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Evaluation of Glycosylated Hemoglobin Levels and Effect of Tobacco Smoking in Periodontally Diseased Non-Diabetic Patients

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ABSTRACT

Background and Objective: Chronic diseases have progressively increased worldwide, impacting all areas and socioeconomic groups. Periodontal disease is an increasing global concern and contains risk factors similar to other chronic illnesses. The main risk factor for periodontitis is smoking. Smoking not only hastens periodontal disease but also complicates periodontal therapy. Serum glycosylated hemoglobin levels, which are derived from the average life span of an erythrocyte, are a good indicator of glycemic management during the preceding one to three months. This study was undertaken to assess the association between tobacco smoking and periodontal disease by evaluating plaque score, gingival score, extent and severity index (ESI), and glycemic status by estimating serum HbA1c in cigarette smoker patients compared to non-smokers.

Methods: The study was conducted with 40 patients in the age range of 20–40 years. Patients were divided into two groups: non-smokers (Group I) and cigarette smokers (Group II). Periodontal clinical parameters such as the plaque index (PI), gingival index (GI), and ESI were recorded during the oral cavity examination. The biochemical marker, serum glycosylated hemoglobin, was measured in both groups. All parameters were measured at baseline and three months after periodontal therapy. The statistical tests used were the paired *t-test*, and *Chi-square* test for comparison between both groups.

Results: The mean difference of PI of non-smokers was 0.33 ± 0.30, and smokers were 0.52 ± 0.32, which was statistically significant. The mean difference of GI of non-smokers was 0.34 ± 0.19 and smokers 0.36 ± 0.303, which was statistically significant. The mean difference of extent in non-smokers was 5.33 ± 1.59, 5.52 ± 2.43, and smokers were 0.18 ± 0.17. The mean difference in severity in non-smokers was 0.18 ± 0.17, and smokers were 0.31 ± 0.25, which was statistically significant. The mean difference of HbA1c in non-smokers and smokers was 0.43 ± 0.277 and 0.415 ± 0.230, which shows a higher mean difference in non-smokers, which was statistically non-significant.

Conclusion and Global Health Implications: This study concluded that each of Group I and Group II showed substantial improvements in all clinical periodontal variables, which include plaque index (PI), gingival index (GI), extent and severity index (ESI), and biochemical marker serum glycosylated hemoglobin. Controlling inflammation with SRP can improve insulin resistance, lower glucose levels, and prevent non-enzymatic glycation of hemoglobin.

Keywords: Diabetes Mellitus, Glycosylated Hemoglobin, Periodontitis, Tobacco Smoking, Non-Smokers

INTRODUCTION

Background of the Study

As one of the most common non-communicable diseases, periodontal disease is caused by poor dental hygiene, smoking, stress, alcohol use, and systemic disorders.[1] According to the World

Health Organization (WHO), Non-communicable diseases (NCDs) accounted for 74% of the fatalities globally in 2019. Socioeconomic shifts are the primary cause of the global diabetes epidemic, with population expansion, bad eating habits, and a shift to a sedentary lifestyle also contributing risk factors.[2] Of the population of India, 77 million have prediabetes, and 62.4 million have type 2 diabetes mellitus.[3] The overall frequency of periodontal disease was 51%, and gingivitis was 46.6%. The average incidence of mild-to-moderate periodontitis was 26.2%, whereas severe periodontitis was 19%.[4]

The group of localized infections known as periodontal disease affects the tissues that support teeth and form the periodontium. Periodontal disease is a term that encompasses both irreversible (periodontitis) and reversible (gingivitis) processes. Inflammatory reactions to infections and damaging substances are undoubtedly the pathogenic basis of periodontal disease. It has been suggested that periodontal disease is the sixth complication of diabetes, and smoking has a negative impact on the clinical result of both nonsurgical and surgical therapy.[5,6] Glycated hemoglobin, or HbA1c, is a measure of the average blood glucose level over the two to three months prior and is used as an indicator of long-term glucose homeostasis.[7] Glycosylation, also referred to as glycation, is the non-enzymatic addition of carbohydrates to polypeptides and proteins that results in the creation of advanced glycated end products (AGEs).[8] It has been demonstrated, according to a number of researchers, that nicotine, which is present in cigarettes, increases blood sugar.[9] Through the alveoli, tobacco's most potent ingredient enters the lungs.[10] Nicotine is linked to increased insulin resistance, which impairs glucose tolerance, in addition to its direct toxic effect on pancreatic beta cells. In addition, nicotine's anti-estrogenic properties could increase the build-up of visceral adipose tissue, and consequently, insulin resistance. Finally, nicotine affects adiponectin, a peptide that controls body weight and food intake, as well as raising cortisol and inflammatory levels, all of which may lead to an elevated HbA1c. Nicotine has a strong addictive potential. Increased heart rate, blood pressure, respiration, and peripheral vasoconstriction can all result from it. This can then contract mouth capillaries, impacting gingival and periodontal tissue blood flow.[6] Studies have shown that smoking can exacerbate the negative effects of elevated HbA1c levels on periodontal health. Smoking combined with poorly controlled diabetes can create a more hostile oral environment, leading to more severe periodontal disease progression. Overall, there is a complex interplay between smoking, HbA1c levels, and periodontitis, with each factor influencing the others and contributing to the overall risk of developing periodontal disease. It is important for healthcare providers to consider these relationships when assessing and managing the oral health of individuals, especially those with diabetes who smoke.[11]

Objectives of the Study

The relationship between smoking and periodontal disease was explained in this study. Furthermore, it was aimed at assessing the degree of periodontal disease in patients, who did not smoke compared to smokers, by looking at their gingival index (GI) and plaque index (PI) scores, extent and severity index (ESI), and glycemic status by estimating serum HbA1c in non-smokers compared to cigarette-smoking patients.

METHODS

A total of 40 patients with an age range of 20–40 years were included in the study. Patients were divided into two groups: Group I included patients with chronic periodontitis who did not smoke, and Group II included patients with chronic periodontitis who smoked. A thorough explanation of the treatment approach was given to each patient, and both, verbal and informed written consent were obtained.

The inclusion criteria were: Patients with chronic periodontitis, who did not smoke and who smoked, who had not undergone any periodontal therapy in the six months prior to the study, in the age range of 20–40 years. The exclusion criteria were: The patients who had underlying systemic disease and were medically compromised.

***Clinical Procedure*:** Twenty patients were included in both groups. Root planning and scaling were administered to every patient in both groups at baseline. An ultrasonic scaler was used for supra- and subgingival scaling, and Gracey curettes were used for root planning. Oral hygiene instructions were given to patients in both groups. Patients were instructed to use a 0.2% chlorhexidine gluconate solution to rinse and brush their teeth twice a day. Every recall visit involved an evaluation of oral hygiene reinforcement. Each patient had a venous blood sample drawn from the antecubital fossa in order to test the baseline and three-month post-treatment HbA1c values. Patients in the smoking group were urged to stop smoking, and instructions were given to maintain oral hygiene. Before and after non-surgical periodontal treatment, patients were scheduled for periodontal examinations and laboratory.

Ethical Approval

The study was conducted in the Department of Periodontology at Seema Dental College & Hospital in Rishikesh, Uttarakhand, India, with approval from the institutional ethical committee

2016/A-115 dated December 12, 2016. The duration of the study was three months.

Statistical Analysis

The obtained data was recorded in Microsoft Excel 2019, and statistical analysis was conducted with the Statistical Package for the Social Sciences (SPSS) version 25 (SPSS, Inc., Chicago, IL, USA) software. Descriptive analyses, i.e., the mean and standard deviations (SDs), were calculated for GI, PI, ESI, and serum glycosylated hemoglobin. The statistical tests used were paired *t-test, and Chi-square* test for comparison of both groups.

The sample size was calculated, and the study took into account the 95% confidence interval (CI) and 95% power. In order to obtain 80% power and 5% significance, a sample size of 40 was determined.

Study Variables

Clinical and Biochemical Parameters Assessed*:* The following parameters were evaluated at baseline and three months after starting periodontal therapy: PI described by Silness and Loe in 1964,[12] GI, described by Loe and Sillness in 1963,[13] ESI, described by Carlos in 1986[14] were assessed for both groups. Serum levels of glycosylated hemoglobin were assessed in both groups. All the parameters were assessed at baseline and at three months.

RESULTS

Demographic Details

The mean age with a standard deviation of non-smokers was 37 ± 2, and for smokers, it was 40 ± 2 with a *P*-value ≥0.05, which was statistically non-significant. There was no statistically significant difference in gender between the two groups, as shown in Table 1.

Clinical Parameters Results

Plaque Index: The mean values of PI in non-smokers at baseline was 1.87 ± 0.49 and at three months, 1.54 ± 0.31 with a mean difference of 0.33 ± 0.30, which was statistically significant with a *t* value of 4.939 as shown in Table 2. The mean values of PI in smokers at baseline was 2.25 ± 0.363 and at three months, 1.72 ± 0.28 with a mean difference of 0.52 ± 0.32, which was statistically significant with a t value of 7.12, as shown in Table 2. The mean difference of PI in non-smokers and smokers was 0.332 ± 0.3006 and 0.52 ± 0.32, respectively, which shows a higher mean difference in smokers with a *t* value of -1.919, which was statistically non-significant.

Table 1: Demographic details in Group I and Group II.

Variable	Non-smokers	Smokers	Mean	*p*-value
Age (Mean ± SD)	37 ± 2	40 ± 2	38.5 ± 2	≥0.05*
Gender (Male: Female)	15:13	17:14	16:13	≥0.05*

SD-Standard deviation.
**p*-value - non-significant.

Gingival Index: The mean values of GI in non-smokers at baseline was 2.23 ± 0.29 and at three months, 1.88 ± 0.36 with a mean difference of 0.34 ± 0.19, which was statistically significant with a *t* value of 7.85 as shown in Table 2. The mean values of GI in smokers at baseline was 1.94 ± 0.407 and at three months, 1.58 ± 0.27 with a mean difference of 0.36 ± 0.303, which was statistically significant with a t value of 5.327, as shown in Table 2. The mean difference of GI in non-smokers and smokers was 0.348 ± 0.198 and 0.36 ± 0.303, respectively, which shows a higher mean difference in smokers with a *t* value of -0.167, which was statistically non-significant.

Extent: The mean values of extent in non-smokers at baseline was 84.20 ± 11.62, and at three months, 78.87 ± 11.69 with a mean difference of 5.33 ± 1.59, which was statistically significant with a *t* value of 14.9 as shown in Table 2. The mean values of extent in smokers at baseline was 90.64 ± 6.61 and at three months 85.12 ± 7.22 with a mean difference of 5.52 ± 2.43, which was statistically significant with a *t* value of 10.16 as shown in Table 2. The mean difference of extent in non-smokers and smokers was 5.33 ± 1.59 and 5.527 ± 2.43, respectively, which shows a higher mean difference in smokers with a *t* value of -0.303, which was statistically non-significant.

Severity: The mean values of severity in non-smokers at baseline was 3.52 ± 0.53, and at three months, 3.33 ± 0.54 with a mean difference of 0.18 ± 0.17, which was statistically significant with a *t* value of 4.949 as shown in Table 2. The mean values of severity in smokers at baseline were 4.01 ± 0.61 and at three months, 3.70 ± 0.57 with a mean difference of 0.31± 0.25, which was statistically highly significant with a t value of 5.53, as shown in Table 2. The mean difference of severity in non-smokers and smokers was 0.188 ± 0.170 and 0.313 ± 0.253, respectively, which shows a higher mean difference in smokers with a *t* value of -1.831, which was statistically non-significant.

Biochemical Parameter Result

Serum Glycosylated Serum: The mean values of HbA1c in non-smokers at baseline was 5.43 ± 0.39 and at three months, 5.005 ± 0.27 with a mean difference of 0.43 ± 0.27, which was

Table 2: A statistical comparison of the mean values of clinical periodontal parameters at different intervals between Group-I (non-smokers) and Group-II (smokers).

Parameter	Non-smokers					Smokers			
	Different interval	Mean ± SD	Mean difference	t	*p*-value	Mean ± SD	Mean difference	t	*p*-value
Plaque index	Baseline	1.87 ±0.50	0.33 ±0.30	4.939	**≤0.001***	2.25±0.363	0.52 ± 0.32	7.12	**≤0.001***
	3 months	1.54 ±0.31				1.72 ±0.28			
Gingival index	Baseline	2.23 ±0.29	0.34 ± 0.20	7.85	**≤0.001***	1.94 ±0.407	0.36 ± 0.303	5.327	**≤0.001***
	3 months	1.88 ±0.37				1.58 ±0.27			
Extent	Baseline	84.20±11.63	5.33 ± 1.59	14.9	**≤0.001***	90.64 ±6.61	5.52 ± 2.43	10.16	**≤0.001***
	3 months	78.87±11.69				85.12 ±7.22			
Severity	Baseline	3.52 ± 0.53	0.18 ± 0.17	4.949	**≤0.001***	4.01 ±0.61	0.31 ± 0.25	5.53	**≤0.001***
	3 months	3.33 ± 0.55				3.70 ±0.57			

SD - Standard deviation.
**p*-value – significant.

Table 3: Comparing the means of clinical periodontal markers and HbA1c at different time points between the smokers and non-smoker groups.

Parameter	Non-smokers					Smokers			
	Different interval	Mean ± SD	Mean difference	t	*p*-value	Mean ± SD	Mean difference	t	*p*-value
Glycosylated hemoglobin	Baseline	5.43 ± 0.40	0.43 ± 0.27	6.89	<0.001*	5.95 ± 0.604	0.40 ± 0.23	8.069	<0.001*
	3 months	5.005 ± 0.27				5.53 ± 0.457			

SD - Standard deviation,
**p*-value – significant.

statistically highly significant with *t* value of 6.93 as shown in Table 3. The mean values of HbA1c in smokers at baseline was 5.95 ± 0.604, and at three months, 5.53 ± 0.457 with a mean difference of 0.41 ± 0.23, which was statistically highly significant with a *t* value of 8.068 as shown in Table 3. The mean difference of HbA1c in non-smokers and smokers was 0.43 ± 0.277 and 0.415 ± 0.230, respectively, which shows a higher mean difference in non-smokers with a *t* value of 0.186, which was statistically non-significant.

DISCUSSION

Glycohemoglobin is created when the amino groups of the lysine and valine residues of hemoglobin combine with glucose through a non-enzymatic process. The amount of the protein glycohemoglobin in red blood cells is determined by blood glucose levels because it is an irreversibly generated protein.[15] In this study, baseline and three-month glycemic status were measured for non-smokers and smokers, with smokers having higher mean values. Forty patients in the age range from 20 to 40 years were divided into two groups: non-smokers and smokers. Non-surgical periodontal therapy was administered to each patient in both groups. The clinical indicators were measured at baseline and three months after the initiation of periodontal therapy. These indicators allow for the monitoring of past periodontal destruction, ongoing illness, and the prognosis of disease development, which makes them essential for treatment planning and outcome evaluation. In both groups, serum levels of glycosylated hemoglobin were measured at baseline and three months after periodontal therapy. Smoking affects the efficacy of active periodontal therapy and has a detrimental effect on treatment outcomes. Smokers had lower gingival scores because nicotine increases vasoconstriction of peripheral blood vessels and reduces the clinical indications of gingival inflammation.

Indurkar recorded the clinical results of non-surgical periodontal therapy in patients with chronic periodontitis and concluded a decline in PI and GI scores over a six-week period.[16] Three months following non-surgical periodontal therapy, this study demonstrated a significant reduction in both PI and GI in non-smokers. A decrease in the non-smokers' scores did not seem to have any statistically significant impact on PI or GI when the mean difference between the groups was evaluated. Aziz *et al.* evaluated the effects of scaling and root planing in the short term on clinical parameters and systemic inflammatory and oxidative stress markers in smokers and non-smokers.[17] The findings demonstrated that compared to non-smokers, smokers had

greater rates of systemic inflammation and oxidative stress, as well as periodontal damage. This finding suggests that non-smokers maintained optimal hygiene throughout the course of the study. The ESI was used in the study to evaluate the degree and severity of periodontal disease in smokers and non-smokers. Since the ESI had not been thoroughly investigated, the findings have been contrasted with those of similar studies. Carlos *et al.* and Thomson *et al.* discovered that the level of periodontal damage was significantly higher in smokers.[14,18] Carlos *et al.* assessed the extent and severity indexes of 369 persons, aged 17–32 years. For each damaged site, 22% of the research locations had attachment loss with a mean severity of 1.48 mm. The severity and scope of the illness had considerably worsened, both over the preceding three years and when the condition was re-examined about three years later.[14]

In this study, the glycemic status of smokers and non-smokers was measured at baseline and three months later, with smokers showing higher mean values. According to Ohkuma *et al.,* the amount of cigarettes smoked each day and the rise in glycosylated hemoglobin were correlated in a dose-response manner.[15,19] Glycated hemoglobin in both groups decreased statistically significantly three months after non-surgical periodontal therapy, according to a study by Vaghani *et al.*[20] Before and after non-surgical periodontal therapy, the levels of glycosylated hemoglobin in healthy individuals and those with periodontitis were compared by Muthu *et al.*[21] and Vaghani *et al.*[20] After three months of non-surgical periodontal therapy, patients with periodontitis showed significantly lower levels of glycosylated hemoglobin. When Muthu *et al.* examined the impact of non-surgical periodontal therapy on glycemic control in patients with periodontitis, they found that their HbA1c was higher than that of healthy controls.[21] Three months after the initiation of periodontal therapy, HbA1c levels dropped, and periodontal parameters improved. According to a Verma study, there was no statistically significant difference in the mean HbA1c values between cigarette and bidi smokers and no correlation between the smoking index and HbA1c.[22] Nevertheless, Group II (smokers) reported a statistically significant decrease in HbA1c three months following non-surgical periodontal therapy.

In a 2013 study, Jyothirmayi *et al.* examined the relationship between glycated hemoglobin levels and cigarette smoking and concluded a correlation between the two.[10] When evaluating the impact of cigarette smoking on the severity of periodontitis in older Thai individuals, Torrungruang *et al.* found a similar conclusion; those who had smoked in the past were more likely to have severe periodontitis than non-smokers.[23] Ali *et al.* evaluated the clinical parameters PI, GI, probing depth, and clinical attachment level in order to compare smokers and non-smokers. The author concluded that smokers had lower GI index scores and more plaque.[24] A study by Urberg *et al.* found that smoking negatively affects plasma glucose levels. The author concluded that smokers had higher average blood glucose than non-smokers when they compared the levels of glycosylated hemoglobin in patients who smoked one pack or more per day with those of non-smokers.[25]

The decline in the non-smokers' assessments on PI or GI did not seem to have any statistically significant impact based on the mean difference between the groups. This implies that non-smokers maintained optimal levels of hygiene throughout the study. Because of this, bacteria were able to multiply and survive in the intricate biofilm ecology while generating a range of virulence factors that allow them to evade the host immune system and cause the destruction of human periodontal tissues.[26] Consequently, periodontal disease diagnosis and therapy were necessary for the management of patients who were susceptible to periodontitis.[27,28] In order to validate the findings of this investigation, further comprehensive research utilizing a larger sample size and follow-up was necessary. Christine Patramurti proposed that the interaction between the number of cigarettes smoked and smoking duration was strongly and independently connected with the risk of T2DM in smokers, as demonstrated by HbA1c.[29] Smoking affects the periodontium in healthy people, but it exacerbates the damage in smokers with diabetes, despite the minimal influence of diabetes on periodontium health.[30]

CONCLUSION AND GLOBAL HEALTH IMPLICATIONS

The outcomes of this study showed that both groups' clinical periodontal parameters and blood glycosylated hemoglobin levels improved following non-invasive periodontal therapy. Compared to the smoking group, the non-smoking group had better treatment outcomes. Controlling inflammation with SRP can improve insulin resistance, lower glucose levels, and prevent non-enzymatic glycation of hemoglobin.

Key Messages

- The interplay between tobacco smoking, glycosylated hemoglobin, and periodontal health underscores the importance of addressing smoking cessation to mitigate the risk of periodontal diseases, particularly in individuals with diabetes.
- The periodontal therapy outcomes are affected in both non-smokers and smokers with chronic periodontitis
- Tobacco smoking has a detrimental effect on periodontal health, as evidenced by a significant correlation with an increase in serum glycosylated hemoglobin levels.

- The elevated levels of glycosylated hemoglobin indicate compromised glycemic control, exacerbating the inflammatory response in periodontal tissues.

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COMPLIANCE WITH ETHICAL STANDARDS

Conflicts of Interest

There are no conflicts of interest.

Financial Disclosure

Nothing to declare.

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Ethics Approval

The research/study was approved by the Institutional Review Board at Seema Dental College, number 2016/A-115, dated December 12, 2016.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent.

Use of Artificial Intelligence (AI)-Assisted Technology for Manuscript Preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

Disclaimer

None.

REFERENCES

1. Shah R, Singh A, Singh HM, Gupta R, Ghosh S. Effect of tobacco smoking on the periodontal health in Parsa, Nepal: A comparative cross-sectional study. J Oral Res Rev. 2024:16;1–5.
2. Pradeepa R, Mohan V. Epidemiology of type 2 diabetes in India. Indian J Ophthalmol. 2021 Nov;69(11):2932–8.
3. Mohan V, Shah S, Saboo B. Current glycemic status and diabetes related complications among type 2 diabetes patients in India: Data from the A1chieve study. J Assoc Physicians India. 2013 Jan;61(1 Suppl):12–5.
4. Janakiram C, Mehta A, Venkitachalam R. Prevalence of periodontal disease among adults in India: A systematic review and meta-analysis. J Oral Biol Craniofac Res. 2020 Oct-Dec;10(4):800–6.
5. Martinez AB, Perez PM, Bermejo ME, Moles MAG, Ilundain JB, Meurman JH. Periodontal disease and diabetes - review of the literature. Med Oral Patol Oral Cir Bucal. 2011 Sep 1;16(6):e722–9.
6. Torkzaban P, Hedayatipanah M. Comparison of periodontal conditions between smokers and non smokers. Avicenna J Dent Res. 2016;8:1–5.
7. Thorstensson H, Hugoson A. Periodontal disease experience in adult long-duration insulin-dependent diabetics. J Clin Periodontol. 1993 May;20(5):352–8.
8. Radvar M, Darby I, Polster A, Arashi M, Moeintaghavi A, Sohrabi K. Pattern of cigarette smoking effect on periodontal pocketing and attachment loss: A retrospective study. Int J Dent Hygiene. 2011 Nov;9(4):291–5.
9. Vidhya K. The effects of cigarette smoking on glycosylated hemoglobin in non-diabetic individuals. Int J Adv Res. 2015;12:566–71.
10. Jyothirmayi B, Kaviarasi S, William E. Study of glycated hemoglobin in chronic cigarette smokers. Int J Pharma Clin Res 2013;5:4–6.
11. Gupta S, Maharjan A, Dhami B, Amgain P, Katwal S, Adhikari B, Shukla A. Status of tobacco smoking and diabetes with periodontal disease. JNMA J Nepal Med Assoc. 2018 Sep-Oct;56(213):818–24.
12. Silness J, Loe H. Periodontal disease in pregnancy. Correlation between oral hygiene and periodontal condtion. Acta Odontol Scand. 1964 Feb;22:121–35.
13. Loe H, Silness J. Periodontal disease in pregnancy. I. prevalence and severity. Acta Odontol Scand. 1963 Dec;21:533–51.
14. Carlos JP, Wolfe MD, Kingman A. The extent and severity index: A simple method for use in epidemiologic studies of periodontal disease. J Clin Periodontol. 1986 May;13(5):500–5.
15. Saleh J. Glycated hemoglobin and its spinoffs: Cardiovascular disease markers or risk factors? World J Cardiol. 2015;7:449–53.
16. Indurkar MS, Bhailume PS. The effect of mechanical nonsurgical periodontal therapy in chronic periodontitis: A clinical study. Int J Dent Clinics. 2015;7:a–d.
17. Aziz AS, Kalekar MG, Benjamin T, Suryakar AN, Patil S, Bijle MNA. Short-term effectiveness of scaling and root planing on periodontal parameters, systemic inflammatory, and oxidative stress markers in smokers with chronic periodontitis. J Oral Health. 2015;7:1–8.
18. Thomson KFR, Do LG, Bartold PM, Daniels J, Grosse A, Meihubers S. Prevalence, extent and severity of severe periodontal destruction in an urban Aboriginal and Torres Strait Islander population. Aust Dent J. 2014 Mar;59(1):43–7.
19. Ohkuma T, Iwase M, Fujii H, Kaizu S, Ide H, Jodai T *et al*. Dose and time dependent association of smoking and its cessation with glycemic control and insulin resistance in male patients with type 2 diabetes mellitus: The Fukuoka Diabetes Registry. PLoS One. 2015 Mar 30;10(3):e0122023
20. Vaghani H, Mehta R, Desai K, Duseja S, Mehta T. Effect of non-surgical periodontal therapy on glycosylated haemoglobin levels in diabetics and non-diabetic healthy controls with periodontitis. Adv Hum Biol. 2016;6:95–8.
21. Muthu J, Muthanandam S, Mahendra J, Namasivayam A, John L, Logaranjini A. Effect of nonsurgical periodontal therapy

on the glycemic control of non-diabetic patient: A clinical biochemical study. Oral Health Prev Dent. 2015;13(3):261–6.
22. Verma N. A Study of HbA1c, fasting and 2 hour plasma glucose levels in current smokers presenting at a tertiary care hospital in North India. Int J Adv Med. 2018;5:38–46.
23. Torrungruang K, Nisapakultorn K, Sutdhibhisal S, Tamsailom S, Rojanasomsith K, Vanichjakvong O, *et al*. The effect of cigarette smoking on the severity of periodontal disease among older Thai adults. J Periodontol. 2005 Apr;76(4):566–72.
24. Ali OH, Ali S, Mazin H, Salah R. The effect of smoking on periodontal disease. Int J Adv Biological Res. 2017;7:74–7.
25. Urberg M, Shammas R, Rajdev K. The effects of cigarette smoking on glycosylated hemoglobin in nondiabetic individuals. J Fam Pract. 1989 May;28(5):529–31.
26. Gaind S, Khatri M, Bansal M, Puri K, Rehan M, Mehrotra S. Host modulation as a therapeutic strategy. Int J Appl Dent Sci. 2021;7:473–6.
27. Aeran H, Tuli A, Gaind S. Periodontal referral patterns for general dentist. Int J Oral Health Dent. 2019;5(1):7–9.
28. Rana MP, Gaind S. Application of digital imaging in periodontics: A review. Int J Scientific Research. 2019;3:56–57.
29. Patramurti C, Fenty. Association between smoking behaviour and glycohemoglobine levels among adult javanese indonesian smokers. J Pharm Sci community. 2020;17:76–85.
30. Battancs E, Gheorghita D, Nyiraty S, Lengyel C, Eördegh G, Baráth Z, *et al*. Periodontal disease in diabetes mellitus: A case–control study in smokers and non-smokers. Diabetes Ther. 2020 Nov;11(11):2715–28.

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