ISSN: 2581-5989
PubMed - National Library of Medicine - ID: 101738774

International Journal of Dental Science and Innovative Research (IJDSIR)

IJDSIR : Dental Publication Service

Available Online at: www.ijdsir.com

Volume – 4, Issue – 5, September - 2021, Page No. : 87 - 95

Prevalence of periodontal disease among tobacco user

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Citation of this Article: Aarti Charokar, Dr Abhishek Gupta, Dr Vikram Singh, "Prevalence of periodontal disease among tobacco user", IJDSIR- September - 2021, Vol. – 4, Issue - 5, P. No. 87 – 95.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

A large body of epidemiological research indicates that tobacco use has deleterious effects producing a wide spectrum of diseases including cancers of the mouth, lung, larynx, pharynx; diseases of the heart, circulatory system and lungs; and if used during pregnancy, produces adverse effects on the fetus. There is clear scientific evidence present which state that nicotine is a causative agent for the development of periodontal disease and is detrimental to healing after periodontal therapy. The present study showed that high Percentage of bacteria present in smokers comparison to Tobacco Chewers and Non Tobacco user. The study conform that smokers have stronger chronic dose dependent effect on peridontium in sever periodontal disease. Tobacco smoking was noted as very potent environmental risk factor in periodontitis.

Keywords: Tobacco Smoking, Chronic, Mouth, Lung, Larynx

Introduction

Many types of deposit exist on the tooth surface above and below the gingival margin. The term plaque is used commonly to describe the bacteria associated with tooth surface and calculus is the attached dental plaque that has undergone mineralization. The combination of bacterial action and the roughness of the resulting calculus injuries the surrounding gum tissues and makes it at risk to infection and recession. The irritation causes swelling, inflammation and bleeding into the cervices between the teeth and gums, which is one of the early signs of impaired tissue health. The inflammation of the gums known as gingivitis can spread to the roots of the teeth if not treated. The gums separate from the teeth, forming pockets that fill up with more food particles and colonies of bacteria. As the disease progress, the bone support for the teeth is weakened and the affected teeth begin to loosen and drift from their normal position. Finally unless the disease is treated in time, the teeth may be lost.[1]

A large body of epidemiological research indicates that tobacco use has deleterious effects producing a wide spectrum of diseases including cancers of the mouth, lung, larynx, pharynx; diseases of the heart, circulatory system and lungs; and if used during pregnancy, produces adverse effects on the fetus. There is clear scientific evidence present which state that nicotine is a causative agent for the development of periodontal disease and is detrimental to healing after periodontal therapy. Nicotine has toxic effects on peripheral circulation, which causes gingival vasoconstriction. As a result a decreased number of immune cells are available in the gingival tissue, which translates into a weakened defence-reparative system.[1] Study also have reported that smokers strongly interconnected with poor oral hygine and in accumulation of plaque and calculus leading to periodontal disease Comparision to nonsmokers.[2] A strong association has been shown between periodontal disease and coronary Heart disease as well as between periodontal disease and cerebrovascular diseases (stroke). The subgingival micro flora and the continuous latent bacteremia (presence of viable bacteria in the circulating blood) and end toxemia (bacterial toxin in the blood) originated from the periodontal pockets might be responsible for the damage of the vascular endothelial integrity, platelet functions and blood coagulation.[3] Though studies in the last two decades have shown a positive correlation between tobacco use and periodontal diseases, public awareness regarding the severity of periodontal disease and its complications is very minimal. As stated above this can be a warning sign for other systemic diseases and can also act as a 'foci' for infections. Tobacco products can alter normal host responses to neutralize infections and can also stimulate pathologic micro-organisms to destroy surrounding tissues.[3]

Study also shows and strongly believes that smoking leads prevalence and severity of periodontitis and greater collection of plaque and calculus compare to nonsmokers However, with the better understanding of the host response, facts suggests that the effect of smoking on periodontal status is independent from the plaque index and oral hygiene of individual. So, this clearly suggests that smoking has a direct influence on periodontal tissues. Smokers have been associated with deeper pockets and greater attachment loss, more pronounced radiographic evidence of furcation involvement, and increased alveolar bone loss.4 there is an established biologic rationale for the negative effect of smoking on periodontal tissues. It has an immunosuppressive effect on the host, adversely affecting host-bacterial interactions, and this alteration may be due to changes in the composition of subgingival plaque. Smoking may also provide a favourable surroundings for some of the periodontopathic species in the plaque and may be one reason why smoking is a risk factor in periodontal disease development.[5, 6]

This study designed to the clinical, microbiological changes in Tobacco user and compared these to the Non Tobacco user.

Materials and Method

The study is based on data obtained from a series of subgingival microbial samples collected at the Department of Periodontology and Processed at the Department of Microbiology of a RKDF Dental College and Research Centre under SRK University Bhopal. Four hundred

Patients aged 18-55 years with periodontitis were enrolled in the present study. Study consists of Tobacco Chewers, Tobacco smokers and Non Tobacco User.

Inclusion criteria

- Subject should be tobacco user for more than 3 years.
- Subject should have 22 natural teeth in situ.
- At least 6 pockets with PPD of > 5mm

- No professional periodontal therapy during last six month.
- Periodontal disease is confirmed by radiologically diagnosis.
- Detail case history and informed consent will be taken from each subject.

Exclusion criteria

- Subjects under antimicrobial therapy
- Subjects using local medication.
- Undergone periodontal therapy in last 6 months.
- Subjects using partial denture.
- Medically compromised disease.

Materials and Method

- Study comprise of 400 subjects age group ranging between 18-55 years were selected.
- Subjects are divided in to three groups
- Group I consist of Male & Female
- Group II consist of tobacco user
  Chewers, Smoker, Chewers and Smokers
- Group III consist of Non tobacco user

Culture Media

VMGA III transport media

Blood Agar with Hemin and Vitamin K

Phosphate buffered saline

Method

- Subjects coming to OPD at RKDF Dental College
- Subject is examined by dental specialist
- A radio logically confirmation is done to confirm the periodontal disease.
- Clinical Parameters were also recorded including Pocket depth, Plaque index, bleeding on probing by specialist.
- Once confirmation is done subjects is consider for the sample collection
- Inform consent and detail history is taken

Sample Collection

- The deepest (>.5mm) six pockets were selected for sampling.
- The sterile paper point is inserted into the periodontal pocket for 20 seconds.
- Sterile paper points were transfered to a test tube containing 1 ml of the VMGAIII transport medium under anaerobic conditions.
- All samples were processed in $\leq$ 24 hrs at room tem. & incubated in anaerobic culture system (anaerobic jar).

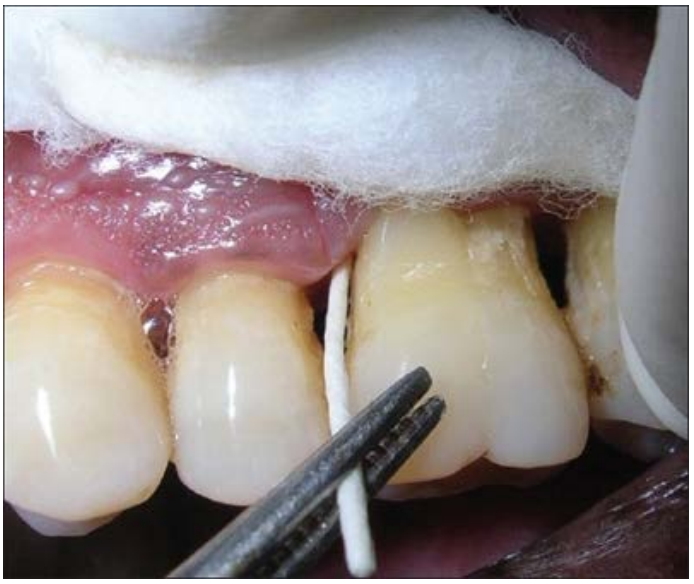


Figure 1

Sample Collection

Microbiological procedure

- All procedure done under LAF.
- Collect sample were vortex for 2 minute and 100 microlitre was used to prepare 10 fold dilution (10^4) in sterile phosphate buffer saline.
- 100 crolitre (sample) from dilution was taken by micropipette and cultured by spreading evenly on Blood Agar plate and incubated anaerobically using anaerobic jar with gas generating system at 37 C for 7 days.

- After incubation we count total viable bacteria or count colony forming unit (CFU) using Colony counter.
- Total viable counts (TVC) were defined as the total number of colony forming units obtained on non selective media plates.

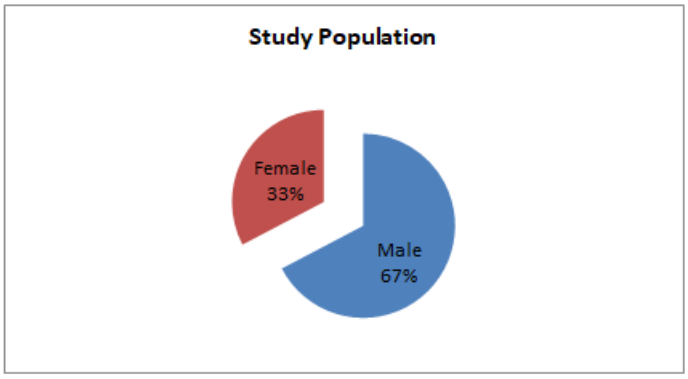
Result

As Study comprised of 400 subjects age group ranging between 18-55 years were selected, patients coming to RKDF Dental College & Research Centre, Bhopal.

Group I

S. No	Male	Female
1	276	134

In study 276 male and 134 female were selected.



Graph 1

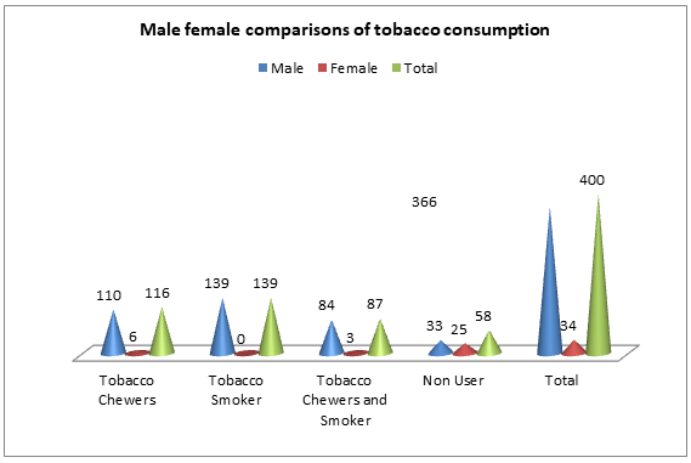
Group II & III

Tobacco Use	**Total**	**Percentage**
Tobacco Chewers	116	29
Tobacco Smoker	139	34.75
Tobacco Chewers and Smoker	87	21
Non User	58	14.5

The table shows the distribution of subjects consuming tobacco in various forms and in nonusers. As mentioned above, 34.75 % of the subjects were smokers while 29% were chewers and 21% were both chewer and smoker. Thus more than 80% of the subjects consume tobacco in any of the forms as mentioned above. When compared to tobacco users, 14.5% of the total sample population abstained from consuming any forms of tobacco.(non user)

Male female comparisons of tobacco consumption

Tobacco Consumption	Male	Female	Total
Tobacco Chewers	110	06	116
Tobacco Smoker	139	00	139
Tobacco Chewers and Smoker	84	03	87
Non User	33	25	58
Total	366	34	400

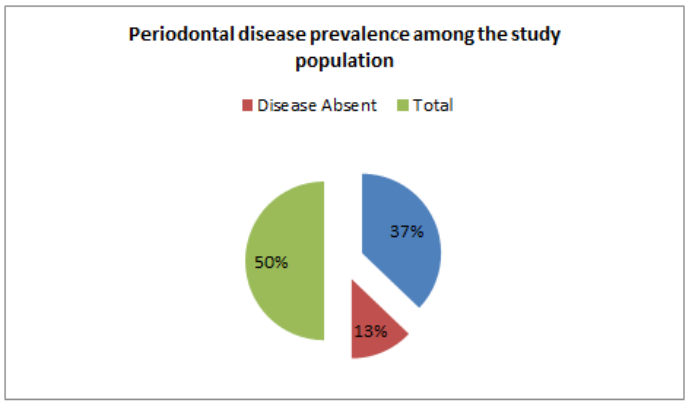


Graph 2

The table shows that, the using of both forms of tobacco and smoking alone were predominantly common among males while in the chewer category, females were less in both the form when compared to males. In the non-user category, of the respective sample population for each sex (366 & 34 respectively for males and females) 8.3% of males and 6.25% of females were not consuming any forms of tobacco.

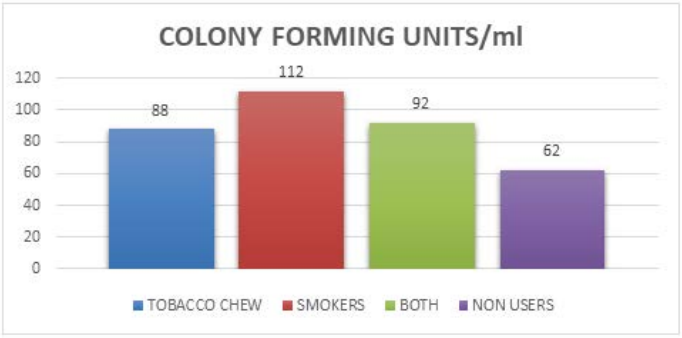
Periodontal disease prevalence among the study population

High Prevalence	297	74.25%
Low prevalence	103	25.75%
Total	400	100%



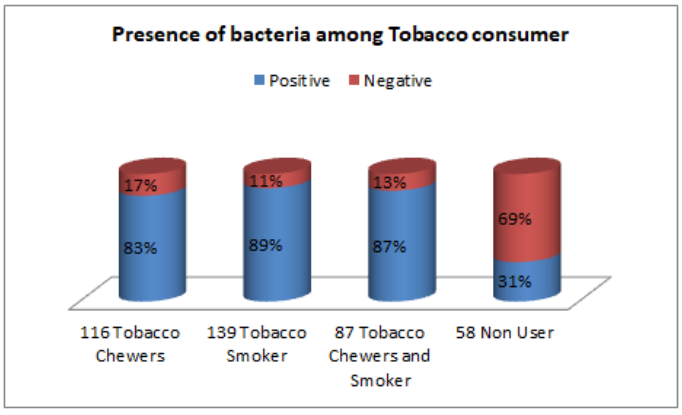
Graph 3

Presence of bacteria among Tobacco consumer



Graph 4

Tobacco Consumption	Prevalence
116 Tobacco Chewers	83%
139 Tobacco Smoker	89%
87 Tobacco Chewers and Smoker	87%
58 Non User	31%



Graph 5

The above table shows presence of bacteria among the tobacco user. Tobacco chewers show 83% of bacteria whereas 89% of bacteria were present in smokers and 87% of bacteria shows in both chewers and smokers. Among the Non user shows 31% of bacteria.

Discussion

The bacterial etiology of periodontal disease is complex, with a variety of organisms responsible for the initiation and progression of disease. Although over 400 different bacterial species have been detected in the oral cavity[14], only a limited number have been implicated as periodontal pathogens. Many of these organisms may also be present in periodontally healthy individuals and can exist in commensal harmony with the host.

The oral cavity is characterized by harbouring indigenous microbiota. The ability of microorganism to colonize the different oral surfaces depends on their binding potential. Various environmental factors and host factors are involved in the harbouring of microorganisms and microbial composition. Many indigenous microbes are anaerobes and these microorganisms can be associated with oral infections and be the origin of distant infection.[13, 15, 16, 22]

The major etiological factor for periodontal disease is the microbial dental plaque. But not only microbial plaque responsible for disease there are several risk factor. One of them is tobacco. Tobacco smoking plays a significant role of their severity and prognosis.

Detrimental effects of smoking on the occurrence and severity of periodontal disease are well documented on the basis of traditional method used in dentistry. However there are limited studies about microbiology finding on tobacco user and non tobacco user with periodontal disease.

In dentistry by traditional procedure such as probing depth and radiographic assessment provide information of what already occurred, but not for what is occurring and what will occur with disease progression and important why it

is occur. So, the present study was conducted to comparatively evaluate microbiological finding in tobacco user (Smokers and Chewers) and non tobacco user with periodontal disease. The numerous bacteria releases the bacterial product in periodontal pocket which contains histolytic enzymes, endotoxin , exotoxin and non toxic fluid that interfere with normal cell function. The collegenase and other enzyme released by bacteria leads to periodontal pocket formation and feature of periodontitis,extensive destruction of collegenase and connective tissue,(Kuramitsu, 1998).[8] Can also lead to bone loss.(Miyata et al., 1997).[17] Sulphides which are released by oral bacteria are low molecular weight metabolites are considered to be cytotoxic molecules in periodontium. (Socransky, 1990).[18] Some of the bacteria can inactivate precise antibody that is able to save you them their own demise by using phagocytosis .A .actinomycetemcomitans releases a leucotoxin that in particular break human leucocytes.

(McArthur et al., 1981).[19] Some of the bacteria can prevent the normal immune defence system of the subject. The bacterium releases toxins which are pathogenic because of its capability to initiate responsive mechanism that destroy periodontal tissue. Bacterial substances can destroy directly or indirectly periodontal tissue, it is difficult to differentiate good bacteria from bad bacteria because one bacterial may act both ways reparative and destructive in humans.

Smoking has important effects on oral bacteria. Although smokers have more plaque than non-smokers, there is no evidence to suggest that smoking increases the rate at which plaque develops. Cigarette smoking could cause a lowering of the oxidation-reduction potential (Eh), and this could cause an increase in number of bacteria in plaque.

Haffajee and Socransky[9] have found that there is a strong correlation between smoking and subgingival bacteria using DNA hybridization checker board. In their study they concluded that prevalence of species in smokers and non smokers was major difference rather than count and proportion. The colonization was more seen among smokers was more seen in shallow pocket, ie less than 4 mm. They also reported that more percentage of colonization was found more in maxillary than mandibular sites. They counseled that periodontal destruction is visible in extra severity in smoker which is because of expanded colonization with the aid of pathogens at extra number of places which leads to boom the chance of similarly breakdown is a combination of neighborhood outcomes from smoking environment for pathogens and deleterious effect at the host reaction is crucial element. Kamma and colleague [10] investigated smoking with periodontitis in topics and there outcomes.

The effective physico-chemical decreasing pastime of carbon monoxide might be an instantaneous mechanism for promoting the increase of anaerobes at superficial web sites as opposed to simple anaerobiosis[11]. A lower oxygen anxiety inside the periodontal pocket of people who smoke may be beneficial for the increase of anaerobic bacteria[12].

Studies have suggested that smokers exhibit increased bleeding upon probing, higher calculus and plaque deposits, increased clinical attachment loss, gingival recession and tooth mobility independent of age, gender and systemic condition.[39] A report from Calsina et al appreciated that those who've been smoking for 10 years or extra have seen super effect in male sufferers and pronounced that the chance of ailment increased to a few 3.7%.

The present study was carried to assess the amount of destruction cause by the any form of tobacco to the

individual. As result clearly indicate that the amount of destruction caused by the smoking is much higher than in the form of tobacco chewing.

Michiya Kubota et al[20] conducted a study to assess the effect of smoking on periodontal disease have find out that smoking causes the deep destruction on oral condition where the my study also concluded the same as.

Kamma JJ, Nakou M et al[21] conducted a study to find out microbial characteristics of smokers with early onset Periodontitis in which they have concluded that the smoker destruction is more and the microorganism are deep seated in periodontal tissue. The present study also completely agree with Kamma study, have seen more amount of microorganism in smoker.

Darby IB, Hodge PJ et al[22] conducted study to find out patients with smokers and non smokers with early onset of periodontitis by microbial comparision by PCR in which they have concluded that the microorganism are more deep seated in smokers as comparative to non-smoker which clear agree with my study where the result strongly agree with this study.

Bergstrom J et al[23] conducted a study to find out the Cigarette smoking as risk factor in chronic periodontal disease, where they have concluded that smoking is a major risk factor for periodontal disease and oral disease where my study also strongly comes with conclusion that smokers at higher risk of oral health disease.

Takeuchi Y, Umeda M et al[24] and present study have common agree that presence of periodontopathic bacteria is more in smokers in comparison of non-smoker and tobacco chewers.

Preber H, Bergstrom J, Linder LE et al[25] have find in their study that Occurrence of periopathogens in smoker is more as compare to non-smoker patients, which come to my conclusion of study where the periopathogens are more in periodontal depth.

Hamdoon SM et al[26] and present study used Anaerobic Culture method to identification of Periodontal microbes and provided information about the susceptibility of the individuals to develop periodontal disease on bone.

Conclusion

The present study showed that high Percentage of bacteria present in smokers comparison to Tobacco Chewers and Non Tobacco user. The study also conform that smokers have more strong chronic dose dependent effect on peridontium in sever periodontal disease.. Tobacco smoking, was noted as very potent environmental risk factor in periodontitis. Tobacco smoking not only affects the oral environment but the gingiva and the blood supply in inflammatory response and immune response and the healing potential of periodontal connective tissue. There is lots of clinical evidence is present which shows there is a smoking with destructive periodontal disease is associated. It was also conform that smoking will suppress gingival angiogenesis, the mechanism by which smoking moisten.

In numerous research specific study become the statement that people who smoke do not reply to mechanical remedy as well as non- people who smoke and that is related to extended ranges of T. for synthesis, A. Actinomycetemcomitans and P. gingival is continue to be within the wallet after therapy within the smoking institution whilst in comparison with non people who smoke.

The smoking suggestion programme should be implemented in smokers to prevent periodontitis and smoking prevention should be done by health education by advising and advocating and facilitating smoking suggestion programmes among the patient.

With present study the result clearly indicates that Tobacco users in the form of smoke are destroying patient oral health as well as personal health. So there need to be

carried out to be further study to find out degree of destruction cause by different bacteria**.**

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