



Original Article

# The Effect of Habitual Water Pipe and Cigarette Smoking on Clinical Gingival Status in Non-Periodontitis Sample

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## Abstract

**Objective:** The study aimed to investigate the effect of the water pipe and cigarette smoking on clinical gingival parameters of non-periodontitis individuals.

**Methods:** This cross-sectional study recruited 176 men and 49 women aged 17–41, and they were divided into three groups (each with 75 participants): control, cigarette, and water pipe. Furthermore, a case sheet and a questionnaire were prepared to obtain the individual's demographic profile and details regarding cigarette smoking and habitual water pipe-taking data. The following gingival parameters: sulcular depth SD, bleeding index BI, plaque index PI, gingival phenotype Gph, recession R and gingival pigmentation, were recorded and assessed for the three study groups. The obtained data were analyzed statistically using SPSS for Windows version 27.0, with  $P \leq 0.05$  considered statistically significant.

**Results:** Smokers had a higher prevalence of mild brown gingival pigmentation, tooth discoloration, and thick gingival biotype compared to non-smokers ( $P < 0.05$ ). Also, there was a statistically significant link between the type of record and the above indicators (SD, BI, and PI).

**Conclusions:** Water pipe and cigarette smoking negatively influence gingival health measures in non-periodontitis individuals.

**Keywords:** Cigarette, Gingivitis, Gingival phenotype, Gingival pigmentation, Sulcular depth, Water pipe.

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## Introduction

Tobacco use is a known risk factor for human health<sup>1</sup>. Smoking is becoming more popular in the Kurdistan region, and it has been linked to an increase in the prevalence of gingivitis, periodontitis, and oral malignancy. Gingival recession and changes in the oral mucosa can be exacerbated by tissue weakness caused by harmful compounds in tobacco products<sup>2</sup>. In addition, cigarette smokers are at a higher risk of tooth loss and edentulism than nonsmokers<sup>3</sup>.

Water pipe tobacco smoking has become common, especially among young people, because of its social acceptance, availability in numerous flavors, and relatively low cost. In addition, the misconception that waterpipe smoking is a safer means of smoking tobacco is another reason behind the global spread of waterpipe smoking<sup>4</sup>.

Smoking can impact epithelial thickness<sup>5</sup> and lead to epithelial changes such as basal layer hyperplasia<sup>6</sup>. Clinical parameters such as probing pocket depth were higher in smokers than in non-smokers. However, it is worth noting that smokers have been found to have less bleeding on probing and a lower inflammatory response to plaque accumulation than nonsmokers<sup>7</sup>. Tobacco use appeared to reduce gingival bleeding. Research confirmed the reduction in inflammatory signs in smokers, implying that smoking may alter the inflammatory response to dental plaque accumulation<sup>5</sup>. It has been reported that smokers have lower vascular density and a smaller lumen area in their gingival vessels<sup>8</sup>. The gingivitis in smoking and non-smoking patients showed that the plaque formation rates were comparable. Smokers had a lower gingival inflammatory response than non-smokers. Less gingival bleeding and suppuration on probing, tissue redness, edema, and the number of blood vessels in the marginal gingival tissue confirms the reduction in clinical inflammatory signs<sup>9,10</sup>. Previous studies showed that tobacco smoking, an important risk factor of periodontal diseases, might affect the immune-inflammatory response, causing impaired neutrophil functions, elevated production of inflammatory cytokines, delayed revascularization, decreased collagen production, and increased collagen production of collagenase<sup>11,12</sup>.

Gingival pigmentation is the deposition of coloring matter, coloration, or discoloration by a pigment related to the gingiva by a focal increase in melanin deposition along with an increase in melanocyte count at the basal cell layer of the oral epithelium. Over the last decade, our understanding of gingival pigmentation (GP) and its etiologies has grown dramatically. In healthy people with dark skin, light brown to black pigmentation may be physiologic or racial. In addition, physical, chemical, and hormonal factors frequently alter the intensity of pigmentation<sup>13</sup>.

Most of the published articles addressed the impact of smoking on periodontitis and the related variables. However, based on our online searches, we found no similar study was published in the scientific journals to determine the effect of cigarette smoking and waterpipe tobacco smoking on gingival parameters; so far, no studies have specifically addressed the impact of these two types of smoking on all gingival variables in non-periodontitis patients. Therefore, the current study aimed to evaluate various influences of smoking on gingival health in relation to habitual water pipe and cigarette smoking by investigating the following gingival parameters: gingival sulcus depth, gingival bleeding, gingival phenotype recession, and gingival pigmentation in non-periodontitis patients.

## Patients and methods

### Study setting, design, and grouping

Data was collected at Sulaimani University, College of Dentistry, Shorsh Dental Center, and the British Smile Design private center. The collection was conducted between April 1, 2022 and July 13, 2022.

This cross-sectional and questionnaire-based study included questions related to smoking habits and oral hygiene measures, and the assigned clinical parameters were obtained from a total of 225 recruited participants who were divided into three study groups:

- Group I: Cigarette smokers – 75 participants.
- Group II: Water pipe smokers – 75 participants.
- Group III: Nonsmokers – 75 participants – Control group.

### Clinical examination

Included the following gingival parameters:

- Plaque index (PI)
- Bleeding index (BI)
- Sulcular depth (SD)
- Teeth staining

### Gingival pigmentation (GP) index

Gingival pigmentation was scored according to Kumar 2012:

Score 0: Absence of pigmentation.

Score 1: Spots of brown to black color or pigments.

Score 2: Brown to black patches but not diffuse pigmentation.

Score 3: Diffuse brown to black pigmentation, marginal, and attached gingiva.

### Gingival phenotype (Gph)

The gingival phenotype was identified at the facial aspects of the maxillary central incisors by inserting a WHO Periodontal probe into the gingival sulcus at the mid-facial points of the central incisors and considered thin if the underlying periodontal probe could be observed through the gingiva. Whereas it was considered thick if the shadow of the periodontal tissue was obscured by the gingival tissue<sup>15,16</sup>.

### Gingival Enlargement (GE)

The clinical gingival examination was performed to assess gingival enlargement using a Williams periodontal probe. The index grades were based on the height of the enlarged gingiva covering the clinical crown and the nonvisible crown surface at six points around each tooth. In addition, these measurements included the depth of the gingival sulcus according to the following criteria:

Zero (0) sulcular depth is considered normal gingiva;

- 1- Slight: <2 mm increase, and gingiva covered the cervical 1/3 or less of the anatomic crown
- 2- Moderate: 2–4 mm increase and/or gingiva extended into the middle third of the clinical crown; and
- 3- Severe: >4 mm and/or gingiva covered more than 2/3 of the clinical crown<sup>17,18</sup>.

### Gingival recession

At sites where the root surface was exposed, the recession was measured in millimeters as the distance from the cemento-enamel junction (CEJ) to the free gingival margin (this measure is generally known as an apparent or visible recession); the remaining sites were considered to have no recession<sup>19</sup>.

### Inclusion and exclusion criteria

Male and female participants were recruited for this study, aged between 17 and 41. They were systemically healthy subjects not undergoing orthodontic therapies or suffering from systemic diseases. Further, participants

were excluded if they were on long-term medication such as steroidal and non-steroidal anti-inflammatory therapy, anti-hypertensive medications, or other medications that might interfere with the examination protocols that would affect the clinical outcome of the study and if they had previously been treated for periodontitis. Pregnant, lactating, and menopausal women were also excluded from this study.

### Sample size calculation

The sample size was calculated using G\*Power 3.1 program at a P-value of 0.05, a power of 90%, and an effect size of 0.2; the result was 207 participants. In the current study and in order to achieve a higher confidence level, 225 participants were recruited for this cross-sectional study.

### Inter and Intra examiner calibration

Inter-examiner calibration was performed between the researcher and an expert periodontist at the Department of Periodontics in the College of Dentistry, University of Sulaymaniyah, at 88%, after ten days of training. Whereas intra-examiner was performed by the researcher at the level of 85% after training of 5 days duration.

### Study registration

The study proposal was registered with the scientific committee of the College of Dentistry, and ethical approval was obtained from the ethical committee of the College of Dentistry (no. 10 on 1/09/2021).

### Statistical analysis

Each analyzed parameter's results were summarized using descriptive statistics. In addition, the mean and standard deviations of continuous variables were displayed. The Chi-square test was used to test for associations, while the Kruskal-Wallis Test was used to determine whether or not there were statistically significant differences in the measurements of individual variables across different sets of records. p-value of  $\leq 0.05$  was used to establish statistical significance. SPSS for Windows version 27.0 was used for the statistical analysis.

### Results

The distribution of gingival biotypes across record types, age ranges, and genders is shown in Table 1. In the case of the gingival biotype, the control records had the highest proportion of thin types (84.85%), followed

by thick types (24.48%). In the cigarette records, the thick type accounted for 38.54%, whereas the water pipe record type accounted for 36.98%. According to the data on age, among participants over the age of 37, the thick gingival phenotype was the most frequent. Participants below the age of 24 had almost equal percentages of thin and thick gingival phenotypes, while among those aged between 25 and 39, the thin gingival phenotype was slightly more frequent. Based on gender, the thick Gph type was found more frequently in the male group, but the thin type was found more frequently in the female group, as shown in Table 1. There was a statistically significant association between record type, gender, and gingival biotype at a level of ( $P = 0.05$ ). It should be noted that the p-values (sig.) of the Chi-square tests of both the record type and gender were equal to 0.000 and 0.000, respectively, values which are less than the level

of significance ( $\alpha = 0.05$ ), meaning they are statistically significant. However, the association between age group and gingival biotype is not statistically significant ( $p > 0.05$ ). Table 2 shows the distribution of gingival pigmentation by record type, age group, and gender. The control record data showed pink gingival pigmentation, but no other pigmentations were found. Meanwhile, most mild brown instances were found in the cigarette and water pipe records, and the vast majority (83.33%) of mixed pink and brown instances were identified in the cigarette records. In terms of age, most instances of mild brown pigmentation were discovered in those participants between the ages of 31 and 36, as shown in Table 2. Only one example of deep brown pigmentation was identified in a smoker participant aged under 24 years old. Males were more likely to have pink and mild brown pigmentation than

females. A statistically significant association was found between record type, age range, and gender and gingival pigmentation at the  $\alpha = 0.05$  level ( $p = 0.000, 0.041, \text{ and } 0.013$ ).

The distribution of gingival hypertrophy in relation to record type, age group, and gender is shown in Table 3. According to the control and waterpipe records, 33.70% of participants were without gingival hypertrophy, followed by 32.60% in the cigarette record type. Additionally, distributions of gingival hypertrophy in control, cigarette, and water pipe records were approximately 31.36%, 31.36%, and 31.82%, respectively. In addition, 40.91% of gingival hypertrophy cases were detected in participants between the ages of 25 and 30.

		Gingival Bio Type		Chi-Square test
		Thin	Thick	
Record Type	Control	28	47	p-value= 0.000
		84.85%	24.48%	
	Cigarette	1	74	
3.03%		38.54%		
Water pipe	4	71		
	12.12%	36.98%		
Age (Group)	<= 24	9	51	p-value= 0.534 NS
		27%	26.56%	
	25 - 30	10	51	
		30%	26.56%	
	31 - 36	12	60	
		36%	31.25%	
37+	2	30		
	6%	15.63%		
Gender	Male	17	159	p-value= 0.000
		51.52%	82.81%	
	Female	16	33	
48.48%		17.19%		
Total		33	192	
		100%	100%	

Table 1: Distribution of record type, age group, and gender in relation to gingival biotype.

NS: Non-Significant

Males made up 79.56% and 72.73% of the cases with and without gingival hypertrophy, respectively, whereas females made up 20.44% and 27.27% of the cases with and without gingival hypertrophy, respectively. The chi-square test showed no significant association between record type, age range, and gender and gingival hypertrophy at the levels of ( $\alpha = 0.05$ ) ( $p = 0.89, 0.082, \text{ and } 0.97$ ), respectively.

The distribution of teeth staining by record type, age group, and gender is shown in Table 4. No cases in the control records were found to have teeth staining. Meanwhile, cigarettes and water pipes accounted for 71.05% and 28.95% of the cases of teeth staining, respectively. The majority of those with stained teeth (52.63%) were in the age range of 31 to 36. A much higher incidence of teeth staining was found amongst

males, who accounted for 94.74% of such cases, while females accounted for 5.26%. The chi-square test indicates that teeth staining is statistically significantly associated with record type, age group, and gender at ( $\alpha = 0.05$ ) ( $p=0.000, 0.024, \text{ and } 0.007$ ), respectively.

Table 5 shows verification of whether the differences between variable measurements are statistically significant or not according to record types. In this study,

the p-values of measurement variables BI and GR equal 0.101 and 0.978, respectively, values greater than the significance level (0.05). This means that record type has no statistically significant effect on BI and GR, while the p-values of measurements SD and PI are equal to 0.000 and 0.018, respectively, which are values lower than the significance level (0.05), meaning that cigarette and water pipe smoking has a statistically significant effect on SD and PI.

Table 2: Distribution of record type, age group, and gender in relation to gingival pigmentation.

		Gingival Pigmentation				Chi-Square test
		Pink	Mild Brown	Mixed Pink and Brown	Deep Brown	
Record Type	Control	75	0	0	0	p-value= 0.000
		46.58%	0.00%	0.00%	0.00%	
	Cigarette	37	27	10	1	
		22.98%	52.94%	83.33%	100.00%	
Water Pipe	49	24	2	0		
	30.43%	47.06%	16.67%	0.00%		
Age (Groups)	<= 24	49	9	1	1	p-value= 0.041
		30.43%	17.65%	8.33%	100.00%	
	25 - 30	48	12	1	0	
		29.81%	23.53%	8.33%	0.00%	
	31 - 36	47	19	6	0	
		29.19%	37.25%	50.00%	0.00%	
37+	17	11	4	0		
	10.56%	21.57%	33.33%	0.00%		
Gender	Male	117	46	12	1	p-value= 0.013
		72.67%	90.20%	100.00%	100.00%	
	Female	44	5	0	0	
27.33%		9.80%	0.00%	0.00%		
		161	51	12	1	
Total		100.00%	100.00%	100.00%	100.00%	

Table 3: Distribution of record type, age group, and gender in relation to gingival enlargement.

		Gingival Hypertrophy		Chi-Square test	
		0	1		
Record Type	Control	61	14	p-value= 0.89 NS	
		33.70%	31.82%		
	Cigarette	59	16		
Age (Group)	<= 24	61	14		p-value= 0.22 NS
		33.70%	31.82%		
	25 - 30	43	18		
	31 - 36	60	12		
37+	25	7			
	13.81%	15.91%			
Gender	Male	144	32	p-value= 0.99 NS	
		79.56%	72.73%		
	Female	37	12		
Total		181	44		
		100.00%	100.00%		

NS: Non-Significant

Table 4: Distribution of record type, age group, and gender in relation to teeth staining.

		Teeth Staining		Chi-Square test	
		Yes	No		
Record Type	Control	0	75	p-value= 0.000	
		0.00%	40.11%		
	Cigarette	27	48		
Age (Binned)	<= 24	11	64		p-value= 0.024
		28.95%	34.22%		
	25 - 30	6	54		
	31 - 36	7	54		
37+	20	52			
	52.63%	27.81%			
Gender	Male	5	27	p-value= 0.007	
		13.16%	14.44%		
	Female	36	140		
Total		2	47		
		5.26%	25.13%		
Total		38	187		
		100.00%	100.00%		

Table 5: Comparison of record types in relation to continuous variables.

Variable	Record type	Mean	Std. Deviation	Kruskal-Wallis Test	Chi-Square test
SD	Control	1.7025	0.37963	63.062	p-value= 0.000
	ette	2.235	0.42774		
	Water pipe	2.2865	0.46409		
	Total	2.0747	0.49921		
BI	Control	0.1999	0.1668	4.584	p-value= 0.101
	Cigarette	0.2306	0.22629		
	Water pipe	0.3186	0.32813		NS
	Total	0.2497	0.25341		
PI	Control	0.5578	0.40407	8.09	p-value= 0.018
	Cigarette	0.6956	0.39469		
	Water pipe	0.5987	0.30257		
	Total	0.6174	0.37282		
GR	Control	0.026	0.14803	0.045	p-value= 0.978
	Cigarette	0.04	0.19728		
	Water pipe	0.0469	0.21304		NS
	Total	0.0388	0.19094		

NS: Non-Significant

## Discussion

Tobacco use carries a known risk of human health decline. Smoking is a confounder risk determinant of periodontal diseases and other systemic diseases such as atherosclerotic cardiovascular disease<sup>20</sup>, diabetes mellitus, respiratory tract infection, oral malignancy, and other health problems. In addition, smoking alters the gingival and periodontal tissue's integrity through the content of tobacco, mainly nicotine and other noxious particles, which may play a role in gingival hyperpigmentation and obliterating the terminal blood vessels and altering the gingival phenotype by increasing the thickness of the gingival keratin layer<sup>21</sup>. Moreover, the majority of smokers in the global population are estimated to be males.

In the present study, and in terms of the association between smoking and gingival phenotype, the gingival thickness was significantly higher among both smoker groups compared to the non-smoker group. This finding is related to histopathological changes such as epithelial hyperplasia, increased keratinization, and changes in connective tissues<sup>5</sup>. The study's results identified a statistically significant association between the record groups and gingival phenotype, which supports the result of a previous study<sup>22</sup>. Furthermore, regarding gingival pigmentation, the results of this study illustrate that younger patients had more significant smoking gingival pigmentation than older patients. Gingival

hyperpigmentation is caused by excessive deposition of melanin in the basal and suprabasal cell layers of the epithelium due to tobacco consumption.

While smoking rates in developed countries continue to fall year after year<sup>23</sup>, Araki et al. stated that smokers who smoked more than ten daily cigarettes had a significantly higher prevalence of gingival pigmentation<sup>24</sup>. In addition, the current study found a statistically significant association of record type, age range, and gender with gingival pigmentation.

Even though smokers' gingiva may appear normal, smoking causes epithelial changes that resemble the early stages of dysplasia and reduce the inflammatory response. De Oliveira Semenzati et al. (2012) investigated smoking's effects on the mucosa of the tongue, pharynx, and larynx in rats. The study focused on epithelial hyperplasia, basal cell hyperplasia, and mild to moderate dysplasia<sup>6</sup>. The findings based on this animal model matched those from the human study. However, the current researcher could find no studies on the effects of smoking on cellular polarity and related changes in human gingival epithelial cells<sup>25</sup>. Furthermore, the chi-square test showed no significant association of record type, age range, and gender with Gingival hypertrophy classification at the level of ( $\alpha=0.05$ ) because, with age, the width and the thickness of the gingiva reduce, which compensates for the thickening that happens due to smoking.

In the case of discoloration of the teeth, smokers had nearly twice as much prevalence of self-assessed tooth discoloration as non-smokers. This, however, was only true for moderate and severe levels. Not only were smokers more likely to notice discoloration, but they were also more likely to be unhappy with their appearance. As a result, the findings imply that smoking has a negative impact on tooth color and perceived dental esthetics<sup>26</sup>. This previous study showed that teeth staining was more prevalent in smoker groups due to nicotine and tar. The nicotine and tar from smoking seep into the tooth's enamel through tiny pores, leaving the teeth discolored. The tar adds a brown tinge to the teeth, while the nicotine, when combined with oxygen, causes yellowing. The chi-square test indicates that teeth staining is statistically significantly associated with record type, age group, and gender at the level of ( $\alpha = 0.05$ ).

While studies on smokers have focused on increasing pocket depth and attachment loss<sup>27,28</sup>, the current study found that record type had no statistically significant effect on BI and GR, and the p-values of measurements SD and PI were equal to 0.000 and 0.018, respectively. Nicotine restricts the blood flow to the gums due to vasoconstrictive effects and masks the signs of gingival disease, so no bleeding is apparent. However, it also affects the neutrophil functions, impairs chemotaxis, reduces antibody production, and decreases the oxygen level, which leads to alteration of the subgingival environment and colonization by anaerobic bacteria, finally leading to periodontal destruction.

Cigarette and water pipes were found to affect the following measures in decreasing order:

1. Sulcular depth measurement.
2. Bleeding index.
3. Had much less impact on the plaque index.
4. Had no statistically significant effect on gingival recession.

The current study highlights the negative influence of both tobacco usage types on most clinical gingival variables, including gingival pigmentation, gingival biotype, tooth staining, and gingival bleeding, with age having an increasing effect on these variables. Moreover, the detrimental effects of smoking on general human health call for restraining action, age limitations on water pipes used as a routine practice in public cafes and restaurants, and restrictions on cigarette consumption among our population.

## Conclusions

Water pipe and cigarette smoking negatively influence non-periodontitis gingival health measures. Water pipe smoking and cigarette smoking were found to have impacts of largely the same magnitude.

## Conflict of Interest

The authors declare no conflict of interest for this study.

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## References

1. Longo DL, Jameson JL, Kasper D. Harrison's Principles of Internal Medicine: Volume 2. Macgraw-Hill; 2011.
2. Newman Mg, Takei HH, Klokkevold PR. Carranza's clinical periodontology. Chicago: Saunders Elsevier. 2006. pp1224.
3. Hanioka T, Ojima M, Tanaka K, Matsuo K, Sato F, Tanaka H. Causal assessment of smoking and tooth loss: a systematic review of observational studies. BMC Public Health. 2011;11(1):1-10.
4. Martinasek MP, McDermott RJ, Martini L. Waterpipe (hookah) tobacco smoking among youth. Curr Probl Pediatr Adolesc Health Care. 2011;41(2):34-57.
5. Villar CC, Lima AFM de. Smoking influences on the thickness of marginal gingival epithelium. Pesqui Odontológica Bras. 2003;17(1):41-5.
6. de Oliveira Semenzati G, de Souza Salgado B, Rocha NS, Michelin Matheus SM, de Carvalho LR, Garcia Martins RH. Histological and immunohistochemical study of the expression of p53 and ki-67 proteins in the mucosa of the tongue, pharynx and larynx of rats exposed to cigarette smoke. Inhal Toxicol. 2012;24(11):723-31.
7. Nassrawin NA. Effect of smoking on the response to nonsurgical periodontal therapy. East Mediterr Health J. 2010;16(2):162-5.
8. Mirbood SM, Ahing SI, Pruthi VK. Immunohistochemical study of vestibular blood vessel density and internal circumference in smokers and non smokers. J Pak Med Assoc. 1994;44(9):210-2.
9. Bergström J. Oral hygiene compliance and gingivitis expression in cigarette smokers. Eur J Oral Sci. 1990;98(6):497-503.

10. Bergström J, Preber H. The influence of cigarette smoking on the development of experimental gingivitis. *J Periodontol Res.* 1986;21(6):668-76.
11. Deng K, Pelekos G, Jin L, Tonetti MS. Gingival bleeding on brushing as a sentinel sign of gingival inflammation: A diagnostic accuracy trial for the discrimination of periodontal health and disease. *J Clin Periodontol.* 2021;48(12):1537-48.
12. Mathew JE, Jacob JJ, Kalra S. Periodontitis management in diabetes care. *J Pak Med Assoc.* 2021;71(8):2097-9.
13. Periodontology AAP. Glossary of periodontal terms. Chicago Am Acad Periodontol. 2001.
14. Kumar S, Bhat GS, Bhat KM. Development in techniques for gingival depigmentation- an update. *Indian J Dent.* 2012;3(4):213-21.
15. Kan JYK, Rungcharassaeng K, Umezu K, Kois JC. Dimensions of peri-implant mucosa: an evaluation of maxillary anterior single implants in humans. *J Periodontol.* 2003;74(4):557-62.
16. Olsson M, Lindhe J. Periodontal characteristics in individuals with varying form of the upper central incisors. *J Clin Periodontol.* 1991;18(1):78-82.
17. Miller CS, Damm DD. Incidence of verapamil-induced gingival hyperplasia in a dental population. *J Periodontol.* 1992;63(5):453-6.
18. Angelopoulos AP, Goaz PW. Incidence of diphenylhydantoin gingival hyperplasia. *Oral Surgery, Oral Med Oral Pathol.* 1972;34(6):898-906.
19. Joshipura KJ, Kent RL, DePaola PF. Gingival recession: intra-oral distribution and associated factors. *J Periodontol.* 1994;65(9):864-71.
20. Zardawi F, Gul S, Abdulkareem A, Sha A, Yates J. Association between periodontal disease and atherosclerotic cardiovascular diseases: revisited. *Front Cardiovasc Med.* 2021;7:625579.
21. Ankola A, Nagesh L, Tangade P, Hegde P. Assessment of periodontal status and loss of teeth among smokers and non-smokers in Belgaum city. *Indian J Community Med.* 2007;32(1):75-6.
22. Taltia A, Arjunkumar R. Assessment of Gingival thickness in smokers and non-smokers-A clinical study. *Int J Pharm Clin Res.* 2016;8(6):574-7.
23. Ng KT, Gillies M, Griffith DM. Effect of nicotine replacement therapy on mortality, delirium, and duration of therapy in critically ill smokers: a systematic review and meta-analysis. *Anaesth Intensive Care.* 2017;45(5):556-61.
24. Araki S, Murata K, Ushio K, Sakai R. Dose-response relationship between tobacco consumption and melanin pigmentation in the attached gingiva. *Arch Environ Heal An Int J.* 1983;38(6):375-8.
25. Jalayer Naderi N, Semyari H, Elahinia Z. The Impact of Smoking on Gingiva: a Histopathological Study. *Iran J Pathol.* 2015;10(3):214-20.
26. Alkhatib MN, Holt RD, Bedi R. Smoking and tooth discolouration: findings from a national cross-sectional study. *BMC Public Health.* 2005;5(1):1-4.
27. Sreedevi M, Ramesh A, Dwarakanath C. Periodontal status in smokers and non-smokers: a clinical, microbiological, and histopathological study. *Int J Dent.* 2012;2012:571590.
28. Calsina G, Ramón J, Echeverría J. Effects of smoking on periodontal tissues. *J Clin Periodontol.* 2002;29(8):771-6.