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Original Research Article

Effect of smoking and dental fluorosis as environmental risk factors in periodontal disease — An observational study

Kharidhi Laxman Vandana[1,*], Vangara Aditya[2], Manchala Sesha Reddy[3], Prasad Sakthidharan Aswin[4]

[1]*Dept. of Periodontics, Former Associate Dean Academics, NAAC Editor, CODSJOD, College of Dental Sciences, Davangere, Karnataka, India*
[2]*Private Practitioner, Hyderabad, Telangana, India*
[3]*Dept. of Periodontics, Gulf Medical University College of Dentistry, United Arab Emirates*
[4]*Private Practitioner, Palakkad, Kerala, India*

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ABSTRACT

To assess the periodontal status of smokers and non-smokers in dental fluorosis subjects from endemic water fluoride areas of Davangere district, Karnataka. A stratified random sample study (n=338) was performed to obtain an equal number of subjects in tobacco and non-tobacco users. Participants had similar perceived oral health status (including self-reports of bleeding gums, loose teeth, and receding gums) low to medium socioeconomic status, and education. The sample for the present study on the tobacco effect consisted of 338 subjects aged 15-74 years. The periodontal status was assessed by clinical parameters Oral Hygiene Index — Simplified (OHI-S), Jackson's Fluorosis Index (JFI), Community Periodontal Index Treatment Needs (CPITN). Overall, periodontitis is significantly higher among smokers as compared to non-smokers. Although dental fluorosis is a risk factor compared in relation to age, sex was statistically not significant. OHI-S, JFI, and CPITN show statistical significance among smokers and tobacco chewers. The risk determinants such as age, sex followed by risk factors such as smoking and oral hygiene status for periodontal disease would make no difference from high to low fluoride areas but the current study concept on tobacco use in dental fluorosis subjects is thought-provoking and has to be considered in further studies.

1. Introduction

Epidemiological studies concerning the prevalence of periodontal disease in relation to dental fluorosis have given contradictory results. In general, a higher level of gingival inflammation has been observed in fluorosis than in non- fluorosis areas.[1–3] However, several studies have related no difference in periodontal conditions between endemic fluoride and non-fluoride areas,[4,5] and better gingival conditions are compared to non-fluoride areas have even been reported by others.[6,7] The effect of fluoride on the reduction of dental caries is well-established and its effect on periodontal tissues is obscure. However, two decades of institutional research projects on the effect of dental fluorosis by Vandana KL et al. 2014,[8] 2021[9] provides valuable information on hard and soft tissue changes caused by dental fluorosis.

It is well-known that tobacco usage plays a causative role in oral cancer and was fatal. Since then the risk-related health events have increased which leads to several other adverse health consequences. For a periodontist the knowledge about the adverse side effect of tobacco usage on periodontal health is important. Both cross-sectional and longitudinal studies have shown that smokers had more

* *Corresponding author*.
E-mail address: vanrajs@gmail.com (K. L. Vandana).

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periodontal tissue destruction and adverse effects on the immune system. There are hardly any studies revealing the periodontal status of tobacco users residing in natural high water fluoride areas. The combination effect of fluoride and tobacco use has never been explored earlier.

Hence, the first attempt has been made to assess the periodontal status of smokers and non-smokers in dental fluorosis subjects from endemic water fluoride areas of Davangere district, Karnataka India.

2. Materials and Methods

We examined tobacco effect on periodontal status of dental fluorosis subjects as part of a larger study investigating the influence of high water fluoride levels on periodontal status which consisted of 1029 subjects who lived in high levels of water fluoride areas The patients for this study were selected from the Department of Periodontics, College of Dental Sciences, Davangere District, Karnataka State. This study was conducted from January 2003 to March 2005 during which a total of 9890 subjects visited the Department of Periodontics. The sample for the present study on the tobacco effect consisted of 338 subjects aged 15-74 years.

A stratified random sample study (n=338) was performed to obtain an equal number of subjects in tobacco and non-tobacco users. Participants had similar perceived oral health status (including self-reports of bleeding gums, loose teeth, and receding gums) low to medium socioeconomic status, and education.

A tobacco user was a subject who was currently consuming tobacco once a day or more often in the form of smoke or smokeless tobacco (betel chewing with tobacco) and had done so during the past year at least, while a non-tobacco user was an individual who had never used tobacco at a frequency of even once a day in the form of smoke or smokeless tobacco. (Amarasena et al. 2002).[10]

The subjects were mutually exclusive smokers (males), betel chewers, and a combination of betel chewers and smokers. The latter two groups had both males and females. Inclusion criteria included subjects suffering from dental fluorosis who were tobacco users reported to the clinic either with bleeding gums and or painful gums and stained teeth. Tobacco users included Exclusion Criteria included subjects with non-systemic diseases and subjects with other intrinsic dental stains. The two groups of this study are tobacco users and non-tobacco users.

Recording of clinical parameters included Oral Hygiene Index — Simplified,[11] Jackson's Fluorosis Index,[12] Community Periodontal Index Treatment Needs.[13] The code of CPITN was interpreted for the purpose of periodontal status evaluation as, code 0- healthy periodontal status; code l as gingivitis, and code 3 and 4 as periodontitis. The treatment need was not considered. All examination was dispensed by one examiner with the help of both an apparent plain mouth mirror and a WHO 621 periodontal probe.[5] Fluoride concentration in water was within the range of 1.5-3.0 ppm, done by Chemical Department, Bapuji Institute of Engineering and Technology College.

The clinical data collected were subjected to statistical analysis wherein chi-square analysis was used for comparative data.

3. Results

A total of three hundred thirty-eight subjects in the age range of 15 to 75 years were included in this study. The results of this present study are presented in detail from Tables 1, 2, 3, 4, 5, 6 and 7. For statistical purposes, lesser and greater than 35 years were considered for the age criteria analysis Healthy periodontal status wasn't found in the smokers group. In both smokers and non- smokers, gingivitis was prevalent in <35 yrs and >35 yrs groups as compared to periodontitis. However, periodontitis was of higher percentage in >35 yrs and < 35 yrs age of smokers group than non-smokers group. The chi-Square analysis didn't provide significant results between age and periodontal status in both smokers and non-smokers groups. [Chi-Square analysis (X^2= 2.23, P=0.69)]. (Table 1)

While comparing the periodontal status between males and females in the tobacco users group, periodontitis was significantly higher (45.6%) in males than females (34.1%), unlike gingivitis which was seen more in females. In non-tobacco users, both females and males had a higher percentage of gingivitis than periodontitis. No significant results were obtained in relation to gender and periodontal status in tobacco users as shown by Chi-Square analysis (X^2=1.76, p=0.18, p>0.05). In non-tobacco users, the Chi-Square analysis demonstrated no significant results (X^2=5.95, p=0.051). Statistically significant results were not obtained between age and periodontal status in non-tobacco usage (34.1%) (Table 2).

In tobacco users, 26.6% of subjects had poor oral hygiene as compared to 24.2% of non-tobacco users. A maximum percent of fair oral hygiene was found in both tobacco users and non-tobacco users. In tobacco users, 51.1% of subjects with periodontitis had poor oral hygiene and 56.4% of subjects with gingivitis had fair oral hygiene. In non-tobacco users, 39% of periodontitis subjects had poor oral hygiene and 83.9% of gingivitis subjects had fair oral hygiene. Chi-Square analysis showed significant results between poor oral hygiene and periodontal status in tobacco users. (X^2=11.83, p<0.05).(Table 3)

The higher percentage of gingivitis (78%) and healthy periodontal status (20.7%) was found only in non-tobacco users. The tobacco users had a higher percentage of periodontitis (42.6%) as compared to non-tobacco users (20.7%). Chi-square analysis showed a significant correlation between tobacco users and periodontal status (X^2=20.1, DF=2), which was highly significant (P<0.01). Statistically significant results were obtained in relation to

Table 1: Distribution of sample based on Age and Periodontal status (in both tobacco users and non-tobacco users)

Group	Age	Healthy		Gingivitis		Periodontitis		Total		X^2	P
		Number	%	Number	%	Number	%	Number	%		
Tobacco Users	< 35	0		79	59.8	53	40.1	132	78.1	3.42	p = 0.18 Not Significant
	> 35	0		18	48.6	19	51.3	37	21.8		
	Total	0		97		72	42.6	169			
Non-Tobacco Users	< 35	2	1.5	105	79.5	28	21.2	132	78.1	2.23	p = 0.69 Not Significant
	> 35	0	0	27	72.9	10	27	37	21.8		
	Total	2	1.1	132	78.1	35	20.7	169			

Table 2: Distribution of sample based on sex and periodontal status in both tobacco users and non-tobacco users

Group	Gender	Healthy		Gingivitis		Periodontitis		Total		X^2	P
		Number	%	Number	%	Number	%	Number	%		
Tobacco Users	Male	0		68	54.4	57	45.6	125	73.7	1.76	P = 0.18 P > 0.05 Not Significant
	Female	0		29	65.9	15	34.1	44	26		
	Total	0		97	57.3	72	42.6	169			
Non-Tobacco Users	Male	1		98	78.4	27	21.6	125	73.9	5.95	p = 0.051 Not Significant
	Female	1		34	77.2	8	18.1	44	26		
	Total	2		132	78	35	20.7	169			

Table 3: Distribution of sample based on OHI — S and periodontal status in both tobacco users and non-tobacco users

Group	OHI - S	Healthy		Gingivitis		Periodontitis		Total		X^2	P
		Number	%	Number	%	Number	%	Number	%		
Tobacco Users	Good	0		14	87.5	2	12.5	16	9.4	7.50	P < 0.05 Significant
	Fair	0		61	56.4	47	43.5	108	63.9		
	Poor	0		22	48.8	23	51.1	45	26.6		
	Total	0		97	57.3	72	42.6	169			
Non-Tobacco Users	Good	1	2.4	34	82.9	6	14.6	41	24.2	11.83	P < 0.05 Significant
	Fair	1	1.1	73	83.9	13	14.9	87	51.4		
	Poor	0	0	25	60.9	16	39	41	24.2		
	Total	2	1.1	132	78.1	35	20.7	169			

Table 4: Distribution of sample based on periodontal status in tobacco and non-tobacco users

Group	No of Individuals		Healthy		Gingivitis		Periodontitis		X^2	P
	Number	%	Number	%	Number	%	Number	%		
Tobacco Users	169		0	0	97	57.4	72	42.6	20.1	P< 0.01 Significant
Non - Tobacco Users	169		2	1.2	132	78	35	20.7		

periodontal status and tobacco usage. (Table 4)

The maximum percentage of subjects who had degree D of Jackson's Fluorosis Index Gingivitis was found highest (40.2%) in degree D, unlike periodontitis which was more in degree C of tobacco usage group. Chi-Square analysis (X^2=12.6, p<0.05) presented the significant correlation between Jackson's Fluorosis Index and periodontal status in tobacco users. The maximum percent of subjects had degree D of Jackson's Fluorosis Index. Similar results for gingivitis and periodontitis were found in the non-tobacco usage group. However, the Chi-Square analysis (X^2=5.68, p=0.34) didn't present a significant correlation between Jackson's Fluorosis Index and periodontal status in non-tobacco users. (Table 5)

It was interesting to note that tobacco users have a strong association with periodontitis. (OR=2.91) (Table 6).

Based on the tobacco form used, gingivitis was found to be higher in smokers (63.3%) and the least was seen when both smoking & chewable forms were used. The occurrence of periodontitis was found to be higher when a combination of smoking and tobacco chewing was used (75%). Chi-Square analysis showed significant results

Table 5: Distribution of sample based on Jackson's Index and periodontal status (in both tobacco users and non-tobacco users)

Group	Jackson's Index	Healthy		Gingivitis		Periodontitis		Total		X^2	P
		Number	%	Number	%	Number	%	Number	%		
Tobacco Users	A	0		3	30	2	2.7	5	2.9	12.6	P < 0.05 Significant
	B	0		16	16.4	7	9.7	23	13.6		
	C	0		28	28.8	40	55.3	68	40.2		
	D	0		39	40.2	17	23.6	56	33.1		
	E	0		6	5.1	3	4.1	9	5.3		
	F	0		5	6.1	3	4.1	8	4.7		
	Total	0		97	57.3	72	42.6	169			
Non-Tobacco Users	A	0	0	16	12.1	3	8.5	19	11.2	5.68	P = 0.34 Not Significant
	B	0	0	11	8.3	5	14.2	16	9.4		
	C	2	3.7	39	29.5	13	37.1	54	37.8		
	D	0	0	55	41.6	9	25.7	64	37.8		
	E	0	0	9	6.8	3	8.5	12	7.1		
	F	0	0	2	1.5	2	5.7	4	2.3		
	Total	2	1.1	132	78	35	20.7	169			

Table 6: Risk of tobacco on periodontal status odds favoring periodontitis

	Periodontitis	Gingivitis	Total
Tobacco Users	72	97	169
Non-Tobacco Users	35	132	169

Table 7: Distribution of sample based on tobacco form used and periodontal status

Group		Healthy		Gingivitis		Periodontitis		Total		X^2	P
		Number	%	Number	%	Number	%	Number	%		
Tobacco Users	Tobacco Chewing	0		76	58	55	41.9	131	77.5	17.0	P < 0.01 HS
	Smoking	0		19	63.3	11	36.6	30	17.7	3.89	P=0.14 NS
	Both	0		2	25	6	75	8	4.7	12.7	P<0.01 Significant
Total		0		97	57.4	72	42.6	169			
Non-Tobacco Users	Tobacco Chewing	1	0.76	1.05	80	25	19	131	77.7		
	Smoking	1	3.3	22	73.3	7	23.3	30	17.7		
	Both	0	0	5	62.5	3	37.5	8	4.7		
Total		2	1.2	132	78	35	20.7	169			

in tobacco chewers (X^2=17, P<0.01) which was highly significant. Chi-Square analysis showed significant results when both forms of tobacco were used (X^2=12.7, p<0.01). (Table 7)

4. Discussion

Even though studies have been done on the periodontal status of high water fluoride areas prior to 1936 until 1993, there are inconsistent and no confirmative data established. Henceforth, these studies don't focus on the periodontal status of high water fluoride areas as research.

Apart from known local and systemic causes for periodontal disease, geographic or environmental factors such as smoking, endemic fluorosis and racial influence on certain forms of periodontal disease requires to be studied. The susceptibility of the population to the occurrence of periodontal disease is to be probed. To a certain extent, genetic constitutions of certain races answer partly the localization or increased prevalence of periodontal disease.

Overall, significant disparities exist in the prevalence of periodontitis by race or ethnicity, education, and poverty level. The prevalence of total and moderate periodontitis increased with increasing age among all adults it was observed that Severe disease was almost three times higher among men (12.5%) than women (4.2%).[14]

A strong association is observed between tobacco use/smoking habits and periodontal diseases in diverse populations. A direct causal relationship between smoking exposure and the prevalence and the severity of periodontal

disease has been firmly established recently (Chahal GS et al. 2017).[15]

The impact of smokeless style of tobacco use was significantly higher on all the periodontal health indicators like plaque index, gingival index, calculus, clinical attachment loss, gingival recession, mobility, furcation, lesion, and probing pocket depth (Singh GP et al. 2011).[16]

The essential features of epidemiology as a way of research, in comparison to clinical research and case studies, are that groups instead of individuals are the main target of study, and persons with and without the condition of interest are included in studies that aim to qualify risk. On the opposite hand, this study being clinical research, also study large groups of people, is confined to patients i.e., those with the disease of interest.[4]

This study is based on the CPITN index, as indicated on the CPITN profile in fluorosis subjects where gingivitis is prominent at all age groups, shallow pockets are observed at all age groups with low to moderate prevalence, and deep pockets observed on advancing age with low to moderate prevalence. Comparatively, high prevalence is usually associated with low to moderate pockets with an average of more than half of sextant per subject similar results are seen in gingivitis where high prevalence is usually associated with low to moderate pockets with an average of more than half of sextant per subject.

This contemporary study was in deliberate need to increase scientific knowledge among combined use of tobacco and fluorosis on periodontal status in high water fluoride areas. One of the interpretations to pop-up from a review on longitudinal studies was the 1996 world workshop in periodontitis and their interaction between environmental and subject-related factors doesn't have to be constant in geographically or racially different populations virtually all the studies hitherto that have recognized tobacco as a true risk factor for periodontitis have been carried out around the west and had focused on the habit of tobacco smoking.[17] This contemporary study sample was representative of a developing country by possibly low standards of oral hygiene, a more rapid progression of periodontal disease,[18] and uncanny mode of tobacco chewing. Additionally, the concluding articulation of periodontitis is established on the complex interaction occurring within an intricate mosaic of host, microbial and environmental factors,[19] it was felt that contribution of tobacco as a risk factor is present in high water fluoride areas might be a worthy investigation.

Subsequently, the periodontal status of exclusive smokers, betel chewers, and a combination of smokers and betel chewers was compared with that of non-tobacco users. The hierarchy of the sample is based on two major categories and the frequency of its matching, in order to overcome the confounding effects of age on periodontitis.[20] Indeed, there was an association between age and periodontal status, which is previously described[18,20–22] was not evident in the present analysis, the older age group (>35 yrs) as well the younger age group (>35 yrs) had higher percentile levels of periodontitis in the tobacco group than the non-tobacco group. In the present study, only a few subjects (2%) had healthy periodontal status in the < 35 yrs group of non-tobacco users. Gingivitis was seen in both the age groups of tobacco and non-tobacco users, similar to the observation by Murray JJ (1972)[12] who reported increased prevalence of gingivitis in high fluoride areas between the age group of 15-65 yrs. The presence of periodontitis was observed in both the age groups of tobacco and non-tobacco users. Haikel et al. (1989)[5] concluded from an epidemiologic study (n=2378) using CPITN index in a population aged 7 to 60 years that extensive gingivitis, low to moderate prevalence of shallow pockets increasing with age in high fluoride areas. However, Reddy J et al. (1985)[13] and Grembowski (1993)[23] reported little evidence of periodontal destruction in high fluoride areas.

Males were affected with a higher percentage of gingivitis and periodontitis in both tobacco and non-tobacco users group as compared to females. This finding is similar to the general trend of males getting affected with periodontitis than females. However, Amarasena et al. (2002)[10] included only male subjects in both tobacco and non-tobacco users.

In this study, higher levels of gingivitis were seen in non-tobacco users than tobacco users contrast to the findings of Amarasena et al. (2002).[10] However, higher levels of periodontitis were seen in tobacco users than non-tobacco users similar to Amarasena et al. (2002)[10] who estimated severity of periodontitis by loss of attachment (LA) reported greater in betel chewers (1.47mm±1.49) and smokers (1.39 mm± 1.44) as compared to non-tobacco users (0.79mm±1.04). In the present study, a higher prevalence of sextants with shallow to moderate pocketing was observed using the CPITN index. The association between tobacco use and gingivitis has been studied both epidemiologically and experimentally in the recent past. However, the results of these studies are conflicting. For example, some workers have observed less gingival bleeding in smokers as compared to non-smokers (Bergstrom & Floderus Myrhed 1983,[24] Preber & Bergstrom 1985,[25] 1986,[26] Bergstrom 2001[27]). The biological phenomenon involves tobacco smoke inducing vasoconstriction of gingival vasculature, thus affecting gingival blood flow. Thus, in turn it may suppress normal gingival inflammatory in response to plaque and also conceal actual levels of gingival inflammation in smokers (Clarke et al. 1981).[28] Some other have found that there was a marked elevation of gingival inflammatory response in smokers as compared with non-smokers (Arno et al. 1958,[29] Linden & Mullally 1994).[30] However, few studies show that there is no conspicuous difference in gingival status between smokers and non-smokers (Bastian & Waite

1978,[31] Bergstrom et al. 2000).[32] A few investigations state that the smokeless tobacco to periodontal relationship had significantly failed to demonstrate any change in the level of gingivitis or gingival bleeding between smokeless tobacco users and non-smokeless tobacco users (Wolfe & Carlos 1987,[33] Robertson et al. 1990).[34] Nevertheless, Amarasena et al. (2003)[35] found a positive association between tobacco betel chewing and gingivitis.

The oral hygiene status was fair in the majority of subjects in spite of the non-availability of regular dental care. In the present study, gingivitis was present at all levels of oral hygiene status and periodontitis was significantly increasing order from good to poor oral hygiene status in both tobacco and non-tobacco users. Surprisingly, gingivitis was variable in nature mostly declining order from good to poor oral hygiene status in both tobacco and non-tobacco users. Amarasena et al. (2002)[10] found a weakly correlated plaque-induced effect in the occurrence of gingivitis and periodontitis. Murray JJ (1972)[12] found that gingivitis is more prevalent in high fluoride areas despite of good oral hygiene. In this study they considered gingival recession (loss of attachment) and also took into account age, the age groups who are residing in low fluoride areas had less recession than members of the high fluoride group. Another study had shown that residents in high fluoride areas might be at a disadvantage in terms of their periodontal status. Oral hygiene (plaque levels) is an important risk factor in causing gingivitis and periodontitis. In the present study, poor oral hygiene status was significantly associated with periodontitis in both tobacco and non-tobacco user groups.

There was a strong association between tobacco users and periodontitis (OR=2.91). The effect of tobacco on periodontal status is well dealt with in dental studies but it is non-conclusive. However, the effect of fluoride intoxication on periodontal is not studied which requires to be paid attention. Many factors have made it difficult to compare the findings of this present study concerning the effect of tobacco use and periodontal status with other studies. The logistic regression analysis revealed a strong association between smokers and periodontitis; non-smokers and gingivitis. (OR = 2.91)

Firstly, most of the studies have included only cigarette smokers, whereas the present sample included only cigarette smokers, whereas the present sample included cigarette and beedi smokers as well betel chewers. Amarasena et al. (2002)[10] had conducted a similar study using tobacco users and non-tobacco users from non-fluoride areas unlike the present study was a comparative analysis from high fluoride areas. Consequently, there may be distinct differences in the quantity of tobacco used along with betel quid as well as the bioavailability of tobacco when consumed in this mode as against tobacco smoking. Moreover, betel chewers are known to use various ingredients such as betel leaf, areca nut, and lime with quid, additionally to tobacco while the periodontal effects of such additives don't seems to be known, it's plausible that hitherto unknown complex interactions between such substances and tobacco might result in a definite profile of periodontitis in betel chewers. Further studies are warranted to explore the periodontal effects of the additives that make up the betel quid alongside tobacco.

Secondly, tobacco periodontal correlations have been extensively studied. It's remarkable that our findings are confirmed as a significant association between the current tobacco users and periodontal pockets as shown by several workers in the west as Loss of Attachment (LOA).[36–38] Nonetheless, oral hygiene doesn't have a significant impact on the severity of periodontitis as proven by studies, in contrast to the present study, periodontitis increased as oral hygiene deteriorated. This observation gives importance to bacterial plaque in determining the role of plaque per se in the tobacco use periodontal disease relationship.

In other words, the effect of tobacco use may be limited and clinical significance of it is lacking as in comparison to oral hygiene disease progression. Although, evaluation may be over-simplified on the context of a chronic episodic multifactorial disease in which, according to the 1996 world workshop in periodontics., there is as yet 'considerable ambiguity in our understanding of the critical pathways or critical elements which are necessary to the pathogenesis[19] hence these estimations should be interpreted with caution. Whether this is because in this included subjects used betel quid chewing or because of the existence of the exceptionally high levels is speculative. What has come up is that, in Indians plaque status may be a key factor that determines the severity of periodontitis with the impact of other factors like tobacco being mediated and controlled by plaque levels controlled. However, in the present study, the effect of fluoride as a geographic or environmental factor needs to pay attention which acts independently of plaque levels.

The interesting part of the study was to correlate tobacco usage and periodontal status in dental fluorosis subjects hailing from high water fluoride areas. The plausible reason to include this category of subjects is based on the hypothesis that fluorides can also be a possible geographic or environmental risk factor for periodontal destruction. The effect of fluorosis on periodontal structures isn't studied in humans, which can be answerable for the destruction of periodontal tissues as reported in a very few of the medical literature. Though the role of dental plaque is well established, it doesn't explain the difference in susceptibility of a given population or individual periodontitis. Many factors have an influence on the etiology and pathogenesis of periodontal disease and one factor among them is fluoride. Krook L (1983)[39] dental fluorosis in cattle shows hypercementosis, necrotic cementum with cyst formation, osteonecrosis, gingival recession, alveolar crest recession.

Vazirani (1968)[40] reported root resorption of the mottled tooth with severe irregular, rough and heavy deposits on the root surface. Radiographically, these affected is presented as osteosclerosis, cementosis, and periapical root resorption. Reddy BD et al. (1969)[41] reported calcification of muscular attachment, ligaments, and ossification histopathologically that interferes with the functional movement of the skeletal system.

Hence, the combination of definite plaque-induced effect (inflammatory) and fluoride toxic effects (non-inflammatory) possibly lead to exaggerated periodontal destruction in dental fluorosis subjects requires to be researched. Therefore, a first attempt is made from this cross-sectional data, to correlate periodontal status in tobacco and non-tobacco users from high water fluoride areas (subjects with dental fluorosis). According to the results of this study, a higher percentage of gingivitis and periodontitis were concentrated in degree C and D in both tobacco and non-tobacco user groups.

Given that this cross-sectional study, the attributes, which were significantly associated with LA, can be considered risk indicators for periodontitis.[4,37,38] The role of fluoride in periodontal destruction has been hypothesized through several studies is focused on this study. However, such risk indicators like fluoride are not always confirmed as risk factors in longitudinal studies. Accordingly, any inference on risk should be interpreted with caution, within the present study.

Two decades review reports on dental fluorosis-induced effects on periodontium by Vandana KL et al. 2014,[8] 2021)[9] extensively reports the SEM evaluated soft and hard tissue changes in the periodontium. Unfortunately, the global awareness of this concept is negligible including WHO and UNICEF. The systemic effects of fluorosis in terms of skeletal fluorosis are much discussed with least awareness about its effect on CNS, thyroids, kidneys, and various systems in the body.

Smoking as an environmental risk factor for periodontal disease is presented in the literature. The geographic endemic fluorosis in global science could play a vital environmental risk factor in causing periodontal disease. Researchers from those areas can wake up to comprehend the reports of those studies by Vandana KL et al. 2014,[8] 2021[9] projects and surveys, so as to plan for the prevention of dental fluorosis-induced periodontal diseases.

In conclusion, the findings confirm the significant effect of tobacco use (in all forms) on the occurrence and severity of periodontal disease as reported by several workers who have investigated the tobacco periodontal relationship in alternative cultures. Both oral hygiene and tobacco use are designated risk indicators through several studies. Nevertheless, the role of fluorides in periodontal destruction needs to be investigated.

5. Conclusion

Since there isn't much effort shown in the literature to know exactly whether there's an association of periodontal disease in high fluoride areas rather than pointing out oral hygiene and plaque levels. Therefore, the risk determinants such as age, sex followed by risk factors such as smoking and oral hygiene status for periodontal disease would make no difference from high to low fluoride areas. Though the regular pathway of gingivitis progressing to periodontitis is through an inflammatory process is well established. In addition, to inflammatory process which remains common for both low and high fluoride areas, is fluorosis. This fluorosis-induced change in hard and soft tissues of periodontium needs to be paid attention whether fluoride is an etiological (environmental) agent for periodontal disease.

Almost many studies comparing periodontal disease conditions among populations have been studied between high and low fluoride areas without any knowledge of how destructive fluoride is on the human periodontium. So much attention is needed in populations who are susceptible to periodontal disease along with an understanding of the etiological and pathogenesis of periodontitis in terms of extensive research (dental fluorosis can be counted as an environmental risk factor in geographic endemic fluorosis belt. The current study concept on tobacco use in dental fluorosis subjects is thought provoking. Further studies from different endemic fluoride belts adds valuable information to the literature. The scarcity of literature on dental fluorosis effect on periodontium might add volumes to the existing research work by the author) is needed to know whether fluorosis is a boon or bane to periodontal structures?

6. Source of Funding

None

7. Conflict of Interest

None

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Author biography

Kharidhi Laxman Vandana, Senior Professor https://orcid.org/0000-0002-1296-7468

Vangara Aditya, Consultant Periodontist and Implantologist https://orcid.org/0000-0003-4886-1005

Manchala Sesha Reddy, Professor

Prasad Sakthidharan Aswin, Consultant Periodontist and Implantologist

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