH [45]. Lactobacilli colony count was found to be significantly higher in smokers as compared to nonsmokers in a study conducted by Al-Weheb et al [46]. This may be attributed to the fact that tobacco smoking depresses the immunoglobulins in oral cavity (IgM and IgA) leading to increase in acidogenic bacteria [47,48,49]. However, a limitation of the present study is that the severity of any associated periodontal disease was not documented. Therefore, a causal association between smoking and tooth loss could not be established which may influence the DMFT index. Caries is also influenced by confounding factors such as the socioeconomic status, oral hygiene, and malocclusion in participants, all of which were not taken into account. Tobacco use was self-reported in this study which may not be very reliable. These shortcomings need to be overcome in the future research.

According to the literature research, even with these limitations, this is the first study ever conducted which associated caries prevalence and severity with tobacco users in diabetic and non-diabetic patients. Hence, this study can be used as a pilot study for further research.

Conclusion

Within the limitations of this study, it may be concluded that, Diabetes is a risk factor for oral health complications. The findings of this study revealed that tobacco users with type II diabetes mellitus exhibited significantly more dental caries, and use of smokeless forms of tobacco is more detrimental than smoked forms of tobacco in caries development.

No previous study has been conducted comparing the prevalence of dental caries in diabetic and non diabetic individuals using tobacco in smokeless and smoked tobacco form, thus emphasizing the importance of present study.

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