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INFLUENCE OF TOBACCO SMOKING ON DENTAL PERIAPICAL CONDITION AMONG ADULT POPULATION- A RADIOGRAPHIC STUDY

[1]Madhumithaa S, [2*]Dr. Sangavi.R and [3]Dr. Adimulapu Hima Sandeep

[1]Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai - 600077

[2]Senior Lecturer, Department of Oral Medicine, Radiology and Special care Dentistry Saveetha Dental College & Hospitals, Saveetha Institute of Medical and technical Science, Saveetha University, Chennai - 600077

[3]Associate Professor, Department of Conservative and Endodontics, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha university, Chennai – 600077, Tamil Nadu, India

151701061.sdc@saveetha.com[1], sangavir.sdc@saveetha.com[2] and himas.sdc@saveetha.com[3]

ABSTRACT

Little research has been done thus far on the association between smoking and apical periodontitis. The current study's goal was to determine whether tobacco use may be linked to periapical lesions' frequency or severity. 247 people in total, 81 current smokers, 63 past smokers, and 103 non-smokers participated in the study. The frequency of endodontic treatment was 58% overall, while the prevalence of apical periodontitis was 52%. The periapical state was expressed as the number and percentage of radiographically identifiable lesions as well as as a severity score. Smoking had no discernible effect on the prevalence of endodontic therapy or apical periodontitis. Controlling for age, the association between smoking and periapical lesions was not statistically significant. The mean number (percentage) of periapical lesions per person was 1.9 (6%) in current smokers, 1.5 (4%) in inform smokers, and 1.0 (3%) in non-smokers. The average degree of periapical damage across all teeth, or just teeth afflicted by periapical lesions, did not substantially differ between the smoking groups. It is determined that there is no evidence to support the notion that cigarette use causes apical periodontitis from the current observations.

Keywords: Periapical conditions, radiographic study, tobacco smoking, adult population

INTRODUCTION

Apical periodontitis (AP), along with tooth decay, is the most prevalent oral illness, according to epidemiological studies. Depending on the population, inflammatory lesions in the periapical area can occur anywhere between 26% and 80% of the time [1-4].

Apical periodontitis is linked to the side effects of tooth decay and its management. Because of the intricate relationship between the mechanism of chronic AP and the immune system's reaction, it is important to take into account both tooth-specific and non-tooth-specific risk factors for AP, such as general illnesses. The most common condition affecting the peri- apical periodontium is diabetes. One of its effects, which may have an impact on the health of periapical tissues, is the impairment of healing processes. When it comes to molecules, In addition to inhibiting regeneration processes and osteoblast activity, hyperglycaemia bone resorption [5]. Smoking is listed as one of the risk factors for developing AP in various studies [2,6]. Addiction to tobacco alters the body's humoral and cellular immune systems as well as the metabolism of bone and connective tissue [7]. The physiological equilibrium between catabolic and anabolic processes is disturbed by changes in tissue and vascular metabolism [8]. Smoking raises the chance of developing oral malignant tumours and making implant treatments ineffective. It decreases saliva's pH and buffering ability, which could make people more likely to develop cavities.

ISSN 2515-8260 Volume 9, Issue 7, 2022

The periodontal bone suffers damage from smoking. Numerous cross-sectional and longitudinal studies have shown that smokers have a higher rate of advanced chronic destructive periodontal disease than non smokers do in terms of marginal periodontal bone loss [7]. In addition, smokers are more likely than non-smokers to have vertical periodontal bone abnormalities, which are resorption craters surrounding the roots of teeth [11]. On the basis of recent research showing that smoking is a significant risk factor for chronic periodontal disease of the marginal periodontium, it was hypothesised that smoking might also have a negative impact on the apical periodontium of teeth with poor endodontics. It has been hypothesised that smoking makes periodontal tissues more vulnerable to or less resistant to local infection from plaque bacteria, despite the fact that the mechanisms of action of smoking as a damaging component in the pathogenesis of chronic periodontal disease are not fully characterised.

The clinical research of the relationship between smoking and pulpal infection with subsequent inflammatory reaction in terms of radiographically visible and measurably destructive changes of the periapical bone seems reasonable to use in order to clarify the plausibility of such a concept. It was thought that smoking would increase the extension of the process of periapical bone degradation and/or interfere with healing and repair events following endodontic treatment since apical periodontitis normally is a process in response to a local infection.

As a result, it would be reasonable to assume a comparable rise in the frequency and/or size of periapical lesions in smokers. Tobacco use and apical periodontitis have almost never been investigated together. However, a connection between smoking and apical periodontitis was identified in a recent epidemiological study addressing the issue of finding risk markers for the condition [12].

The purpose of this exploratory study was to look into any potential links between smoking and the prevalence or severity of periapical lesions under the presumption that smoking may have an impact on periapical periodontitis.

Studies of numerous populations have confirmed the harmful effects of tobacco smoke's constituents on the periodontium. Marginal periodontium is discussed in the bulk of articles [7,9,10]. However, there is insufficient data in the literature about the prevalence of AP in smokers.

MATERIALS AND METHODS

The records of patients who visited Saveetha Dental College and Hospital between February 1, 2022 and June 31, 2022 were analysed in this retrospective analysis. The institutional review board/ SDC/SIHEC/DIASDATA/0619-0320 provided ethical approval. Patients ranging in age from 18-50 years old were enrolled in the study. The study sample consisted of both male and female participants, the majority of whom were South Indians. A total of 159 patients aged 18-50 years who visited a university hospital were included in the study. The study included 159 patients who were identified with dental periapical conditions who use tobacco in the hospital database. The necessary information, such as age, gender, and restoration type, was recorded. Incomplete patient records were omitted from the study. Data was entered into Microsoft Excel and then exported to the statistical package for social science for Windows (SPSS), where it was analyzed statistically. The chi square test is used to compare groups.

ISSN 2515-8260 Volume 9, Issue 7, 2022

RESULTS

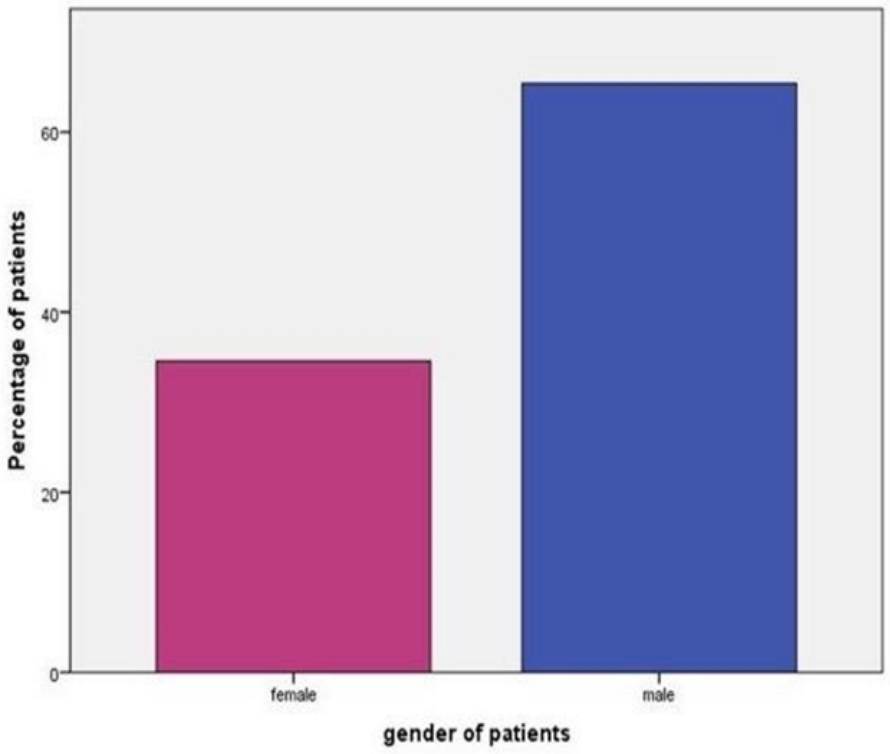

Figure 1: Bar chart shows gender distribution in the sample population. Pink denotes the female population and blue represents the male population.

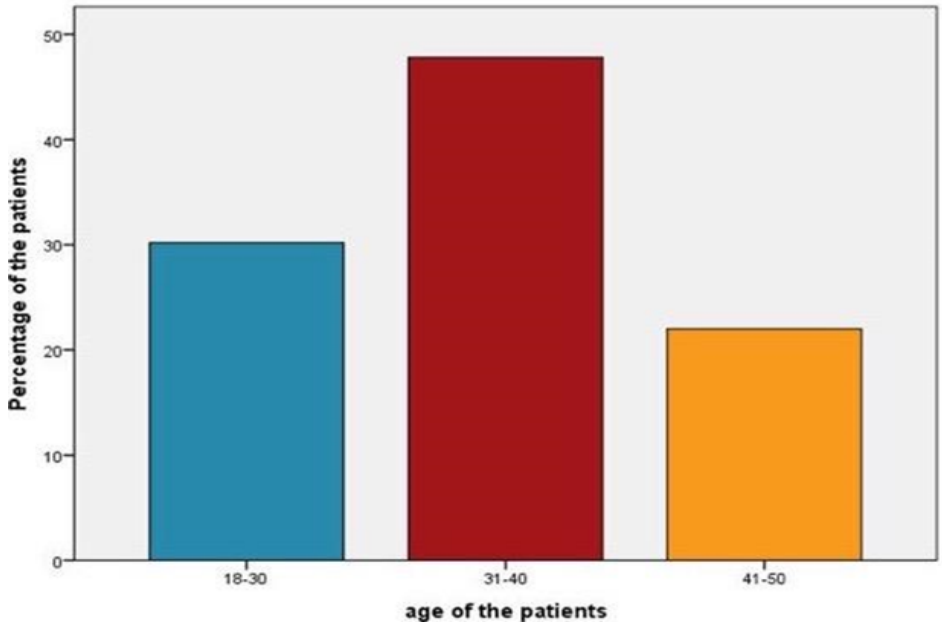

Figure 2: Bar chart shows the age distribution in the sample population. Blue denotes the population from age 18- 30, red represents the population from age 31- 40 and yellow represents the population from age 41- 50.

ISSN 2515-8260 Volume 9, Issue 7, 2022

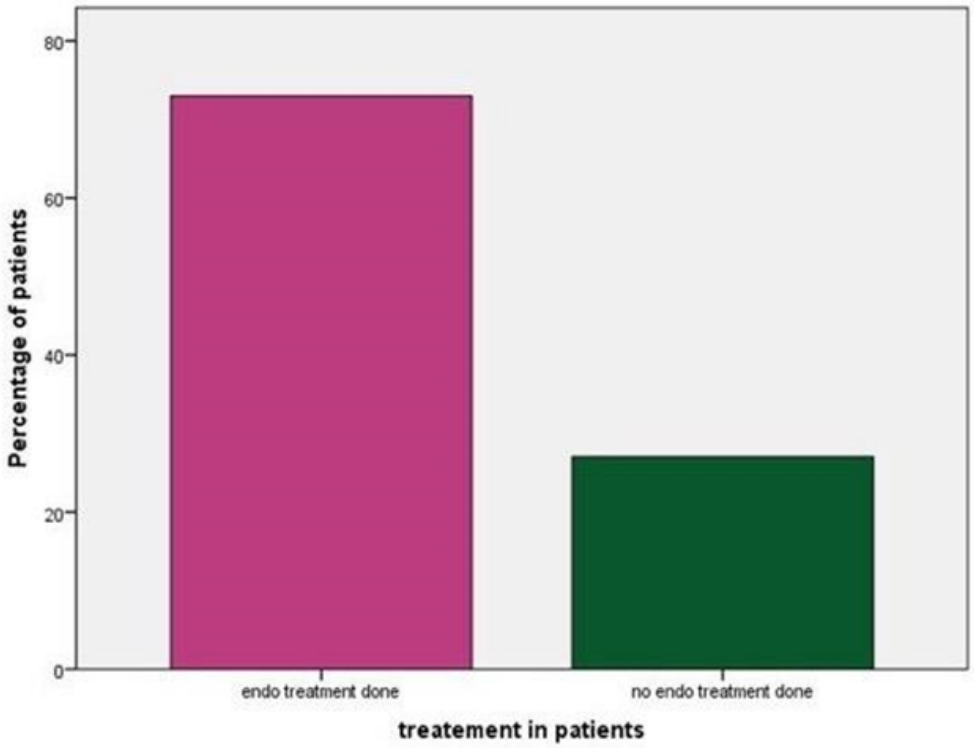

Figure 3: Bar chart shows treatment done in apical periodontitis patients in the sample population. Pink denotes the population where endodontic treatment is done and green represents the population where no endodontic treatment is done.

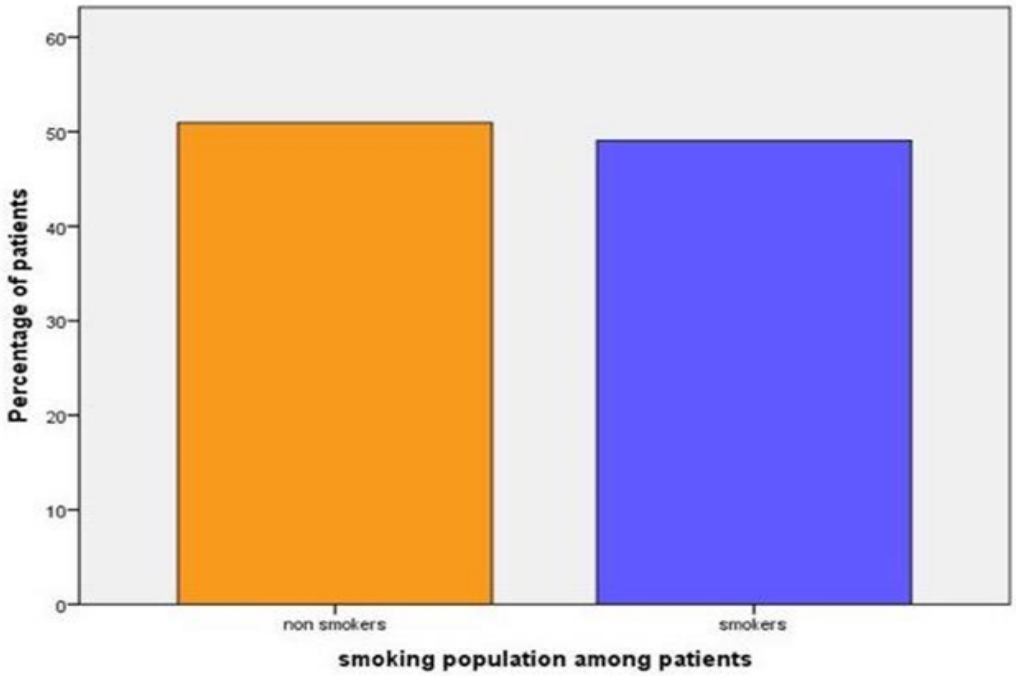

Figure 4: Bar chart shows smoking population in the sample population. Yellow denotes the population where they don't smoke and blue represents the population where they smoke.

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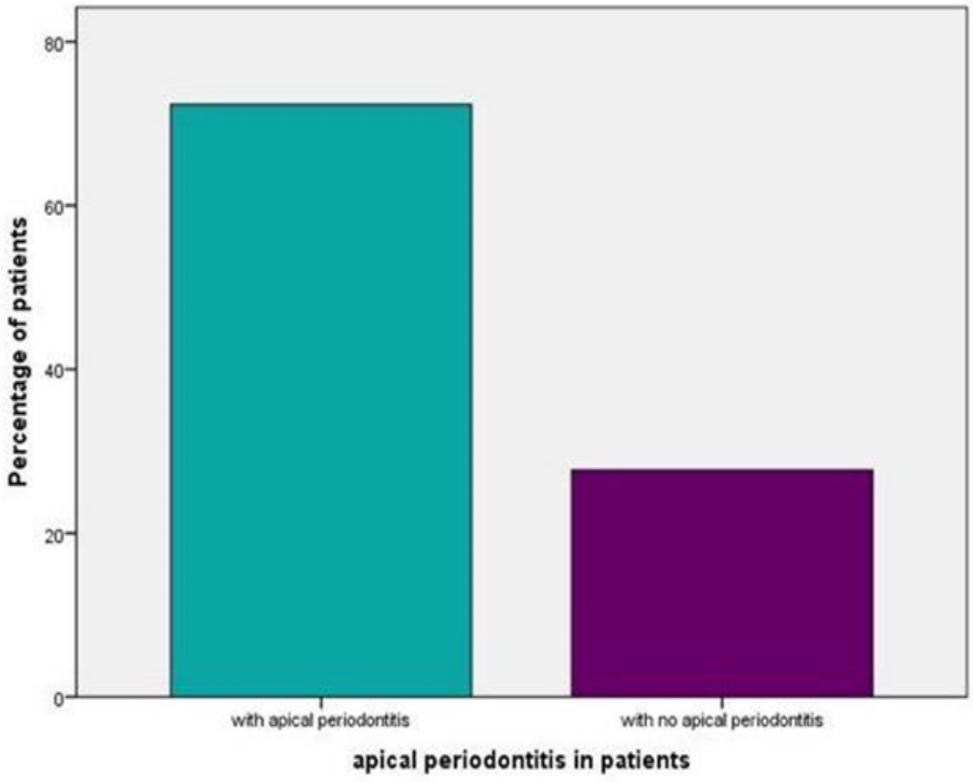

Figure 5: Bar shows patients with apical periodontitis in the sample population. Blue denotes the population with apical periodontitis and purple represents the population with no apical periodontitis.

The study sample consisted of 108 current smokers (42 %) and 151 never smokers (58 %). The mean age was 38.8 ± 13.6 years for current smokers and 41.9 ± 16.2 years for never smokers (P = 0.106). The average number of teeth per patient was 22.9 ± 5.2 and 23.2 ± 4.9 in smoking and never-smoked patients, respectively (P = 0.636).AP in one or more teeth was more often found in smok- ers (86.1%) than in never smokers (78.1%; P=0.143). One or more endodontically treated teeth were found in 89 (82.4%) and 119 (78.8%) of smoking and never- smoked subjects, respectively (P=0.576). Among smoking patients with endodontically treated teeth, 67 (72.0 %) had AP affecting at least one treated tooth. Among never smokers with endodontically treated teeth, 93 (78.8%) had AP affecting at least one treated tooth (P = 0.328) .In univariate analysis smokers had a higher frac- tion of teeth with AP than never smokers (0.13 vs. 0.10; P=0.025), whereas fractions of endodontically treated teeth and endodontically treated teeth with AP did not differ significantly

A current smoker was on average 16.4 times more likely to have AP than a never smoker (95 % CI : 5.7–47.7; P < 0.001), and if the person was a man, he was 3.1 times more likely to have AP than if the person was a woman (95% CI: 1.1–8.9; P=0.039;). The probability of AP increases with increase in age . Age, gender, smoking and fraction of endodontically treated teeth do not predict probability of AP on endodontically treated teeth .The number of teeth with AP increases with increase in age, and male gender and smoking are also related to increased number of teeth with AP. The difference in the number of teeth with AP is expected to be 2.2 units higher for smokers compared to nonsmokers, while holding the other variables constant in the model. The number of AP on endodontically treated teeth increases with increase in age and decreases with increase in fraction of endodontically treated teeth

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DISCUSSION

The study included adult patients attending dental service at the Dental Clinic of the Rijeka Clinical Hospi- tal Centre, Croatia for the first time. The recruitment of subjects, exclusion and inclusion criteria were similar to those used in previous studies [12, 16]. In the pres- ent study subjects with DM and former smokers were excluded. In several previous studies, a statistically sig- nificant association between DM and radiographically diagnosed chronic AP was found, suggesting that diabe- tes can act as a confounding factor [17, 18].(29)

A time period of 10–20 years was recently suggested for a resolution of oral tissues after heavy tobacco use [19]. The detrimental effect of tobacco is affected by the number of cigarettes the patient currently smokes, the intensity and duration of smoking and/or time since cessation [20]. This suggests that a broad categorization of tobacco use is inadequate and makes the identification of former smokers as a separate category necessary. Since data considering smoking cessation obtained by an interview were not reliable, former smokers were excluded.

The overall prevalence of AP in the present study was 82% which is comparable to similar studies conducted on patients referred to institutions, such as universities or specialist dental clinics [21, 22]. The major shortcoming of such study design is that the patients can be more severely diseased and therefore are not representative for the whole population. The prevalence of individuals with at least one endodontically treated tooth was 80 % which is comparable to 76% of participants in a recently con- ducted study in Croatia [5].(30)

The main purpose of the study was to investigate the possible influence of smoking on the prevalence of radiographic changes characteristic of AP. It was hypothesised that the periapical tissues of endodontically com- promised teeth would be negatively affected by smoking, through influence on repair and healing events follow- ing endodontic treatment or disruption of catabolic and anabolic mechanisms and facilitation of periapical bone destruction. Consequently, increased size and/or number of radiologically detectable lesions would be expected in smokers [10]. Furthermore, smokers have increased caries prevalence due to decreased saliva- buffering effect and a higher number of lactobacilli and Streptococcus mutans, poorer oral hygiene, different eat- ing habits (presumably consuming higher amount of and reduced frequency of dental recall than non- smokers [6–8, 23–25]. Taken together, the above points can be construed to contribute to the increased prevalence of dental caries and consequently the periapical disease in smokers. Although other studies found a higher prevalence of edentulism among smokers [26, 27], in the present study there were no significant differences between current smokers and never smokers considering the average number of teeth per patient. In the present study the prevalence of AP in current smokers was higher than in never smokers (86.1 vs. 78.1) although the difference was not statistically significant. However, a significantly higher fraction of teeth with AP was identified in cur- rently smoking patients. Considering data from cross-sectional studies, several studies revealed a slight, however, significant association with a reported OR between 1.4 and 4.4 between periapi- cal pathosis and cigarette smoking in populations studied [11, 12, 28]. A recent study, analysing data from hyperten- sive patients, reported a very strong positive association with an OR 16.8 (95 % CI: 4.6–61.3) [13]. Bergstrom et al. did not find significant association between smoking and periapical lesions (OR 0.7; 95 % CI: 0.5–1.1; P = 0.128) [10]. In the present study, on average, a smoker was 16.4 times more likely to have AP than a nonsmoker. A strong association between smoking habit and increased number of teeth with AP was also demonstrated and smokers will on average have two more teeth with AP than nonsmokers, while controlling for gender, age and overall number of teeth. The present findings suggest that smoking might facilitate the extension of the periapical bone destruction, making it possible to detect the higher number of the periapical lesions on the conventional radiographs.(31)

Age has previously been identified as risk indicator for AP [11]. This was confirmed in the present study, and basically each decade tends to increase the number of teeth with AP to three.

Gender appears to be related to AP, and present data demonstrated that men were more likely to have AP than women, and more teeth affected. This difference may reflect the greater interest of women in receiving dental care and attendance for check-ups.

Both endodontically treated teeth and endodontically treated teeth with AP appear to be present in similar numbers in current smokers and nonsmokers, and smoking does not seem to be related to AP in endodontically treated teeth. Although several studies reported a positive relationship between endodontically treated teeth and increased probability of AP [10–12], this was not confirmed by the present data. Moreover, when gender, age and smoking were controlled for, a higher fraction of endodontically treated teeth was related to decrease of AP in endodontically treated teeth. It may imply that quality of treatment is a significant predictor and that well-conducted endodontic treatment decreases AP, irrespective of smoking status or gender of patient.

This study has several limitations. It has been demon- strated by Brynolf et al. that apical inflammation is often present in the absence of radiological signs [29]. In the present study, only radiographs were used to assess the presence of AP, which might have resulted in underestimation of periapical pathosis. Similar to previous studies, smoking habit was treated as a dichotomous variable, whereas the influence of the intensity and duration of smoking habit was not investigated [11–13]. Although cross-sectional studies may demonstrate differences in the prevalence of AP, longitudinal studies may show differences between the smoking groups regarding the incidence, that is, the development of new periapical lesions within a time frame. The present study strongly supports the hypothesis that smoking influences the periapical status of teeth, but not endodontically treated teeth. However, since this study was cross-sectional by design, conclusions should be regarded as temporary until con- firmed by long-term observations. Our team has extensive knowledge and research experience that has translate into high quality publications(32-41)

CONCLUSION

Smokers are a group facing an increased risk of AP and, therefore, there is a need for an early detection and treatment of caries and its complications in this group of patients. The probability of AP increases with increase of age. Smokers will on average have two teeth with AP more than non-smokers, while controlling for gender, age and overall number of teeth. The multifactorial etiology of AP indicates the necessity to undertake further studies on the effect of smoking on the periapical status.

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CONFLICTS OF INTEREST

None declared Source of Funding : Self Ethical Clearance: It is taken from "Saveetha Institute Human Ethical Committee" (Ethical Approval Number- SDC/SIHEC/2020/DIASDATA/0619-0320)

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