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Assessment of the correlation between exhaled carbon monoxide level and periodontitis

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Abstract

Assessment of the correlation between exhaled carbon monoxide levels and periodontitis. There were 2 group . Study group and control group. Study group included somkers with periodontitis and control group included non smokers without periodontitis. They were further subdivided into groups.

Group 1: Smokers with mild periodontitis. Group 2: Smokers with moderate periodontitis. Group 3: Smokers with severe periodontitis. Group 4: Non-smokers with mild periodontitis.

Group 5: Non-smokers with moderate periodontitis. Group 6: Non-smoker with severe periodontitis. And the exhaled carbon monoxide level was measured with the help of breath testing machine called PicoSmokerlyzer. Clinical evaluation included Probing depth, Bleeding on probing,clinical attachment level and plaque index. Comparison of carbon mono-oxide levels among smokers with different levels of periodontitis. Statistical analysis showed that there was a statistically significant difference in carbon mono-oxide level among smokers and non smokers. On overall correlation between carbon monoxide level and various parameters of periodontitis are correlated among smokers group (study group) than the non-smokers group (control group). Although the bleeding on probing showed weak relation with carbon mono-oxide level among study group (smokers) than control group (non-smokers). The results indicated that the exhaled carbon monoxide level among the case group were higher and found to be significant in comparison to the control group which is the suggestive of the fact that periodontitis is related to increased level of carbon monoxide level. From the above study, it can be stated that Smoking has significant deleterious effect on periodontal health.

Keywords: Chronic Periodontitis, Smoking, Carbon monoxide, Clinical attachment level, Pico Smokerlyzer.

Introduction

The periodontal diseases are highly prevalent and affect up to 90% of worldwide population.[1] It is one of the most common chronic disease. It is second only to dental caries as a cause of tooth loss among adults in developed countries.[2]Periodontal are a dynamic phenomenon with cyclical patterns of progression and resolution at any given site[3].A number of risk factors have been implicated for the onset, progression and severity of periodontal diseases. These include genetics, ethnicity, advancing age, smoking, diabetes, poor oral hygiene, immune-compromised status, etc. Among these, smoking is an important global public health issue. Gingival bleeding has been consistently reported to occur less in smokers due to nicotine induced vasoconstriction in smoker's gingiva as well as heavy gingival keratinization. Pocket depth measurements are found to be greater in smokers due to increased alveolar bone loss. Tobacco smoking probably plays a significant role in the development of refractory periodontitis.[4] Periodontal breakdown has been shown to be more severe among current smokers compared to former smokers. Those who have never smoked have been observed to have the lowest risk.[5] Smoking has a strong negative impact on regenerative therapy, including osseous grafting, guided tissue regeneration, or a combination of this treatment.[6]

Tobacco smoke contains numerous amounts of noxious chemicals, and it comprises a gaseous phase and a particulate phase. The gaseous phase contains carbon monoxide, ammonia, formaldehyde, hydrogen cyanide and many other harmful and irritant compounds, including more than known carcinogens, such as benzopyrene and dimethyl nitrosamine. The particulate phase includes nicotine "Tar" [7].

Potential molecular and cellular mechanisms in the pathogenesis of smoking associated periodontal diseases has been reported and these include, immuno-suppression, exaggerated inflammatory cell responses, and impaired stromal cell functions of oral tissues. The association between cigarette smoking and periodontal diseases represent a significant oral health problem.[8]

Carbon Monoxide (CO) is a colorless, odorless and tasteless flammable gas that is slightly less dense than air. It is a low molecular weight gas, present as a product of organic combustion. Carbon monoxide arises in the human body as the natural product of hemoprotein turnover, most of which originates from circulating hemoglobin.[9] The CO produced forms a compound with hemoglobin (Hb) called carboxyhemoglobin (COHb), which is red in color and has more affinity towards hemoglobin than that of oxygen.[4] COHb values are higher in habitual smoker than non-smokers.[10]

Bergstrom reviewed the correlation between smoking and periodontal diseases and found the negative impact of smoking on periodontal health .[11]Multiple cross sectional and longitudinal studies have demonstrated that pocket depth, attachment loss and alveolar bone loss are more prevalent and severe in smokers as compared to non-smokers.

Measurement of the exhaled CO level in smokers is an easy and quick method for health professionals to check the smoking status of the patients. A non – invasive portable method that uses a Pico Smokerlyser (Bedfont Instruments; Kent, UK) which does not require any specific skill to assess the carbon monoxide level has been used in the present study to assess CO levels.

Material and method

Study Population

This study was carried out in Himachal Institue of Dental Sciences Paonta sahib and the patient were

recurited from the outpatient Department of Periodontology, This study included 60 patient. Patient was selected depending on the following criteria

Inclusion Criteria

1. Participants above 19 years.
2. Smokers and non-smokers diagnosed with periodontitis having a minimum of 24 teeth present

The participants were divided into subgroups based on the severity of periodontitis:

- **Mild Periodontitis**

Probing depth (PD): ≥ 3mm and ≤ 5mm

Bleeding on probing (BOP) - Present.

Clinical attachment level (CAL) – 1 to 2 mm.

- **Moderate Periodontitis**

PD: ≥ 5mm and ≤ 7mm

BOP - Present.

CAL – 3 to 4 mm.

- **Severe Periodontitis**

PD: ≥ 7mm

BOP – Present.

CAL ≥ 5mm.

Exclusion Criteria

1. Any history of chronic cardiovascular, metabolic or immunological diseases and chronic disorder.
2. Pregnant and lactating women.

The participants were scored for

(1) Bleeding on probing (BOP),

(2) Plaque index (PI), and

(3) Periodontal pocket depth (PD)

(4) Clinical attachment level (CAL) were noted.

Breath Co Monitoring

Breath CO monitoring was done with the help of PicoSmokerlyzer used to measure the exhaled carbon monoxide level of the participants. The reading were given in ppm. To standardize the breath being analyzed by the smokerlyser the subjects were asked to exhale completely, inhale fully and then hold their breath for 15s. If the subject was unable to hold their breath for 15sec, they were asked to hold it for as long as possible and then it was recorded. Following the breath hold, the subjects were asked to exhale slowly into the Picosmokerlyser and were encouraged to exhale fully in order to record the value.

Results and Discussion

Carbon Mono-oxide Levels Among Smokers

Table 1 shows the comparison of carbon mono-oxide levels among smokers with different levels of periodontitis. Statistical analysis showed statistically significant difference in CO level among smokers (P<0.001).

Number of Cigarette Smoked per day Among Smokers

Table 2 On comparison between the three sub-groups of periodontitis smokers and the number of cigarettes smoked per day among them, there was statistically significant difference between the severity of periodontitis and no. of cigarettes smoked.

Carbon Mono-oxide Levels Among Non-Smokers

Table 3 shows the comparison of CO levels among non-smokers with different levels of periodontitis. Statistical analysis showed that there was a statistically significant difference in CO level among non-smokers (P=0.001).

Comparison between Smokers and Non-smokers

The comparison of CO levels between smokers and non-smokers is shown in Table 4. Statistical analysis showed that:

- There was a statistically significant difference in CO levels between G1 and G4 (P<0.001). The CO levels in smokers was significantly higher than that in non-smokers.
- There was a statistically significant difference in CO levels between G2 and G5 (P<0.001). The CO levels

in smokers was significantly higher than that in non-smokers.

- There was a statistically significant difference in CO levels between G3 and G6 (P<0.001). The CO levels in smokers was significantly higher than that in non-smokers.

Table 5 depicts that the overall correlation between CO level and various parameters of periodontitis are correlated among smokers group (study group) than the non-smokers group (control group). As the various parameters of periodontitis (clinical attachment loss, plaque index) are correlated that means the periodontitis is strongly correlated with the CO level in smokers (study group). Although the bleeding on probing showed weak relation with CO level among study group (smokers) than control group (non-smokers) which suggests that study group (smokers) have less association with bleeding on probing.

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific micro-organisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both [13].

Periodontal diseases are infectious caused by dental plaque, but risk factors can modify the host response to microbial aggression [14].

It is well established that smoking has a major adverse effect on the periodontal supporting tissues, increasing the risk of periodontitis by 2 to 5-fold.[15]

Various investigators have attempted to identify the role of tobacco smoking in etiology of periodontal diseases. The study suggest that smoking is a single, modifiable environmental risk factor responsible for excess prevalence of periodontal disease in the population and has a direct influence on periodontal variables.[16]

The measurement of breath CO level may provide an immediate, non- invasive method of assessing smoking status.

A study by S. Erhan Deveci et al (2004) [17]. reported that measuring breath CO levels provides an immediate, non-invasive, simple and effective way of confirming a person's smoking status. The study also reported that exhaled CO levels were higher in healthy smokers than in healthy non-smokers. Also the exhaled CO level with the Smokerlyser was significantly higher in healthy smokers than healthy non- smoker. Cross-Sectional studies have consistently observed a higher prevalence and a greater extent and severity of periodontal destruction in current smokers than in never or former smokers.[18]

Johannsen et al 2014[18] has quoted that exposure of leukocytes, and other cells, to cigarette smoke condensate had resulted in a variety of effects, some of which could be of importance for pathogenesis in periodontitis. Chronic cigarette smoking has been shown to increase the total white blood cell count, and to have a greater effect on neutrophilic granulocytes than on other types of white blood cells. Smoking activates inflammatory cells, increasing the systemic levels of several inflammatory markers, including C-reactive protein, fibrinogen, interleukin-6 and haptoglobin. Many of this substance and mediators have been associated with periodontitis.

Cigarette smoke contains at least 400 potentially toxic substances, including hydrogen cyanide, carbon monoxide (resulting in formation of carboxyhemoglobin), free radicals, nicotine, nitrosamines (potent carcinogens) and a variety of

oxidant gases (causing platelet activation and endothelial dysfunction).[19]

In the present observational study, a total of 60 subjects were enrolled and categorized into study groups and control groups on basis of clinical examination. Patients with any history of systemic disease, chronic cardiovascular disease were excluded because it may affect periodontal health due to inflammation as it plays a pivotal role in progression of atherosclerosis. Individuals with diabetes mellitus (DM) were excluded because of complex interrelations between periodontitis and DM and pregnant females were excluded because of hormonal imbalance which occur during pregnancy.

On Comparison of severity of periodontitis with CO level among smokers shows that there was no statistically significant difference in CO level between group 1 and group 2 (p=0.071) and there was statistically significant difference in CO levels between group 1 and group 3 (p<0.001). The CO level in group 3 was higher than group 1. There was a statistically significant difference in CO level between group 2 and group 3 and group 3 has higher CO level than group 2. These result was supported by the author Dr. Shalu Bathla in her book entitled Periodontics Revisited [20] by taking in consideration role of smoking in pathogenesis of periodontal disease: Tobacco smoke contains powerful reducing agent such as carbon monoxide that produce a marked immediate reduction of redox potential at mucosal surfaces. The powerful physicochemical reducing activity of carbon monoxide is direct mechanism to promote growth of anaerobes at superficial sites and through which the molecular byproducts of smoking influence progression of periodontitis by damaging the cells and it further cause damage to leukocytes.

The comparison of number of cigarettes smoked per day with the different levels of periodontitis. Statistical analysis showed that there was statistically difference in number of cigarettes smoked per day among the different level of periodontitis in smokers (p=0.001).

These results were similar in accordance with the study done by Kitti Torrungruang et al (2005) [21] in which there was a positive correlation between the level of cigarette consumption and severity of periodontitis. The more cigarette consumption the worse periodontal condition was observed in their study. This finding strongly suggested that cigarette smoking was related to more severe periodontitis and the current smokers had the lowest proportions of subjects with mild periodontitis and highest proportion with severe periodontitis.

The similar findings were also noticed in a study performed by Jasim M Albander et al (2000) [22] in which current cigarette smokers had worst periodontal status among the 3 groups followed by former smoker and non-smokers. There was a significantly higher prevalence of moderate/severe periodontitis among current cigarette smokers and higher prevalence and extent of attachment loss.

On comparison with CO level of carbon monoxide level among non-smoker with different level of periodontitis showed statistically significant in CO level among non- smoker (p=0.002).

In some non-smokers the CO values can be seen raised. This conclusion can be attributed to passive smoking or exposure to environmental tobacco smoking (ETS). This finding can be positively correlated with the study conducted by Deveci SE et al (2004) [23] in which a person deemed to have been exposed to ETS if a household member had regularly smoked cigarettes in their presence or if any co-worker had done so in same

indoor room in their presence for more than one year during the past 10 years.

Nicotine, which is a highly psychoactive tertiary amine, on chronic exposure in the brain, it stimulates the release of dopamine and other neurotransmitter into CNS neuronal clefts, which on repeated exposure causes proliferation of nicotine acetylcholine receptors on the post synaptic neuron that leads to intraneuronal changes occur that alter gene expression.

The reason for increase in CO level in smoker as compared to non-smoker is that the smoking is the major source of carbon monoxide, and it is responsible for various health risk and diseases. Carbon monoxide is eliminated from the body via expired air following dissociation in pulmonary capillaries. As COHb remain equilibrium with expired carbon monoxide level. The Smokerlyser measures breath CO level in parts per million based on the conversion of CO to carbon dioxide over a catalytically active electrode. On breath holding the CO in the blood forms an equilibrium with the CO in the alveolar air.

In support of the present study result the study conducted by Grossi SG et al (1994)[24] quoted that there is a strong association between the amount of smoking and the severity of attachment loss and it could be explained by a number of biologic phenomenon. Cigarette smoking, nicotine and its byproduct have a vasoconstrictive effect not only on peripheral circulation but on gingival blood vessel as well. In addition to this smoking reduce the functional activity of leukocyte and macrophages in saliva and crevicular fluid as well as decreasing chemotaxis and phagocytosis of blood and tissue polymorphonuclear leukocytes, thereby likely depressing phagocyte mediated protective responses to periodontal pathogens.

The reason for decrease in Bleeding and probing in study group as compared to control group is due to the presence of nicotine in the cigarette smoke that can stimulate sympathetic ganglia releasing neurotransmitters, including catecholamines. Catecholamines are the potent vasoconstrictors causing a reduction in the blood flow of gingiva and this result in decreased bleeding on probing. The decreased gingival bleeding on probing can also be attributed to the heavier keratinization of gingiva in smokers.

In this present study, the results indicated that the exhaled carbon monoxide level among the case group were higher and found to be significant in comparison to the control group which is the suggestive of the fact that periodontitis is related to increased level of carbon monoxide level. From the above study, it can be stated that Smoking has significant deleterious effect on periodontal health.

Figure 1: PICOSMOKERLYZER

Table 1. Carbon Mono-Oxide Levels among three study groups.

Smoker's Periodontal Status	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		P value
					Lower Bound	Upper Bound	
Mild (G1)	10	10.3500	2.10139	1.31535	3.4376	11.1624	
Moderate (G2)	10	18.6700	4.54625	2.11319	9.3150	20.1830	<0.001*
Severe (G3)	10	25.2000	6.34363	2.65136	15.3133	28.6717	
Total	30	22.8000	9.65693	1.96635	6.1393	26.0607	

*Statistically significant (P<0.05, Analysis of Variance)

Table 2: Comparison of mean cigarette smoked per day among the three study groups.

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		P value
					Lower Bound	Upper Bound	
Mild	10	8.5000	4.15650	2.15667	1.0705	10.1895	
Moderate	10	11.5000	8.38781	4.17842	2.6357	17.3743	0.001*
Severe	10	17.3000	9.66215	5.18621	5.9989	32.1021	
Total	30	36.9000	22.16971	7.19425	7.0805	44.8965	

Table 3 Carbon mono-oxide levels among three control groups.

Non-Smoker Periodontal Status	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		P value
					Lower Bound	Upper Bound	
Mild (G4)	10	1.3000	.15650	.15667	1.0705	3.1895	
Moderate (G5)	10	2.4000	.48781	.17842	1.6357	3.3743	0.001*
Severe (G6)	10	3.3000	.66215	.18621	1.9989	4.1021	
Total	30	2.9000	1.16971	.19425	2.0805	4.8965	

*Statistically significant (P<0.05, Analysis of Variance)

Table 4. Comparison of carbon mono-oxide levels between smokers (study group) and non-smokers(control group).

	Group	N	Mean	Std. Deviation	P value
CO Levels in Mild Periodontitis	Smokers(G1)	10	10.3500	2.10139	<0.001*
	Non-Smokers(G4)	10	1.3000	0.15650	
CO Levels in Moderate Periodontitis	Smokers(G2)	10	18.6700	4.54625	<0.001*
	Non-Smokers(G5)	10	2.4000	.48781	
CO Levels in Severe Periodontitis	Smokers(G3)	10	25.2000	6.34363	<0.001*
	Non-Smokers(G6)	10	3.3000	.66215	

*Statistically significant (P<0.05, Independent t-test)

Table 5. Overall correlation between CO level and various parameters of periodontitis among study group (smokers) and control group (non-smokers).

Pearson Correlation	Plaque Index	Bleeding on Probing	Clinical Attachment Loss	P Value
CO SMOKERS (study group)	0.84	0.21	0.86	0.002*
NON-SMOKERS (control group)	0.26	0.41	0.24	0.004*

Conclusion

The present study was carried out to assess the correlation between exhaled carbon monoxide levels and periodontitis in smokers and non- smokers patients.Within the limit of the study on the overall correlation between carbon monoxide level and various parameters of periodontitis are correlated among Study group (smokers) than the Control group (non smokers). As various parameters of periodontitis are correlated that means periodontitis is strongly correlated with the carbon monoxide level in smokers. Although the bleeding on probing showed weak relation with CO level among Study group (smokers) than control group (non smokers) which suggests that Study group (smokers) have less association with bleeding on probing.

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