The Saudi Dental Journal (2023) **35**, 525–533

King Saud University

The Saudi Dental Journal

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ORIGINAL ARTICLE

The effect of different cigarette smoking levels on gingival crevicular fluid volume and periodontal clinical parameters in Saudi Arabia

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Received 23 March 2023; revised 1 May 2023; accepted 2 May 2023
Available online 8 May 2023

KEYWORDS

Tobacco;
Cigarette;
Smoking;
Gingivitis;
Periodontitis;
Periodontal disease;
Gingival crevicular fluid;
GCF;
Saudi Arabia

Abstract *Introduction:* Periodontal disease is a chronic inflammatory condition of the periodontium. It is the main cause of tooth loss and is considered one of the biggest threats to the oral cavity. Tobacco smoking has long been associated with increased risk for periodontal, *peri*-implant, and other medical diseases.

Objective: To evaluate the effect of smoking and its level on periodontal clinical parameters (probing depth (PD), plaque index (PI), gingival index (GI), clinical attachment level (CAL), bleeding on probing (BOP), and the volume of gingival crevicular fluid (GCF)) in healthy and chronic periodontitis individuals.

Material and Method: A total of 160 participants were recruited in the present study, who were equally divided into the following five groups: healthy controls (C), healthy smokers (HS), non-smokers with periodontitis (PNS), light smokers with periodontitis (PLS), and heavy smokers with periodontitis (PHS). GCF volume and periodontal clinical parameters (PD, PI, GI, CAL, and BOP) were assessed for each participant and compared between the study groups.

Result: There was a statistically significant difference in PD, PI, GI, CAL, and BOP between healthy and periodontitis patients ($p < 0.001$). The mean PI, PD, and CAL were considerably higher in heavy smokers than light smokers and non-smokers ($P < 0.001$). In contrast, the mean GI and BOP were significantly lower in heavy smokers than in light smokers and non-smokers. There was a statistically significant difference in GCF between healthy and periodontitis patients ($p < 0.001$). The mean GCF readings were higher in heavy smokers than light smokers or non-smokers ($P < 0.001$).

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Peer review under responsibility of King Saud University. Production and hosting by Elsevier.

Production and hosting by Elsevier

https://doi.org/10.1016/j.sdentj.2023.05.001

Conclusion: The present study confirms the influence of smoking on periodontal clinical parameters. Smoking was associated with increased PD, PI, CAL, and GCF readings; however, GI and BOP were decreased in smokers. The number of cigarettes played a key role in the volume of GCF and periodontal clinical parameters.

1. Introduction

Periodontitis is a chronic, destructive, inflammatory condition affecting the supporting structures of the teeth. It has been listed in the global burden of chronic diseases (Caton et al., 2018). There are approximately 800 species of bacteria in the oral cavity (Deo and Deshmukh, 2019). The etiology of periodontitis involves a complex interaction between bacterial infection and host response, modified by behavioral factors such as smoking, resulting in periodontal tissue breakdown (Holtfreter et al., 2015). Advanced stages of periodontitis lead to tooth loss, which negatively affects mastication, speech, quality of life, and self-confidence. It has been reported that the prevalence of periodontitis shows that severe periodontitis affects approximately 700 million people worldwide (Dye, 2012). The Prevalence of lack of periodontal health was estimated to be 90% in Saudi Arabian residents aged 25 and over, and more recent estimates reported rates around 50% (Guile, 1992). The prevalence of periodontitis was higher in Saudi Arabia compared to western countries (Mokeem et al., 2004). Many researchers have reported the adverse effects of smoking on periodontal tissues (Anand et al., 2012, Razali et al., 2005, Ustun and Alptekin, 2007, Goultschin et al., 1990). Previous studies (Anand et al., 2012, Razali et al., 2005) have revealed that smokers are at an increased risk of periodontitis development and tend to have greater probing depth when compared to non-smokers. A few studies also indicate the harmful effects of smoking on surgical and non-surgical treatment outcomes (Ustun and Alptekin, 2007). The habit of smoking and the number of cigarettes smoked per day both have detrimental effects on the periodontal status of the individual (Goultschin et al., 1990). Cigarette smoking is associated with higher plaque and calculus deposits (Pankaj et al., 2007). On the other hand, smoking tends to mask gingival inflammation due to gingival vasoconstriction caused by nicotine (Bergstrom and Floderus-Myrhed, 1983, Bergstrom, 1990). Thus, clinically, smokers usually present with decreased signs of inflammation compared to non-smokers (Bergstrom et al., 1988, Baharuddin and AI-Bayaty, 2008).

Gingival crevicular fluid (GCF) is a transudate and inflammatory exudate present in the gingival sulcus or periodontal pocket between the gingival epithelium and the adjacent tooth surface (Muller et al., 2000). During normal physiological activities like mastication and brushing, GCF volume increases, while pathologically, it grows in the presence of inflammation (Preber and Bergstrom, 1985). The volume of GCF is a recognized marker of gingival health status (Goultschin et al., 1990). The role of GCF and its importance in periodontitis have been studied by various authors (Muller et al., 2000, Preber and Bergstrom, 1985, Ojima and Hanioka, 2010). Periodontitis was previously classified into two types: aggressive and chronic. Although they are both basically the same disease process, they advance at varying rates and with varying degrees of severity (Papapanou et al., 2018). The severity and complexity of management determined the four stages of periodontitis, I, II, III, and IV. The grades A, B, or C, indicate the rate of advancement as slow, moderate, or rapid (Berglundh et al., 2018).

Traditional clinical periodontal parameters, such as probing pocket depth (PD), bleeding on probing (BOP), and clinical attachment level (CAL), commonly used for periodontal diagnosis, are often of limited usefulness because they are indicators of previous periodontal disease rather than current disease activity. A more accurate assessment of disease activity may assist with early intervention in patients with this disease. The volume of GCF could provide an indication of the status of tissue breakdown. Researchers from different parts of the world reported clinical periodontal parameters, including plaque index (PI), gingival index (GI), PD, CAL, and BOP, in their respective populations (Gupta et al., 2016, Erdemir et al., 2004, Lira-Junior et al., 2017), while others have also assessed GCF readings (BinShabaib et al., 2019, Mokeem et al., 2014). There is limited research on clinical periodontal parameters and GCF volume in smokers with periodontal diseases in Saudi Arabia. No studies have looked into the effect of the number of cigarettes on periodontitis. Our hypotheses consist of two parts: 1) Saudi patients experience the same effects of smoking on GCF volume and periodontal clinical parameters as patients from other papulations. 2) The number of cigarettes will have a greater impact on GCF, PD, PI, GI, CAL, and BOP.

In an attempt to understand the relationship between smoking and periodontitis and the reflection of smoking on GCF and periodontal parameters, the present study aimed to evaluate PI, GI, PD, CAL, BOP, and volume of GCF in the Saudi Arabian population and to establish the influence of smoking and its level on healthy and chronic periodontitis patients.

2. Material and methods

2.1. Ethical considerations

Approval of the Ethical Committee was obtained from the College of Dentistry Research Center (CDRC), approval No. PR0129, and the Institutional Review Board (IRB) at King Saud University, Saudi Arabia, approval No. E-21–6211. All participants were informed of the study protocol and informed that they had the option to withdraw from the study at any time with no penalties or consequences. Information regarding the detrimental effects of tobacco smoking on oral health and hygiene instructions were given to all individuals, regardless of their decision to participate or decline participation in the present study.

2.2. Participants and grouping

The sample size was calculated using a power sample of (0.80) and a significance level of $p \leq 0.05$. The study sample consisted of one hundred and sixty systematically healthy Saudi patients, and 32 subjects were assigned to each of five groups. Only healthy individuals attending the outpatient dental clinic at King Saud University, Saudi Arabia, were included in the study. Participants were recruited between January 1, 2022, and July 31, 2022″. Periodontitis is defined according to the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions as Stages I-IV with any Grade (Papapanou et al., 2018). All the study participants received an explanation of the research and were recruited upon signing an informed consent form. A simple Arabic and English assessment form was used to gather information regarding age, sex, and smoking duration and frequency. Medical and dental histories were also recorded. Based on their periodontal health and smoking status, participants were categorized into the following five groups:

H n = 32 Periodontally healthy control nonsmokers.

HS n = 32 Periodontally healthy who are smokers.

PNS n = 32 Patients with periodontitis who are nonsmokers.

PHS n = 32 Patients with periodontitis who are heavy smokers; >10 cigarettes/ day (Tonetti, 1998).

PLS n = 32 Patients with periodontitis who are light smokers; <10 cigarette/ day (Tonetti, 1998).

2.3. Exclusion criteria

Individuals who smoke other nicotinic products such as water pipes, cigars, pipes, and e-cigarettes, as well as alcohol users; people with systemic diseases such as diabetes, cardiovascular disease, acquired immune deficiency syndrome, hepatic and renal diseases, pregnancy, breastfeeding, osteoarthritis, osteoporosis, and osteopenia; patients who reported using antibiotics, probiotics, steroids, and nonsteroidal anti-inflammatory drugs within the past four months; patients who are using contraceptives and anti-depressant, and those who refused to sign the written informed consent form were also excluded from the present study.

2.4. Clinical parameters

Clinical evaluation of the participants was done before GCF collection, which included full mouth probing depth (PD) (Ainamo and Bay, 1975), plaque index (PI) (Silness and Loe, 1964), gingival index (GI) (Loe, 1967), clinical attachment level (CAL) (van der Velden, 2005), and bleeding on probing (BOP) (Ainamo and Bay, 1975) at six surfaces (mesiobuccal, mid-buccal, distobuccal, distolingual/palatal, mid-lingual/palatal, and mesiolingual/palatal) of all maxillary and mandibular teeth. PD and CAL were measured using the same periodontal probe. (UNC-15, HuFreidy's, USA) CAL measurements were made from the cementoenamel junction to the bottom of the sulcus. One examiner recorded all clinical data (AZ).

2.5. Gingival crevicular fluid sampling

The GCF was collected one day after clinical and radiological evaluations. All GCF samples for the chronic periodontitis groups (PNS, PHS, and PLS) were collected from the two deepest non-adjacent interproximal pockets. For the healthy groups (H, HS), samples were collected from the deepest interproximal periodontal pocket. Before GCF collection, the selected site was isolated with sterile cotton rolls, and supragingival oral biofilm was gently removed. The tooth was then dried with gentle air pressure using a triple syringe. A sterile paper strip (PerioPaper, Oraflow Inc., Hewlett, NY, USA) was inserted into the target pocket until resistance was felt and held in place for 30 s. Samples contaminated with blood were discarded, and another site was used for GCF sample collection. The GCF volume was calculated based upon measurements obtained from a calibrated digital machine (Periotron 8010, Oraflow Inc., Hewlett, NY, USA).

2.6. Data analysis

Descriptive statistics were conducted for the data analysis using IBM SPSS Statistics for Windows (Version 21.0, IBM Corp., Armonk, NY, USA). The data were expressed as means and standard deviations. The statistical significance of differences between groups was tested according to the nonparametric analysis of variance (ANOVA; Friedman) and Mann–Whitney *U* test. Intergroup comparisons were performed using a one-way analysis of variance. The Bonferroni post-hoc correction test was used for multiple comparisons (within the groups and among the groups) with a power of 95%. P-values < 0.05 were considered statistically significant.

3. Results

The distribution of the participants in the study was summarized in Table 1. Overall, 160 participants were involved in the study, of whom 65% (104) were male and 35% (56) were female. The mean age of the participants in this study was 40 ± 12.62 years. The mean age for H, HS, PNS, PHS, and PLS was 33.43 ± 10.3, 32.31 ± 6.3, 50.12 ± 11.84, 48.68 ± 10.47, and 36.25 ± 10.58 years, respectively. The comparison was not statistically significant ($p > 0.05$). In H, HS, PNS, and PLS, males (56.3%) outnumbered females (43.8%). In the PHS group, the entire population was limited to only males, and no females were presented. The gender-based comparison among all the groups was statistically significant ($p = 0.001$).

The mean comparison among the groups was illustrated in Fig. 1. The overall mean PD, CAL, GI were 4.89 ± 2.01 mm, 1.79 ± 1.87 mm, 1.25 ± 1.15 mm respectively. The overall mean PI was 38.43 ± 22.37% and BOP was 30.30 ± 23.85 %. The overall mean GCF reading of the study population was 69.66 ± 41.96. (Table 2).

The mean PD was higher in PHS (6.65 ± 1.4 mm), followed by PLS (6.46 ± 1.5 mm), and lower means were associated with PNS (5.69 ± 0.86 mm). The mean CAL was higher in PHS (3.53 ± 1.67 mm), followed by PLS (3.31 ± 1.35 mm), and lower means were associated with PNS (2.13 ± 1.07 mm).

Table 1 Age and gender distribution of the study population among the groups.

Population	Age (mean ± SD)	Gender				
			Female	Male	Total	P value
H	33.4 ± 10.5	N	14	18	32	0.001
		% within Group	43.8%	56.3%	100.0%	
		% within Gender	25.0%	17.3%	20.0%	
HS	32.3 ± 6.3	N	14	18	32	0.001
		% within Group	43.8%	56.3%	100.0%	
		% within Gender	25.0%	17.3%	20.0%	
PNS	50.1 ± 11.8	N	14	18	32	0.001
		% within group	43.8%	56.3%	100.0%	
		% within Gender	25.0%	17.3%	20.0%	
PHS	48.7 ± 10.5	N	0	32	32	0.001
		% within group	0.0%	100.0%	100.0%	
		% within Gender	0.0%	30.8%	20.0%	
PLS	36.3 ± 10.6	N	14	18	32	0.001
		% within group	43.8%	56.3%	100.0%	
		% within Gender	25.0%	17.3%	20.0%	

H: Healthy (control); HS: Periodontally healthy who are smokers PNS: Patients with Periodontitis who are nonsmokers PHS: patients with Periodontitis who are heavy smokers; PLS: patients with Periodontitis who are light smokers.

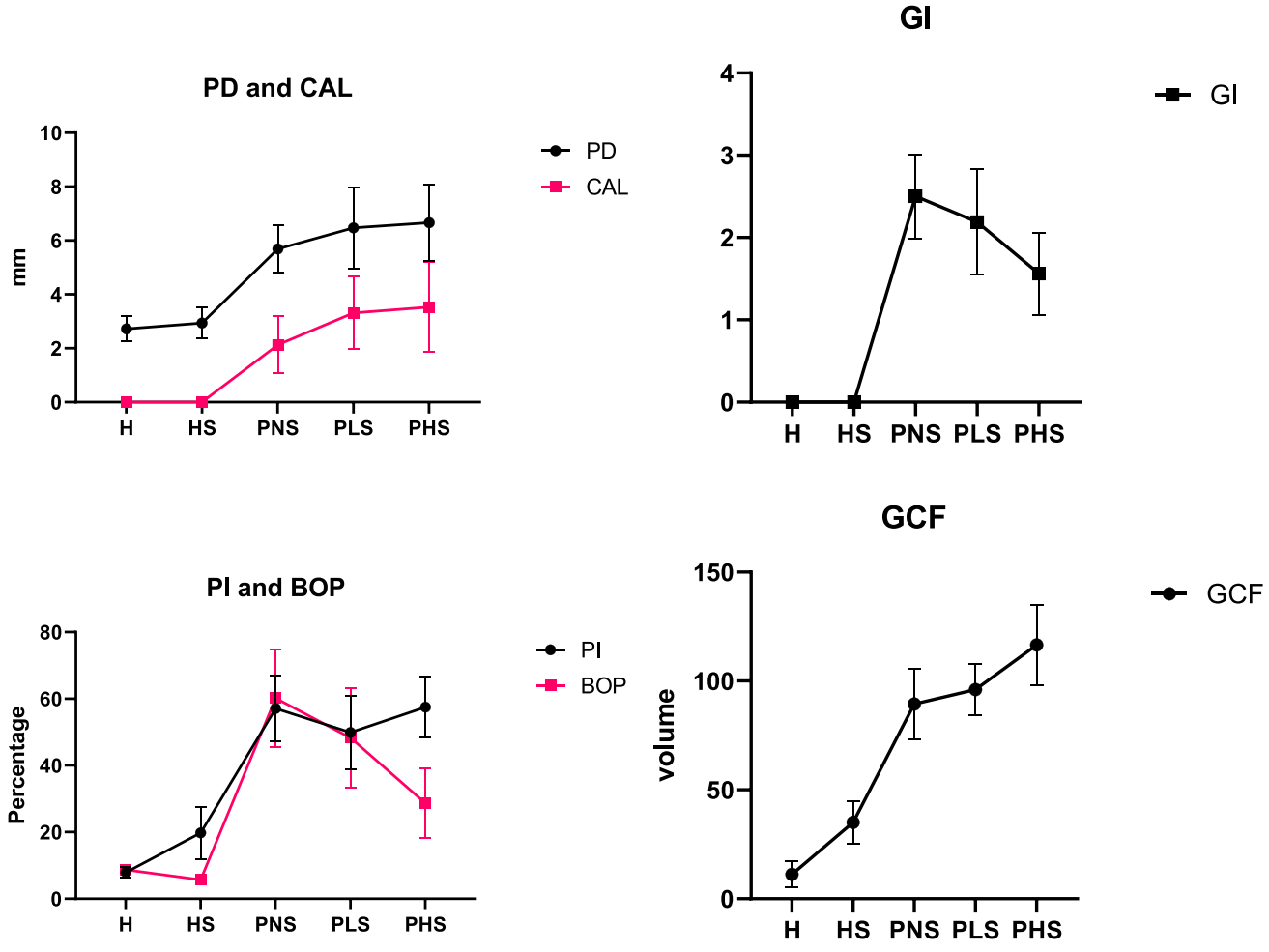

Fig. 1 Comparison of mean probing depth (PD), clinical attachment level (CAL), gingival index (GI), plaque index (PI), bleeding on probing (BOP) and gingival crevicular fluid (GCF) volume between all the five groups. (H: healthy, HS: Periodontally healthy who are smokers, PNS: Patients with Periodontitis who are nonsmokers, PHS: patients with Periodontitis who are heavy smokers, PLS: patients with Periodontitis who are light smokers (< 10 cigarette/ day).

Table 2 Overall values of clinical parameters of study populations.

Parameters	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean	
					Lower Bound	Upper Bound
PD (mm)	160	4.89	2.01	0.16	4.58	5.21
PI (%)	160	38.43	22.37	1.77	34.93	41.92
GI	160	1.25	1.15	0.09	1.07	1.43
CAL (mm)	160	1.79	1.87	0.15	1.50	2.09
BOP (%)	160	30.30	23.85	1.89	26.58	34.02
GCF (μL)	160	69.66	41.94	3.32	63.11	76.20

In periodontitis subjects, both heavy and light smokers were significantly higher in PD and CAL than nonsmokers ($p = 0.035$, 0.003, and 0.000). High BOP scores have been observed in PNS (69.19 ± 14.61%), lower scores in PLS (48. 31 ± 14.96%), least in PHS (28.63 ± 10.46%), and the comparison among the groups showed statistical significance ($P = 0.000$). The volume of collected GCF was significantly higher in PHS compared with PLS and PNS ($p = 0.000$). (Table 3).

Regardless of smoking status, the comparison between periodontitis (60%) non-periodontitis (H and HS) (40%) showed a statistically significant difference in all parameters (Table S1). The mean PD was higher in subjects with periodontists (6.27 ± 1.35 mm) compared to non-periodontitis (2.83 ± 0.52 m m) with a statistically significant difference ($p < 0.001$). No clinical attachment loss was evident in healthy subjects compared to subjects with periodontitis (2.99 ± 1.5 mm) and the difference was statistically significant ($p < 0.001$). PI scores were higher in subjects with periodontitis (54.83 ± 10.61%) than non-periodontitis (13.81 ± 8.23%), and the comparison was statistically significant ($p = 0.008$). GI mean scores were very low (0.0 ± 00) in non-periodontitis subjects compared to those with periodontitis ($p < 0.001$). The mean BOP scores were higher in subjects with periodontitis (45.71 ± 18.7%) compared to non-periodontitis (7.19 ± 1.96 %), a significant difference ($p < 0.001$). The mean volume of GCF was lower

Table 3 The comparison of clinical parameters between study groups.

Characteristics		N	Mean	Std. Deviation	95% Confidence Interval for Mean		P-value
					Lower Bound	Upper Bound	
Probing Depth (mm)	H	32	2.72	0.46	2.55	2.88	0.001
	HS	32	2.94	0.56	2.73	3.14	
	PNS	32	5.69	0.86	5.38	6.00	
	PHS	32	6.66	1.41	6.15	7.16	
	PLS	32	6.47	1.52	5.95	7.02	
Plaque Index (%)	H	32	7.88	1.60	7.30	8.45	0.001
	HS	32	19.75	7.90	16.90	22.60	
	PNS	32	57.13	9.98	53.53	60.72	
	PHS	32	57.50	9.20	54.18	60.82	
	PLS	32	49.88	11.07	45.88	53.87	
Gingival Index	H	32	0.00	0.00	0.00	0.00	0.001
	HS	32	0.00	0.00	0.00	0.00	
	PNS	32	2.50	0.51	2.32	2.68	
	PHS	32	1.56	0.50	1.38	1.74	
	PLS	32	2.19	0.64	1.96	2.42	
Clinical Attachment Level (mm)	H	32	0.00	0.00	0.00	0.00	0.001
	HS	32	0.00	0.00	0.00	0.00	
	PNS	32	2.13	1.07	1.74	2.51	
	PHS	32	3.53	1.67	2.93	4.13	
	PLS	32	3.31	1.35	2.82	3.80	
Bleeding on probing (%)	H	32	8.69	1.12	8.28	9.09	0.001
	HS	32	5.69	1.38	5.19	6.18	
	PNS	32	60.19	14.61	54.92	65.46	
	PHS	32	28.63	10.46	24.85	32.40	
	PLS	32	48.31	14.97	42.92	53.71	
Gingival Crevicular fluid volume (μL)	H	32	11.19	6.06	9.00	13.37	0.001
	HS	32	35.09	9.79	31.56	38.62	
	PNS	32	89.41	16.37	83.50	95.31	
	PHS	32	116.50	18.29	109.91	123.09	
	PLS	32	96.09	11.62	91.90	100.28	

in non-periodontitis subjects (23.14 ± 14.5) than in those with periodontitis (100.67 ± 19.36), and the findings were statistically significant ($p = 0.006$).

Regardless of periodontal status, the comparison between smokers (60%) and non-smokers (40%) showed a statistically significant difference in all parameters (Table S2). The mean PD was higher in smokers (5.35 ± 2.11 mm) compared to non-smokers (4.2 ± 1.64 mm), a statistically significant difference ($p = 0.045$). The mean CAL was 1.06 ± 0.16 mm in non-smokers compared to 2.04 ± 0.16 mm in smokers, with a statistically significant difference ($p < 0.001$). PI scores were higher in smokers (42.38 ± 18.8%) than non-smokers (32.5 ± 25.81%), with the comparison being statistically significant ($p < 0.001$). The mean BOP scores were higher in non-smokers (34.44 ± 27.91%) compared to smokers (27.54 ± 20.40%), with a significant difference ($p < 0.001$). The mean volume of GCF was lower in non-smokers (50.3 ± 41.28) compared to smokers (82.56 ± 37.32), and the findings were statistically significant ($p = 0.008$).

4. Discussion

Smoking is one of the most commonly observed adverse habits among individuals in developing countries. It has a definitive role in periodontal breakdown (Gautam et al., 2011). Nicotine, a cytotoxic substance in tobacco smoke, can adhere to the tooth surface, penetrate the oral mucosa, and enter the bloodstream (Al-Tayeb, 2008). Various cellular and molecular mechanisms implicated in the pathogenesis of smoking-induced periodontal diseases include stromal cell dysfunction in the oral cavity, immunosuppression, and exaggerated inflammatory cell responses (Raulin et al., 1988, Gani et al., 2012).

The present study recruited a total of 160 participants, who were divided into five groups of 32 participants each: healthy (no periodontal disease and never smoked) as a control group; healthy non-periodontitis smokers; periodontitis non-smokers; periodontitis heavy smokers; and periodontitis light smokers. Periodontal clinical parameters (PD, CAL, GI, PI, and BOP) were recorded for each participant. The mean age of the participants in the healthy groups was 32.87 ± 8.6, and in the periodontitis groups, the mean age was 45.02 ± 12.5.

The mean GI in periodontitis groups was significantly higher as compared to healthy groups ($p < 0.05$). However, within the periodontitis groups, the mean GI score was the highest in non-smokers (2.50 ± 0.5) and the lowest in heavy smokers (1.56 ± 0.5), thus suggesting reduced gingival inflammation in smokers. There is one study that did not find any significant difference in the GI between the smoking and non-smoking groups (Ustun and Alptekin, 2007). The contrasting findings could be attributed to the variation in sample size and population. Nevertheless, most similar studies reported a difference in GI between smokers and non-smokers (Baharuddin and AI-Bayaty, 2008, Muller et al., 2000). This could be due to the constriction of gingival blood vessels, which conceal the gingival inflammation (Bergstrom and Floderus-Myrhed, 1983, Bergstrom, 1990, Gani et al., 2012). The mean BOP was 60.19 ± 14.6 in the periodontitis non-smokers group, while the mean BOP in the periodontitis heavy smokers' group was 28.63 ± 10.4 %. These findings suggested that the mean BOP was inversely associated with smoking. A study also found a lower mean BOP score in smokers (Al-Bayaty et al., 2013). The observations made in the present study are in accordance with other studies done (Feldman et al., 1983, Gautam et al., 2011) which found that smokers with periodontal disease had lower BOP and clinical inflammation when compared to non-smokers with periodontal diseases.

It has been shown that smoking causes changes in neutrophil activities, antibody production, expression of adhesion molecules, and production of cytokine inflammatory mediators that ultimately result in alteration of the host response (Ryder et al., 1998) which may explain the reduction in inflammation and reduced GI and BOP in the smokers' group in our study. In smokers, the gingiva appears swollen, but there is reduced vascular density and angiogenesis due to a suppressed inflammatory response, which explains the impaired wound healing in smokers compared to non-smokers (Bergstrom et al., 1988). Gingival inflammation and BOP are well-known clinical markers used by clinicians to monitor the oral health status of individuals, but smokers often present with reduced gingival inflammation and BOP (Baharuddin and AI-Bayaty, 2008). Hence, it becomes of utmost importance for general practitioners to be aware of the effects of smoking on all the clinical parameters of periodontal health.

Cigarette smoking is an important environmental factor that hastens the periodontal tissue destruction process (Linden and Mullally, 1994). Early observations report that smokers showed a higher prevalence of plaque than non-smokers, suggesting more severe periodontal disease in smokers because of the amount of plaque accumulation (Kristoffersen, 1970, Preber et al., 1980). The mean PI in the present study was found to be higher (57.5 ± 9.2) in heavy smokers in the periodontitis group as compared to other groups. Several studies found that smokers had the highest mean PI score when compared to non-smokers (Al-Bayaty et al., 2013, Pankaj et al., 2007, Ustun and Alptekin, 2007, Ibraheem et al., 2020).

The mean PD in periodontitis patients was the highest in heavy smokers (6.46 ± 1.52) and the lowest in non-smokers (5.68 ± 0.85). Also, the mean CAL was highest (3.53 ± 1.66) in the heavy smokers' group and lowest (2.12 ± 1.07) in the nonsmokers' group. Smoking demonstrated a dose-dependent effect on clinical periodontal parameters in the current study. The lowest means of PD, CAL, PI, and GCF volume were associated with non-smokers, followed by light smokers, while heavy smokers demonstrated the highest means. Periodontal parameters were always higher in heavy smokers compared to light smokers, with GCF volume being significantly higher in heavy smokers. This indicates that the number of cigarettes consumed has a positive relationship with clinical periodontal parameters, GCF volume, and the overall severity of periodontal disease. One explanation of this observation could be the increased duration of exposure of periodontal tissue to the heat and harmful chemicals in cigarettes. The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions (Papapanou et al., 2018) recognized the influence of the number of cigarette consumption on periodontal health. It was established in the newer classification that smoking < 10 cigarettes per day indicated a moderate progression of the disease, while smoking > 10 cigarettes per day indicated a rapid progression.

A study (Gomes et al., 2009) evaluated the effects of smoking on GCF volume during the treatment of gingivitis and observed that higher GCF volumes were significantly associated with deeper periodontal pockets. They also suggested that smoking affects the GCF volume independently of the presence of BOP and PD. Indeed, we had a similar observation in the current study. The volume of GCF was significantly higher in healthy individuals who are smokers compared to healthy individuals who are non-smokers, despite both groups lacking the signs and symptoms of a periodontal disease. This could indicate that GCF's volume, as a marker, has lower specificity to periodontal inflammation as it was increased due to smoking in the absence of periodontal inflammation. From another perspective, it can be presumed that healthy individuals who are smokers are at greater risk to develop periodontitis owing to the higher levels of GCF compare to none smokers.

Smoking has detrimental effect on clinical parameters and GFC in Saudi population. Complete smoking cessation should an ultimate goal and part of periodontal treatment. However, since this may not be easily achievable, reducing the number of cigarettes gradually may be a more practical approach given the negative effect associated with heavier smoking levels.

Based on power analysis, a sufficient sample was used. The sample size was comparatively large relative to published studies. Erdemir et al. investigated 41 Turkish subjects including 22 volunteer smokers (Erdemir et al., 2004). Üstüna and Alptekinb utilized only 13 male Turkish patients (Ustun and Alptekin, 2007); Mokeem et al. performed their study with 60 Saudi subjects 30 smokers and 30 non-smokers (Mokeem et al., 2014). Gupta et al. included 60 Indian subjects (Gupta et al., 2016); BinShabaib et al. included 134 Saudi individuals 45 e-cigarette smokers, 44 smokers, and 45 non-smokers (BinShabaib et al., 2019). This study is one of the very few studies evaluate the effect of smoking on clinical parameters in a Saudi population. It is also the first to consider different levels of smoking (light /heavy). It is also unique in the large sample that was utilized in this study compared to existing studies. One of the limitations of this study is the absence of female subjects in the PHS group. It is indeed still difficult to encounter a female individual with such characteristics in the Saudi population, despite the general increase in smoking prevalence in recent years (Algabbani et al., 2018). Also, this study was limited only to cigarette smokers. Other forms of smoking, like waterpipes and vape devices, could theoretically have a variable impact on the clinical parameters of periodontitis. Nevertheless, the present study involved both males and females in the evaluation of clinical parameters, and this is considered one of the few studies that studied both genders in the Middle East. The present study is the first of its kind to evaluate clinical parameters and GCF readings among the various patient groups and healthy subjects. This study also compared the clinical parameters among healthy subjects, subjects with periodontists, and smokers and non-smokers.

5. Conclusions

The present study establishes the influence of smoking on clinical periodontal parameters. Smoking was associated with increased PD, PI, CAL, and GCF readings; however, GI and BOP were decreased in smokers. The mean GCF readings were higher in periodontitis patients than healthy individuals, and in smokers than non-smokers. The number of cigarettes consumed also has a considerable influence on GCF, PD, PI, GI, CAL, and BOP.

6. Institutional review board statement

The study protocol was approved by the institutional review boards (IRBs) of King Saud University Medical City (approval No. E-21–6211) and College of Dentistry Research Centre (CDRC) (approval No. PR 0129).

7. Informed consent statement

Informed consent was obtained from all subjects involved in the study.

CRediT authorship contribution statement

Abdullah F. AlZamil: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data curation, Writing – original draft, Writing – review & editing, Visualization, Supervision, Project administration, Funding acquisition. **Montaser N. AlQutub:** Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data curation, Writing – original draft, Writing – review & editing, Visualization, Supervision, Project administration, Funding acquisition.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

The authors would like to thank the Deanship of Scientific Research at King Saud University for funding and supporting this research through the initiative of DSR Graduate Students Research Support (GSR).

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sdentj.2023.05.001.

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