Original Research Article

UNVEILING THE SMOKING - PERIODONTITIS LINK FOR DIVERSE PATIENT GROUP

Abstract

Background:

Periodontitis is a chronic inflammatory disease affecting the supporting structures (periodontium) of teeth and progress to tooth loss. Smoking is a well-established modifiable risk factor that influences periodontal disease through altered immune response and changes in the oral environment. Due to demographic and behavioral factors impact of smoking on periodontal health may vary among diverse patient group.

Objective:

This study aims to investigate the relationship between smoking and periodontitis in diverse patient groups, with special importance on salivary pH changes and breath analysis (Exhaled Carbon Monoxide) as an additional diagnostic indicators used in this study .

Methodology:

A cross-sectional analytical study was conducted among diverse patient groups from varied demographic backgrounds. Periodontal parameters such as probing depth, clinical attachment loss, bleeding on probing and mobility of tooth were recorded. Smoking status, duration were assessed using structured questionnaires. Unstimulated saliva samples were collected to measure salivary pH, and breath analysis was performed to assess carbon monoxide associated with and without periodontal disease. Comparative and statistical analyses were carried out between smokers and non-smokers across different patient groups.

Results:

Smokers demonstrated significantly increased periodontal destruction, altered salivary pH levels, and higher levels of exhaled carbon monoxide compared to non-smokers. Periodontal health is compromised positively with smoking, among diverse patient groups.

Conclusion:

Smoking adversely affects periodontal health by altering both clinical parameters and oral biochemical markers. Incorporating salivary pH and breath analysis (Exhaled Carbon Monoxide) may enhance Early detection and management of smoking-related periodontitis can enhanced by salivary pH and breath analysis(Exhaled Carbon Monoxide)

Keywords:

Smoking, Periodontitis, Salivary pH, Breath Analysis

Introduction

Periodontitis is a chronic inflammatory condition that affects the supporting structures of the teeth(Periodontium) . Periodontitis remains highly prevalent worldwide and continues to play a major public health challenge. Primary etiological factor of periodontitis is Dental Plaque Biofilm, the onset and progression of periodontal disease are strongly influenced by host-related and environmental risk factors. Among these, tobacco smoking has been consistently recognized as one of the most important and modifiable contributors to periodontitis [2,4–11].

Early epidemiological studies exhibit that periodontitis are more likely to develop in smokers and experience greater virulent of periodontal tissue breakdown compared with non-smokers [7]. Bergstrom first spotlight a chronic periodontal disease also caused by risk factor of cigarette with the features of increased attachment loss and alveolar bone destruction among smokers [4]. Following studies and investigation prove that smoking not only increases the prevalence of periodontitis but also accelerates disease progression and compromises periodontal treatment outcomes [5,9,10]. These findings have been reinforced by longitudinal and cross-sectional studies across different populations, firmly establishing smoking as a major determinant of periodontal health [6,8,11].

Periodontal disease are complex and multifactorial, one of the most evident factor is Tobacco smoking. Smoking is an act due to burning substance of tobacco (ex-Cigarette, Bidi,Cigar etc…). Majority of smoker associated with smoking for stress relief, social interaction, improving concentration. Smoking has both direct and indirect effect. Tobacco smoke contains numerous toxic substances such as nicotine, carbon monoxide, heavy metals, oxidative gases and free radicals etc,These toxic substance are nasty locally and systemically. Smoking alters the host immune response by impairing neutrophil function, reducing immunoglobulin production, and disrupting cytokine balance [10,11]. In addition, nicotine-induced vasoconstriction leads to reduced gingival blood flow, which may suppress classical signs of inflammation such as redness and bleeding on probing. This masking effect delayed the diagnosis and unfavorable to estimate the severity of disease in smokers.

Indirect effect of Smoking causes significant alterations in the oral environment, particularly in saliva. Saliva plays a vital role in maintaining oral health by facilitating lubrication, buffering acids, providing antimicrobial protection, and promoting tissue repair [16–19]. Normal pH of saliva is 6.2-7.8. Normal saliva is slightly acidic when resting and slightly alkaline when stimulated. More investigations highlight the relationship between smoking, salivary changes, and periodontal disease. Kumar et al. reported interaction between periodontitis due to changing properties of saliva like lower salivary pH ,high thiocyanate level and tobacco [13]. Long-term studies have also demonstrated that continue smoking for a long period of time leads to persistent alterations in salivary pH, which may continue even after smoking cessation [14,15].So saliva is the non-invasive biomarker and diagnostic finding to relate the periodontal disease and smoking.

Tiny amount of carbon monoxide is naturally produced in the human body-when heme is broken down by enzyme heme oxygenase, oral bacteria can generate small amount of carbon monoxide. Body constantly makes carbon monoxide in small amounts ,it exhale through both

nose and mouth. Low concentration of Carbon monoxide acts as a signaling molecule like vasodialation, anti-inflammatory, neurotransmitter.

Exhaled carbon monoxide has increasingly been recognized as a faithful and objective biomarker for assessing smoking exposure. Carbon monoxide is a toxic gas produced during incomplete combustion of tobacco and readily binds to hemoglobin, form a carboxyhemoglobin which reduce oxygen deliver to tissues. Other way of production of carbon monoxide from faulty boiler, car exhaust etc..Measurement of carbon monoxide levels in expired breath provides a simple, rapid, and non-invasive method [1,23–27]. Early studies established a strong connection between breath carbon monoxide levels and carboxyhemoglobin concentrations, supporting with the use of breath analysis give an accurate measure of smoking intensity. [23,26,30].

Deveci et al. give an idea of exhaled carbon monoxide is a biological marker of tobacco exposure on smokers compared to non smokers [1]. Middleton and Morice further supported the role of breath carbon monoxide analysis as an effective tool for identifying smoking habits in clinical and epidemiological settings [24]. Recent studies have delivered this application to passive smokers, revealing elevated carbon monoxide levels among individuals exposed to environmental tobacco smoke [31]. Breath carbon monoxide analysis has provide the objective mean of valuable in verifying self-reported smoking status, particularly in young adults and institutional populations [32].

From a periodontal perspective, elevated carbon monoxide levels may have important biological implications. Chronic exposure to carbon monoxide can induce tissue hypoxia, impair wound healing, and exacerbate periodontal destruction. Therefore, salivary parameters and exhaled carbon monoxide levels to assess the periodontal health and give a more comprehensive assessment of oral health of smokers

Methodology

Study Design and Study Population

A **cross-sectional comparative study** was conducted to evaluate the association between smoking, periodontal status, salivary pH, and exhaled carbon monoxide levels among adult participants. The study population consisted of systemically healthy individuals aged above **20 years**..

Sample Selection and Grouping

A total of **90** participants were enrolled using convenience sampling and were divided into three groups based on smoking status:

- **Group I:Smoking with periodontitis**.
- **Group II :Smoking without periodontitis**
- **Group111:Non Smoking with periodontitis**

. Self-reported smoking status was further verified using **exhaled carbon monoxide measurement**.

Inclusion Criteria

- Individuals aged above 20 years
- Systemically healthy non-smokers (Group II)
- Willingness to participate

Exclusion Criteria

- Users of smokeless tobacco
- Individuals with systemic diseases affecting periodontal status or salivary flow (e.g., diabetes mellitus, autoimmune disorders)
- Use of antibiotics, anti-inflammatory drugs, or mouth rinses within the past **three months**
- History of periodontal therapy in the last **six months**

Clinical Periodontal Examination

All participants underwent a comprehensive periodontal examination performed by a **single calibrated examiner** to eliminate inter-examiner variability. The following periodontal parameters were recorded using a **WILLIAMS PROBE**

- Probing Pocket Depth
- Clinical Attachment Level
- Bleeding on Probing
- Mobility of tooth

Periodontitis was diagnosed and classified based on standard clinical criteria, considering probing depth and clinical attachment loss.

Saliva Sample Collection

Unstimulated saliva samples were collected from all participants.Participants were instructed to quit from eating, drinking, smoking, or performing oral hygiene procedures for at least **one hour** prior to sample collection.

Patients were seated comfortably in an upright position and asked to allow saliva to accumulate in the floor of the mouth. The collected saliva samples were immediately analyzed for pH

Measurement of Salivary pH

Salivary pH was measured using a **pH paper strip**. The paper strip was immersed in the saliva sample, and the pH reading was noted using manufacture guidence.

Measurement of Exhaled Carbon Monoxide

Exhaled carbon monoxide levels were assessed using a **Bedfont EC50 Smokerlyzer handheld machine**. Participants were instructed to inhale deeply, hold their breath for **15 seconds**, and then exhale slowly into the device's mouthpiece. The CO concentration was recorded in **parts per million (ppm)**.

The measurements were performed in a well-ventilated area, and the device was calibrated according to the manufacturer's instructions.

Data Collection and Management

All clinical findings and measurements were recorded on a standardized. Data were entered into a spreadsheet and cross-checked for accuracy prior to statistical analysis.

Statistical Analysis

Statistical analysis was performed using **Statistical Package for the Social Sciences (SPSS) software**. Descriptive statistics were used to calculate mean and standard deviation for continuous variables. Comparisons between smokers and non-smokers were carried out using the **independent t-test** for normally distributed data and the **Mann–Whitney U test** for non-parametric data.

Correlation between periodontal parameters, salivary pH, and exhaled carbon monoxide levels was assessed using **Pearson's or Spearman's correlation coefficient**, as appropriate. A **p-value < 0.05** was considered statistically significant.

RESULTS

Table 1: Distribution of Salivary pH Among Study Groups

Salivary pH	Smoking with Periodontitis (n=41)	Smoking without Periodontitis (n=35)	Non-smoking with Periodontitis (n=32)
5	5	NIL	NIL
6	18	7	8
7	13	28	24

Table 1 and Graph 1 represents the distribution of salivary pH among the study groups shows notable differences. In the smoking with periodontitis group, most participants had a pH of 6 (18/41), followed by pH 7 (13/41) and pH 5 (5/41), indicating a tendency toward slightly acidic saliva. In the smoking without periodontitis group, the majority had a pH of 7 (28/35), suggesting near-neutral saliva, while fewer had pH 6 (7/35). Among non-smoking

participants with periodontitis, most had pH 7 (24/32) and pH 6 (8/32), with none showing pH 5. Overall, participants with periodontitis who smoked tended to have lower pH, reflecting a more acidic oral environment, which may contribute to disease progression.

Graph 1 . Distribution of salivary pH among the study groups

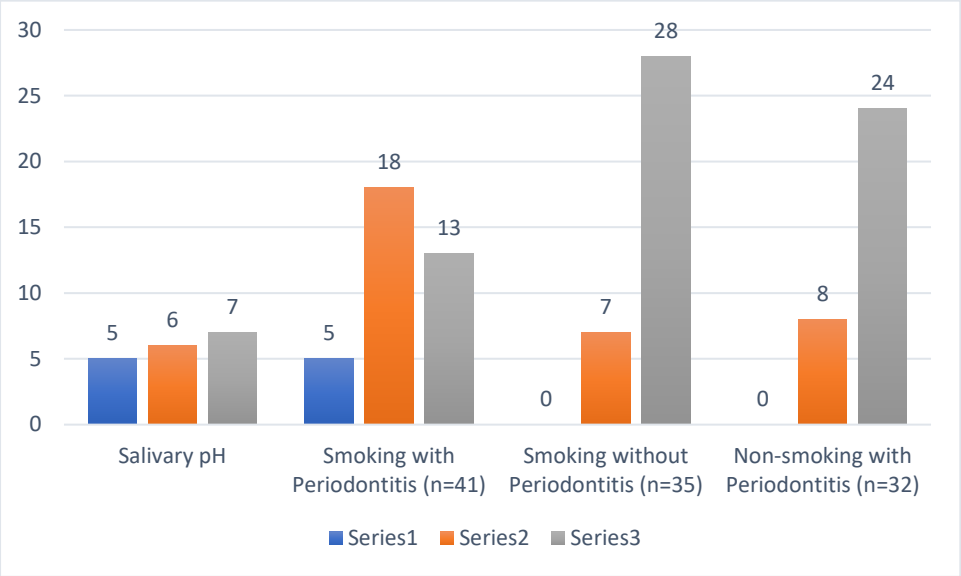

Table 2: Distribution of Breath Analyzer (PPM) Values Among Study Groups

Breath Analyzer (PPM)	Smoking with Periodontitis (n=41)	Smoking without Periodontitis (n=35)	Non-smoking with Periodontitis (n=32)
1	NIL	2	18
2	NIL	3	11
3	3	5	3
4	3	9	NIL
5	2	7	NIL
6	3	6	NIL
7	8	2	NIL
8	10	1	NIL
9	3	NIL	NIL
10	4	NIL	NIL

Table 2 and Graph 2 The distribution of breath analyzer (PPM) values reveals distinct patterns across the study groups. In the smoking with periodontitis group, higher PPM values (7–10) were more frequent, indicating increased oral volatile compounds associated with periodontal disease. Moderate values (3–6) were observed less frequently, while low values (1–2) were absent. For smoking without periodontitis, most participants had lower PPM values (1–6), with few in the higher range, reflecting better oral health despite smoking. Among non-smoking participants with periodontitis, PPM values were predominantly very low (1–3), suggesting that smoking exacerbates volatile compound accumulation. Overall, higher breath PPM levels were associated with smoking and periodontal involvement.

Graph 2 . Distribution of breath analyzer (PPM)

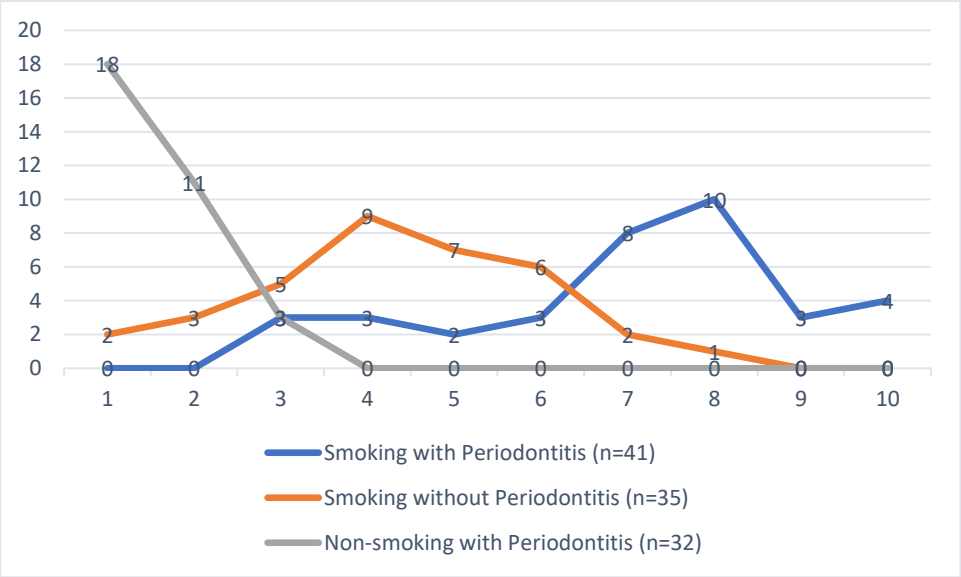

Table 3: Diagnosis Distribution Among Study Groups

Diagnosis	Smoking with Periodontitis (n=36)	Smoking without Periodontitis (n=35)	Non-smoking with Periodontitis (n=32)
Localized Periodontitis	17	NIL	25
Generalized Periodontitis	19	NIL	7
Without Periodontitis	NIL	35	NIL

Table 3 and Graph 3 represents the diagnosis distribution shows clear differences among the study groups. In the smoking with periodontitis group, participants were almost evenly divided between localized (17/36) and generalized periodontitis (19/36), indicating a high prevalence of periodontal disease among smokers. The smoking without periodontitis group had all participants (35/35) without periodontitis, highlighting that smoking alone does not

necessarily cause periodontal disease in the absence of other risk factors. Among non-smoking participants with periodontitis, most had localized periodontitis (25/32), with fewer cases of generalized disease (7/32). Overall, smoking appears to be associated with increased severity and extent of periodontal involvement.

Graph 3 Distribution of smoking with periodontitis among different groups

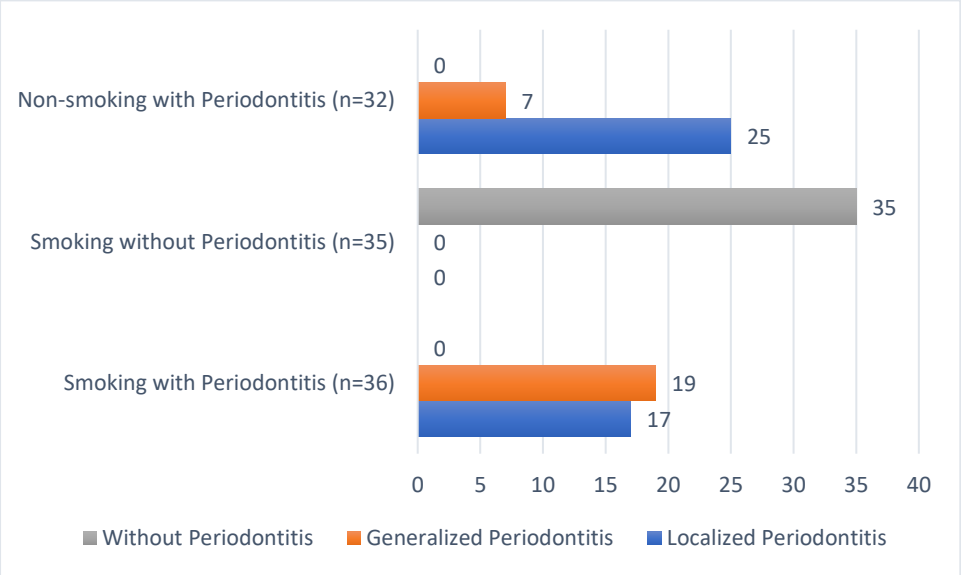

Table 4: ANOVA for Salivary pH

Group	Mean ± SD	95% CI	n	ANOVA	SIG
Smoking with Periodontitis	6.22 ± 0.68	6.00 – 6.44	36	12.99	0.000
Smoking without Periodontitis	6.80 ± 0.41	6.67 – 6.93	35		
Non-smoking with Periodontitis	6.75 ± 0.44	6.60 – 6.90	32		

Table 4 and Graph 4 represents the ANOVA analysis of salivary pH across the three study groups showed a significant difference ($F = 12.99$, $p < 0.001$). The smoking with periodontitis group had the lowest mean pH (6.22 ± 0.68), indicating a more acidic oral environment. Both the smoking without periodontitis (6.80 ± 0.41) and non-smoking with

periodontitis groups (6.75 ± 0.44) had higher, near-neutral pH levels, with overlapping 95% confidence intervals. Post hoc analysis confirmed that the smoking with periodontitis group differed significantly from the other two groups, while no significant difference existed between smoking without periodontitis and non-smoking with periodontitis. This suggests that smoking combined with periodontitis reduces salivary pH, potentially promoting disease progression.

Graph 4 ANOVA analysis of salivary pH across the three study groups

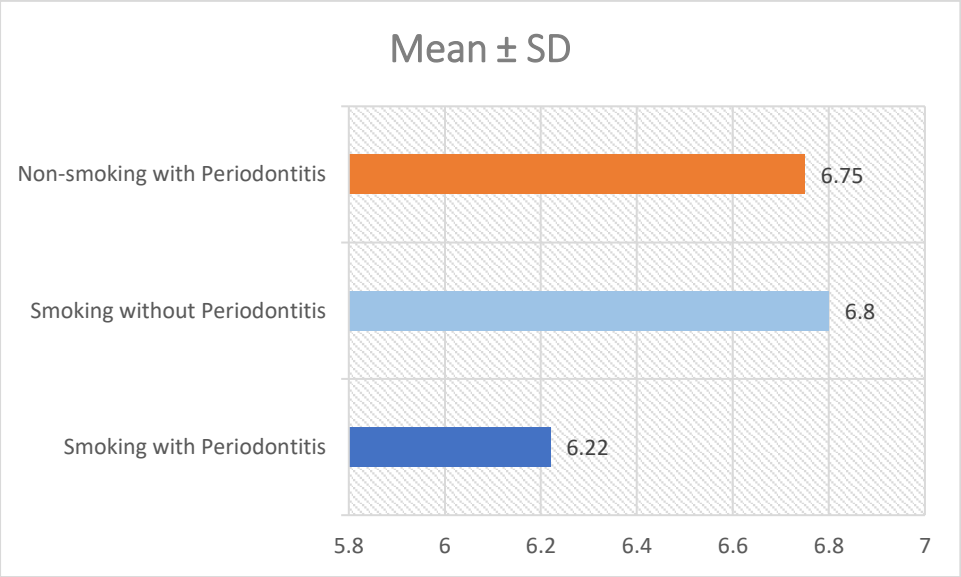

Table 5: Post Hoc Pairwise Comparison (Bonferroni-corrected)

Comparison	Mean Difference	95% CI	p value
Smoking with Periodontitis vs Smoking without Periodontitis	-0.58	-0.84 – -0.32	0.00015
Smoking with Periodontitis vs Non-smoking with Periodontitis	-0.53	-0.80 – -0.26	0.00115
Smoking without Periodontitis vs Non-smoking with Periodontitis	0.05	-0.15 – 0.25	1.0

The post hoc analysis revealed significant differences in salivary pH between groups. The smoking with periodontitis group had a significantly lower mean pH compared to both smoking without periodontitis (mean difference = -0.58, 95% CI: -0.84 to -0.32, p = 0.00015) and non-smoking with periodontitis (mean difference = -0.53, 95% CI: -0.80 to -0.26, p = 0.00115), indicating a more acidic oral environment associated with smoking and

periodontitis. No significant difference was observed between smoking without periodontitis and non-smoking with periodontitis (mean difference = 0.05, p = 1.0), suggesting that salivary pH is primarily influenced by the combined effect of smoking and periodontal disease rather than either factor alone.

Discussion

This study was conducted to evaluate the relationship between smoking and periodontitis with additional support on Salivary pH and exhaled carbon monoxide level. The findings of this study support existing evidence that tobacco smoking exerts harmful effect on periodontal health and significantly alters the oral environment. Smokers exhibit poor periodontal health, lower salivary pH, and higher levels of exhaled carbon monoxide when compared with non-smokers.

According with the earlier studies, the present findings firmly established association between smoking and periodontal disease severity. Bergstrom and colleagues reported that smokers are at a higher risk for chronic destructive periodontal disease and exhibit greater attachment loss and alveolar bone destruction than non-smokers [4,5,9]. Similar observations were reported by Haber et al. and Johnson and Hill, who identified smoking as a major independent risk factor for periodontitis, regardless of oral hygiene status [6,10]. Lower bleeding on probing, although periodontal destruction present, due to vasoconstriction that reduce inflammatory reaction.

The altered host immune response in smokers plays a crucial role in periodontal breakdown. Tobacco smoke impairs neutrophil, chemotaxis and phagocytosis, disrupts cytokine balance, and compromises antibody production, resulting in diminished host defense against periodontal pathogens. These impaired reaction increase the periodontal destruction and cause poor prognosis .

Salivary analysis in this present study revealed lowered salivary pH values among smokers compared to non-smokers. Finding from this study agree with previous studies, that reported reduced salivary pH and flow rate in individuals with long-term tobacco exposure [3,12,15,21,22]. Singh et al. observed that chronic smokers exhibit a shift toward acidic salivary pH, which may enhance microbial colonization and promote periodontal inflammation [3].Acidic saliva appealing the growth for periodontal micro-oragnism and progress the destruction of periodontitis.

Furthermore, studies by Kumar et al. and Grover et al. demonstrated that smokers with chronic periodontitis show significantly lower salivary pH and altered salivary composition compared with non-smokers [13,15]. Tobacco induced altered salivary pH with dental plaque biofilm increase the progression of periodontitis.

From this study Exhaled carbon monoxide levels were significantly higher among smokers, so its consider a objective biomarker of smoking exposure. These findings are hardset with earlier studies by Deveci et al., Middleton and Morice, and Wald et al., who demonstrated a strong correlation between smoking intensity and breath carbon monoxide levels [1,24,26]. The elevated carbon monoxide levels observed in smokers reflect recent tobacco exposure and provide a non-invasive method to validate self-reported smoking habits.

From a periodontal perspective, decrease oxygen delivery through the formation of carboxyhemoglobin by the chronic exposure to carbon monoxide cause tissue hypoxia. Hypoxic conditions within periodontal tissues can impair fibroblast function, enhance connective tissue breakdown, delay wound healing, and thereby aggravating periodontal destruction. Although direct clinical evidence linking carbon monoxide levels to periodontal damage remains limited, the findings of the present study suggest a possible association between elevated exhaled carbon monoxide and poor periodontal health in smokers.

The integration of periodontal assessment with salivary pH and exhaled carbon monoxide measurement offers a more comprehensive understanding of the biological impact of smoking on oral health. Saliva and breath analysis are simple, non-invasive, and patient-friendly diagnostic tools that may help in early detection of smoking-related periodontal changes. For supporting smoking cessation counseling and monitoring the patient quietness objective biomarker such as exhaled carbon monoxide can be effectively used

.

Conclusion

Within the limitations of the present study, it can be concluded that **smoking has a significant adverse effect on periodontal health**. Compare to non-smoker, smokers exhibit higher level of exhaled carbon monoxide, **lower salivary pH** ,poor periodontal parameters. These findings indicate that tobacco smoking not only directly damages periodontal tissues but also alters salivary properties and induces tissue hypoxia, thereby creating a biologically unfavorable environment for periodontal health.

The assessment of **salivary pH and exhaled carbon monoxide** serves as a valuable adjunct to conventional periodontal examination and may aid in early identification of individuals at increased risk for smoking-related periodontal disease. So consolidate these non-invasive diagnostic tools into routine clinical practice may enhance periodontal risk assessment and reinforce smoking cessation strategies.

Reference

1.The measurement of exhaled carbon monoxide in healthy smokers and non-smokers S. Erhan Devecia, Figen Devecib, Yasemin Acik, A.Tevfik Ozana, Respiratory medicine(2004)98,551-556

2.RIVERA-HIDALGO F. Smoking and periodonlal disease. A review of the literature. J Periodontoi 1986; 57: 617-24

3. Singh M, Ingle NA, Kaur N, et al. Effect of long-term smoking on salivary flow rate and salivary pH. J Indian Assoc Public Health Dent 2015; 13: 11

4.Cigarette smoking as risk factor in chronic periodontal disease J Bergstrom. Community Dent Oral Epidemiol. 1989 Oct;17(5):245-7

5.Bergstrom J: Tobacco smoking and risk for periodontal disease. J Clin Periodontol Blackwell 2003; 30: 107–113

6. Haber, J., Wattles, J., Crowley, M., Mandell, R.,Joshipura, K. & Kent, R. L. (1993) Evidence for cigarette smoking as a major risk factor for periodontitis. Journal of Periodontology 64, 16–23.

7. Ismail II, Burt BA, Eklund SA. Epidemiologic patterns of smoking and periodontal disease in the United States. J Am Dent Assoc 1983;106:617-623.

8. Bergstrom J, Eliasson S, Dock J: Exposure to tobacco smoking and periodontal health. J Clin Periodontol 2000; 27: 61–68

9. Bergström J. Tobacco smoking and chronic destructive periodontal disease. Odontology. 2004;92(1):1–8.

10.Johnson GK, Hill M.Cigarette smoking and the periodontal patient. J Periodontol. 2004;75(2):196–209.

11. Reibel J. Tobacco and oral diseases. Med Princ Pract. 2003;12(Suppl 1):22–32.

12. Fazail Ahmad, Aarati Panchbhai, Impact of Tobacco on Salivary Flow Rate and Salivary pH, J Res Med Dent Sci, 2022,10 (10): 235-238.

13. Assessment of salivary thiocyanate levels and pH in the saliva of smokers and nonsmokers with chronic periodontitis - A comparative study C Naresh Kumar et al. Indian J Dent Res. 2021 Jan-Feb.;32(1):74-78

14. Long-Term Effect of Smoking on the Salivary pH Corresponding Author: Hussein Haleem Jasim Department of Oral Diagnosis, College of Dentistry, University of Wasit, Iraq Article Received: 01-June-2024, Revised: 21-June-2024, Accepted: 11-July-2024

15. Grover N, Sharma J, Sengupta S, et al. Long-term effect of tobacco on unstimulated salivary pH. J Oral Maxillofac Pathol. 2016;20(1):16-19.

16. Sreebny LM. Saliva in health and disease: An appraisal and update. Int Dent J. 2000;50:140–161

17.Gordon B. Proctor.The Physiology of salivary secretion. Periodontology 2000,vol 70,2016,11-25

18. Dodds MW, Johnson DA, Yeh CK. Health benefits of saliva: a review. J Dent 2005: 33: 223–233.

19. Vila, T.; Rizk, A.M.; Sultan, A.S.; Jabra-Rizk, M.A. The power of saliva: Antimicrobial and beyond. PLoS Pathog. 2019, 15, e1008058

20. Saputri, D., Nasution, A. I., Surbakti, M. R. W., & Gani, B. A. (2017). The correlation between pH and flow rate of salivary smokers related to nicotine levels labelled on cigarettes.Dental Journal,50(2) 61-65

21. Petrušić N, Posavac M, Sabol I, Mravak-Stipetić M. The effect of tobacco smoking on salivation. Acta Stomatol Croat. 2015; 49(4): 309–15

22. Kanwar A, Sah K, Grover N, Chandra S, Randeep Singh R. Long-term effect of tobacco on resting whole mouth salivary flow rate and pH: An institutional based comparative study. Eur J Gen Dent 2013;2(3):296-9

23. Ringold A, Goldsmith JR, Helwig HI, Finn R, Schoutte F.Estimating recent carbon monoxide exposures. Arch Environ Health 1962;5:308–18.

24. Middleton ET, Morice AH. Breath carbon monoxide as an indication of smoking habit. Chest 2000;117:758–63.

25. Breath analysis to detect recent exposure to carbon monoxide A J Cunnington et al. Postgrad Med J. 2002 Apr.

26. Wald NJ, Idle M, Boreham J, Bailey A. Carbon monoxide in breath in relation to smoking and carboxyhaemoglobin levels. Thorax 1981;36(5):366–9.

27. Cunnington AJ, Hormbrey P. Breath analysis to detect recent exposure to carbon monoxide. Postgrad Med J 2002;78:233–7.

28. Jimenez-Ruiz CA, Masa F, Miravitlles M, et al. Smoking characteristics. Chest 2001;119:1365–70.

29. Smoking practices in relation to exhaled carbon monoxide in an occupational cohort BMC Public Health December 2020.20(1)

30. Carbon monoxide in breath in relation to smoking and carboxyhaemoglobin levels.N J Wald, M Idle, J Boreham, A Bailey 1981 May;36(5):366–369

31. Evaluation of Exhaled Carbon Monoxide Levels in Individuals Exposed to Passive Tobacco Smoke in Indoor and Outdoor Environments: How Far Can We Getaway Under the Same Roof?Dursun E Afşin et al. Cureus. 2023 Sep 11;15(9)

32. Evaluation of smoking status: comparison of self-reports with exhaled carbon monoxide analysis in university students in the Islamic Republic of Iran Arash Nikkholgh et al. East Mediterr Health J. 2021.