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The impact of electronic cigarette smoking on periodontal status and proinflammatory cytokine levels: a cross-sectional study

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Abstract

Background E-cigs(E-cigs) use is increasing worldwide. Recent studies suggest that E-cigs contain harmful elements that could lead to adverse oral health outcomes. Therefore, the aim of this study was to evaluate the impact of E-cigs smoking on periodontal health among current male smokers in Al-Kharj city in Saudi Arabia by assessing periodontal parameters and proinflammatory cytokine levels.

Methods Fifty-three male individuals (25 E-smokers and 28 non-smokers) participated in the study. This study compared periodontal parameters, including the plaque index (PI), bleeding on probing (BOP), probing pocket depth (PPD), clinical attachment loss (CAL), and marginal bone loss (MBL), as well as levels of unstimulated whole saliva (UWS), interleukin IL-1B, and IL-6, between E-smokers and non-smokers.

Results The E-cigs users consumed approximately 432.6 ± 425.22 puffs on average daily, with a nicotine content of approximately 45.2 ± 11.23 mg on average. There was no statistically significant difference between the groups in terms of BOP and PI. A statistically significant difference was detected in PPD and CAL ($p < 0.05$) between the two groups, in which the PPD (4.10 ± 1.87) and CAL (2.72 ± 0.89) were greater in E-smokers. The mean MBL was also higher among E-smokers, which was statistically significant ($p < 0.05$). The mean cytokine IL-1B level was found to be (640.75 ± 138.78) among non-smokers and (889.05 ± 540.56) among E-smokers, and this difference was statistically significant ($p < 0.05$). However, while IL-1B had shown a significant difference between groups in the bivariate analysis (t-test), its association with E-cigs use became non-significant in the multivariate model (OR = 1.01, 95% CI: 1.00–1.02, $p = 0.194$). The mean IL-6 level among non-smokers was (19.49 ± 11.90) and among E-smokers, it was (17.07 ± 8.21). And, this difference was not statistically significant ($p > 0.05$).

Conclusions This study revealed that E-cigs smoking had a negative effect on periodontal status (especially PPD, CAL and MBL). These results may contribute to the pathogenesis of periodontal diseases and tissue destruction. Efforts must be made to educate and create awareness among patients and the general community regarding the risks associated with E-cigs usage especially in young populations.

Keywords Electronic cigarettes, Cytokines, Periodontitis, Clinical attachment loss, Interleukin

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Background

Cigarette smoking is associated with significant health complications [1, 2]. Long-term nicotine use has the potential to induce various adverse health effects, such as respiratory problems, attention deficits, mood disorders, and periodontal diseases [3–5]. Moreover, one study reported that nicotine exposure in any age group leads to reduced insulin sensitivity and can contribute to the development of diabetes mellitus [6]. Other studies have shown a direct link between cardiovascular diseases and continued consumption of nicotine, which can also affect the immune system [7]. In females, nicotine exposure can impact the brain development of fetus during pregnancy and can induce preterm delivery with low birth weight [8]. Electronic cigarettes, also known as "E-cigs," represent a contemporary form of nicotine consumption that is increasingly prevalent among young adults and carries a notable health hazard [9]. E-cigs are typically battery-operated and function by heating a solution comprising nicotine, various carrier substances, and fruity flavouring agents. Upon heating, a vapour is produced and is inhaled and exhaled by users, as with traditional tobacco smoking [2, 10]. Ever since E-cigs were introduced worldwide and in Saudi Arabia, they have become increasingly popular. Many young individuals who were previously non-smokers adopted E-cigs as a novel social habit [9]. A study performed among health science students in Saudi Arabia (KSA) reported that the percentage of E-cigs users was 27.7% [9]. Another cross-sectional study performed in KSA reported that 26.3% of participants used E-cigs at least once in their lifetime [11]. On the other hand, although some studies have shown that smoking E-cigs is somewhat less harmful than traditional smoking is, recent evidence indicates that vaping emits a number of potentially toxic substances that could lead to adverse health outcomes [12]. A study from the University of North Carolina revealed that the two main components found in E-cigs—vegetable glycerin and propylene glycol—are toxic to cells and that the greater the number of elements in an e-liquid is, the greater the degree of toxicity [13]. Moreover, flavourings in E-cigs contain lung toxins such as menthol, diketones and diacetyl and contribute more to cell mortality than regular tobacco flavouring does [14, 15]. Periodontitis is influenced by systematic factors such as diabetes, genetics, and smoking, which are linked to increased risk, prevalence, and severity [16]. Studies have shown that individuals who smoke cigarettes tend to have higher levels of periodontal inflammation than non-smokers do, as indicated by clinical parameters such as the plaque index (PI), bleeding on probing (BOP), probing pocket depth (PPD), clinical attachment loss (CAL), and significant marginal bone loss (MBL) [10, 17].

Interleukin-1B (IL-1B) and interleukin-6 (IL-6) are widely studied in oral health research because of their crucial roles in the inflammation and pathogenesis of periodontal diseases [18]. IL-1B is a key cytokine that promotes tissue destruction and bone resorption in response to bacterial infection. It stimulates the production of matrix metalloproteinases (MMPs), enzymes that degrade collagen and other components of the extracellular matrix, leading to the destruction of periodontal ligaments and alveolar bone. Additionally, IL-1B promotes osteoclast formation and activity, which accelerates bone resorption, a hallmark of advanced periodontitis [19]. On the other hand, IL-6 is a multifunctional cytokine with both proinflammatory and anti-inflammatory properties. In the context of periodontal disease, IL-6 is involved primarily in the recruitment of immune cells to the site of infection and the stimulation of acute-phase proteins, which are part of the body's early defence mechanism. However, chronically elevated IL-6 contributes to sustained inflammation by promoting the survival of inflammatory cells and inhibiting their apoptosis. This prolonged inflammatory state leads to further tissue destruction and bone loss. IL-6 also plays a role in bone metabolism by stimulating osteoclast activity, which contributes to the resorption of alveolar bone, a critical feature of periodontitis [20, 21]. Elevated levels of these cytokines are often observed in periodontal pockets and correlate with disease severity, making them important biomarkers for diagnosing and monitoring periodontal conditions. Furthermore, their involvement in the breakdown of periodontal tissues highlights their potential as therapeutic targets to control inflammation and slow disease progression in periodontal tissues [18, 21]. According to many studies, the levels of these cytokines are significantly greater in cigarette smokers than in E-smokers and non-smokers, which contributes to the degradation of periodontal tissues [22, 23]. On the other hand, a cross-sectional study revealed no significant difference in periodontal health status between E-cigs users and those who had never smoked [24]. A study by Wadia et al. revealed significantly greater gingival inflammation in patients vaping E-cigs; however, all individuals in this study were former cigarette smokers [25]. For that reason, the exclusive role of E-cigs in oral health, particularly with respect to periodontal parameters, is still limited or unclear. Additionally, to the best of our knowledge, no studies have investigated the correlations of clinical or radiographic markers of periodontal inflammation and proinflammatory cytokine levels with E-cig usage characteristics, such as the duration and frequency of smoking. Moreover, since vaping is associated with an increased risk of inflammatory conditions such as bronchiolitis, awareness of the impact of this type of smoking is important for removing the delusion that vaping is safer than

traditional smoking is, especially for oral health, in the Saudi community. Therefore, the aim of this study was to address the existing discrepancies and limited data regarding clinical parameters and laboratory findings necessary to fully understand the impact of e-cigarettes on periodontal health. The study is based on two research hypotheses: (1) the clinical parameters are significantly worse in E-cigs smokers than in non-smokers, and (2) the levels of the proinflammatory markers IL-1B and IL-6 are greater in E-cigs smokers than in non-smokers.

Materials and methods

Ethical approval

This study was carried out at the College of Dentistry, Prince Sattam Bin Abdulaziz University. Approval was obtained from the standing committee of bioethics research (SCBR) (No. 153/2023). The study followed the guidelines of the Declaration of Helsinki and was conducted between September and August 2023/2024. Patients aged 18 years or older were recruited for the study. The participants were informed about the study's objectives and provided written informed consent before the study began.

Study groups

The study consisted of 2 groups as follows: In Group 1, the E-smokers group consisted of all patients who identified themselves as E-cigs users through self-reports and were using E-cigs regularly every day for at least one year (Fig. 1). In addition, patients using only two electronic cigarette devices (pod systems and vape pens) were included. With respect to Group 2, we included patients who confirmed that they had never smoked. For sample size calculation, post hoc calculation of power using G*Power software (version 3.1) was performed. At an alpha of 0.05 (with two tailed p values) and an effect size (Coden's d) of 0.8, to achieve a power of 0.81 (which is greater than the traditional acceptable level of 0.80), each group needed at least 25 participants. The exclusion criteria were as follows: (a) refusal to provide consent by signing the consent form; (b) individuals who self-identified as cigarette smokers; (c) individuals who both smoked with vaping (referred to as dual smokers); (d) individuals who self-reported the use of smokeless nicotine products; (e) individuals who self-identified as former cigarette smokers; (f) individuals who self-reported systemic diseases, including acquired immune deficiency syndrome, cardiovascular disorders, diabetes mellitus, and renal disorders; and (g) individuals who had used antibiotics, nonsteroidal anti-inflammatory drugs, or steroids within 90 days prior to the study. Additionally, those who underwent periodontal therapy or surgery within the previous six months were excluded from the study.

Questionnaire

A questionnaire was adapted from a previous similar study on the use of E-cigs [26]. The data consisted of two parts: part one included the demographic data of the participants, and the second part included data concerning the duration of E-cigs smoking, daily consumption of cigarettes, duration of vaping, daily vaping frequency, number of puffs taken during each electronic nicotine delivery system (ENDS) session, and nicotine concentration in electronic cigarettes (e-liquid). The questionnaires were administered by a trained interviewer, and it was attached as Additional file.

Clinical status assessment

The assessment of periodontal status was conducted in outpatient clinics at the College of Dentistry, PSAU. Measurements of the O'Leary plaque index (PI) [27], bleeding on probing (BOP) [28], probing pocket depth (PPD) (≥4 millimetres [mm]) and clinical attachment level (CAL) in mm were recorded for each patient using a Williams periodontal probe (Hu-Friedy). Full-mouth clinical measurements were obtained from six sites around each tooth (mesiolingual /palatal, distolingual/palatal, mid-lingual/palatal, mesiobuccal, mid-buccal and distobuccal), with the exception of the third molars [29]. Patients were diagnosed with periodontitis in line with the 2017 World Workshop according to these criteria: their interdental CAL was evident at ≥2 nonadjacent teeth; their buccal or oral CAL was ≥3 mm with pocketing>3 mm detectable at ≥2 teeth; and the observed CAL could not be attributed to non-periodontitis causes [30]. Radiographic assessments were completed by one calibrated and trained examiner (overall kappa was 0.9) who was blinded to the project groups. Bitewing radiographs were obtained using a dental radiograph machine (CareStream Dental LLC, Atlanta) and viewed on a calibrated computer screen (XL2270 SyncMaster digital TV monitor, Korea). Marginal bone loss (MBL) measurements were conducted for the right and left molars and premolars, excluding the wisdom tooth, and then the average was calculated in mm. MBL was defined as the vertical distance from 2 mm below the cementoenamel junction (CEJ) to the most coronal part of the marginal alveolar bone [17]. Teeth surfaces on which the CEJ or the bone crest was not clearly detectable for technical reasons (such as restoration, dental caries, overlapping of teeth and poor radiographic quality) were excluded. The number of remaining teeth was also determined and documented. However, broken teeth with embedded root remnants were counted as missing [31]. Clinical examinations and sample collection were performed by an investigator who was not involved in patient enrolment and was blinded to the group assignments of the study. Intra-examiner variability was assessed in 12 regular

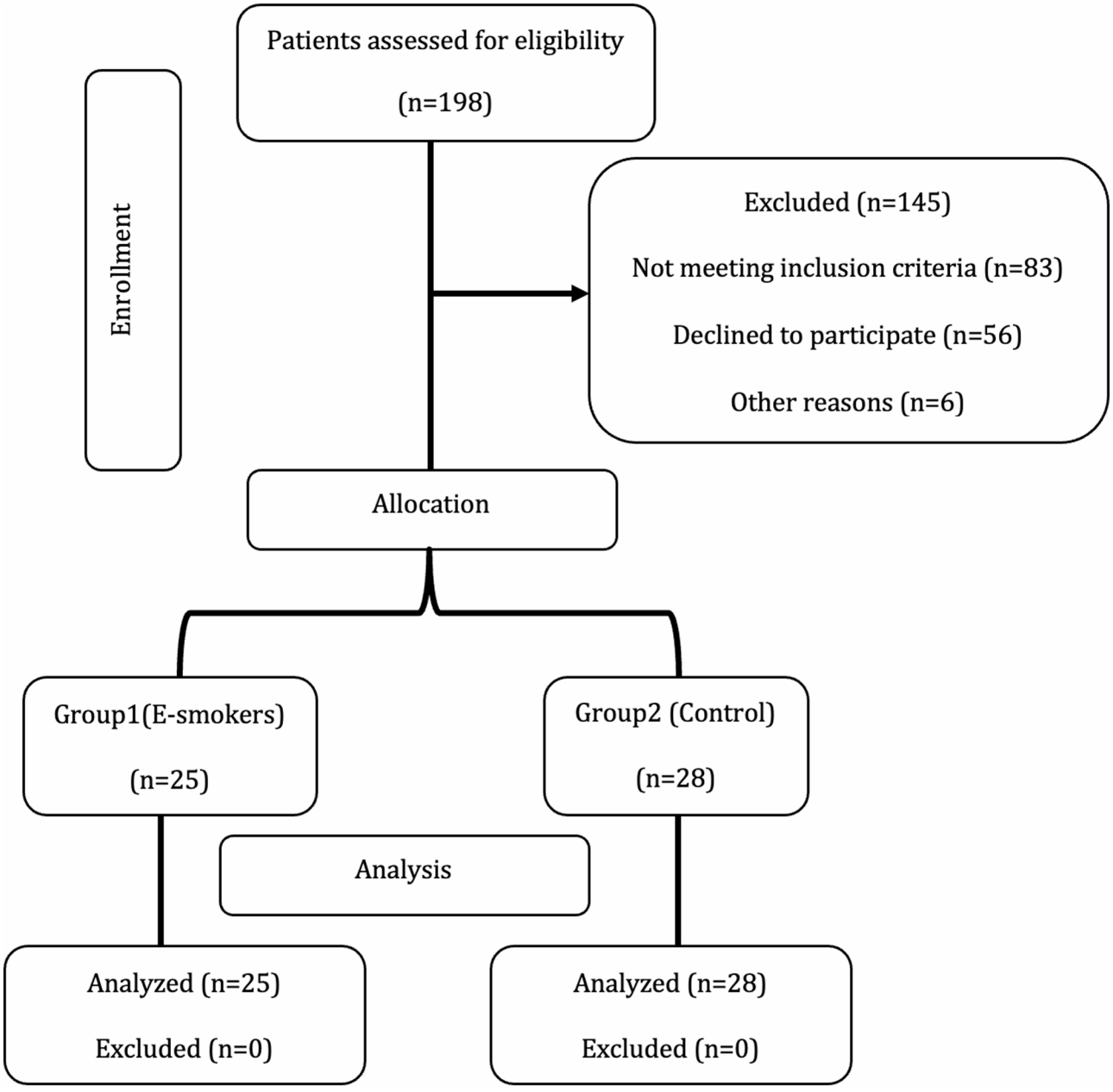

Fig. 1 The flow chart of patient's enrolment

patients with periodontitis (not from the study) for CAL and PPD. The kappa coefficients were 0.75 and 0.83, respectively.

Collection of unstimulated whole saliva samples and assessment of salivary IL-1B and IL-6 protein concentrations

Unstimulated whole saliva (UWS) samples were obtained via a non-invasive method, as detailed in a previous study [32]. To summarize, UWS collection took place in the early morning hours, with participants in a fasting state. The participants were seated comfortably and instructed to allow saliva to accumulate in their mouths for a continuous period of 5 min. Then, they were advised not to swallow or make any jaw movements during this time. After the 5-minute period, the participants discharged the UWS into a disposable plastic container, and the collected supernatant was preserved at -80 °C. The concentrations of IL-1B and IL-6 in the saliva samples were measured via enzyme-linked immunosorbent assay (ELISA) via a commercially available human IL-1B ELISA kit (Elabscience®, E-EL-H0149, Houston, Texas, USA) and a human IL-6 ELISA kit (Elabscience®, E-EL-H6156) following the manufacturer's instructions. The

Table 1 Demographic characteristics of the study groups

Parameters	Non-smokers	E-smokers
Number of participants (n)	28	25
Gender (male)	28	25
Age in years (mean ± SD)	37 ± 1.4	24.52 ± 2.29
consumption	NA	432.6 ± 425.22
Nicotine concentration (mean ± SD)	NA	45.2 ± 11.23

NA: not applicable

*There is a significant difference in age between study groups ($P < 0.001$)

absorbance at 450 nm was detected with a microplate reader (BioTek Synergy HT plate reader, Winooski, VT, USA). All assays were performed in duplicate from two independent experiments. The mean concentrations of the replicates were used to indicate the respective IL-1B and IL-6 protein concentrations in each saliva sample. All saliva samples were collected by one calibrated investigator (Kappa 0.88).

Statistical analysis

Statistical analysis was performed using a computer software (IBM SPSS Statistics, Version 27, Chicago, IL, USA). Clinical (PI, BOP, PPD and CAL) radiographic (mesial and distal MBL) parameters and cytokine levels were compared between the two groups using independent t tests at a 0.05 level of significance. Descriptive statistics, including means, standard deviations (SDs), and frequencies, were calculated for demographic and clinical variables. Independent-samples t tests were used to compare means between study groups (clinical parameters: plaque index [PI], bleeding on probing [BOP], probing pocket depth [PPD], and clinical attachment loss [CAL]; radiographic parameters: marginal bone loss [MBL]; and cytokine levels: IL-1B, IL-6). Scatter plots with linear regression lines are presented for the associations between the consumption of E-smoking (duration and consumption) and each of the periodontal parameters. For multivariate analyses, binary logistic regression models were constructed to assess associations between E-cigsuse and periodontal or inflammatory markers, adjusting for age as a potential confounder. A backwards step-wise elimination approach was applied to refine the periodontal indices model, retaining significant or near-significant variables. Odds ratios (ORs) with 95% confidence intervals (CIs) were reported for logistic regression outcomes.

Results

The demographic characteristics of the study participants are shown in Table 1. Overall, 53 male individuals (25 E-smokers and 28 non-smokers) participated in the current study. There was no statistically significant difference in the mean ages of E-smokers (24.52 ± 2.29 years) or non-smokers (37.14 ± 1.4 years). The E-cigs users consumed approximately 432.6 ± 425.22 puffs on average daily. Among E-cig users, the average nicotine content was 45.2 ± 11.23 mg. Table 2 shows a comparison of periodontal parameters between non-smokers and E-smokers. The mean BOP among non-smokers was 34.0% ± 24.29, and among E-smokers, it was 28.60 ± 13.74. The difference in the mean BOP between the groups was not statistically significant ($p > 0.05$). The mean PI among Non-smokers was 41.0% ± 19.35, and among E-smokers, it was 48.16 ± 20.70, which was not statistically significant ($p > 0.05$). The mean CAL among non-smokers was 1.64 ± 2.04, and among E-smokers, it was 2.72 ± 0.89; the difference was statistically significant ($p < 0.05$). The mean PPD was 2.72 ± 0.67 among non-smokers and 4.10 ± 1.87 among E-smokers, and the statistical analysis revealed that this difference was significant ($p < 0.05$). The mean number of teeth present among non-smokers was 30.40 ± 1.63, whereas among E-smokers, the mean number of teeth present was 29.42 ± 2.91; this difference was not statistically significant ($p > 0.05$) (Table 2). The mean

Table 2 Comparison of different periodontal parameters between Non-Smokers and E-smokers

Periodontal parameters	Group	Mean	Std. Deviation	Mean difference	CI (95%) Lower limit	CI (95%) Upper limit	*p* value
BOP (%)	Non-smokers	34.03	24.29	0.05	-0.06	0.17	0.315
	E -Smokers	28.60	13.74				
PI (%)	Non-Smokers	41.00	19.35	-0.07	-0.18	0.04	0.199
	E -Smokers	48.16	20.70				
CAL (in mm)	Non-Smokers	1.64	2.04	-0.61	-1.44	0.21	0.040*
	E -Smokers	2.72	0.89				
PPD (in mm)	Non-Smokers	2.72	0.67	-1.08	-1.80	-0.36	0.004*
	E -Smokers	4.10	1.87				
No. of remaining Teeth (n)	Non-Smokers	30.40	1.63	1.03	-0.36	2.41	0.140
	E -Smokers	29.42	2.91				
Marginal bone loss (MBL) (in mm)	Non-Smokers	1.59	0.22	-1.39	-2.22	-0.56	0.002*
	E -Smokers	2.81	0.53				

Note: * = significant difference between groups

Table 3 Comparison of cytokine levels between Non-Smokers and E-smokers

Cytokine levels	Group	Mean	Std. Deviation	Mean difference	CI (95%) Lower limit	CI (95%) Upper limit	t value	*p* value
IL-1B	Non-Smokers	640.75	138.78	-248.30	-476.63	-19.96	2.348	0.034*
	E-Smokers	889.05	540.56					
IL-6	Non-Smokers	19.49	11.90	2.41	-3.19	8.02	0.849	0.400
	E-Smokers	17.07	8.21					

Note: * = significant difference between groups

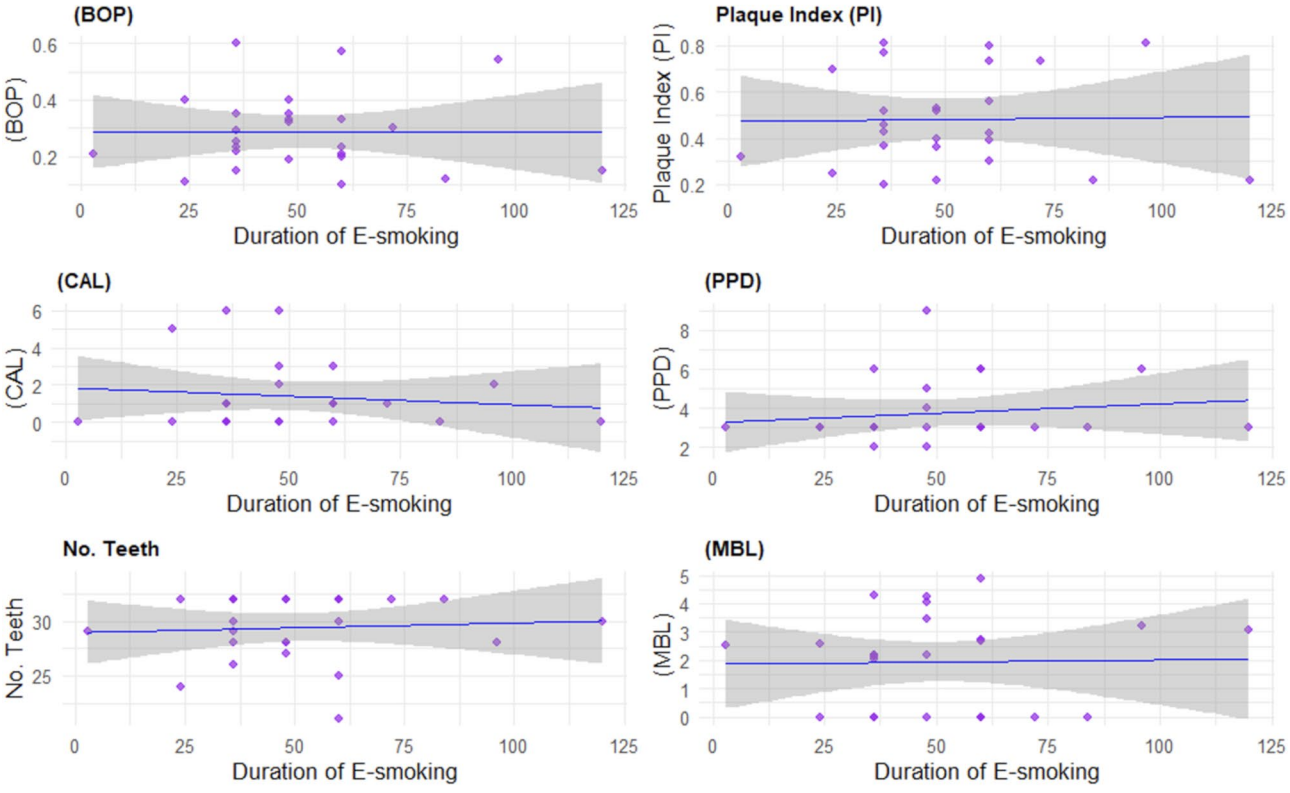

Fig. 2 Correlations between the duration of E-smoking and the periodontal parameters of smokers

MBL was 1.59 ± 0.22 among non-smokers and 2.81 ± 0.53 among E-smokers, which was a statistically significant difference ($p < 0.05$) between the two groups.

Table 3 shows a comparison of cytokine levels between non-smokers and E-smokers. The mean IL-1B level was found to be 640.75 ± 138.78 among non-smokers and 889.05 ± 540.56 among E-smokers, which was statistically significant ($p < 0.05$). The mean IL-6 level among non-smokers was 19.49 ± 11.90, and among E-smokers, it was 17.07 ± 8.21. However, this difference was not statistically significant ($p > 0.05$). With respect to the associations between E-smoking characteristics and periodontal parameters, no significant associations were found, and the scatter plots in (Figs. 2 and 3) show no correlation patterns. (The assessment was in the E-smokers group, with only 25 participants; the sample size could be a limitation for the identification of these consumption–response relationships.

The results of the multivariate analysis using backwards step-wise regression, adjusted for age, revealed significant associations between specific periodontal indices and E-cigs use (Table 4). Marginal bone loss (MBL) (OR = 2.15, 95% CI: 1.124.11, $p = 0.021$) was identified as a significant predictor of E-cigs use. An increase in MBL was associated with 2.2 times greater odds of E-smoking. Probing pocket depth (PPD) tended to increase with E-smoking (OR = 2.07, 95% CI: 0.76 to 3.06, $p = 0.002$), and this increase was statistically significant. The odds of E-smoking significantly decreased with increasing age, which means that younger participants are more likely to engage in E-smoking than older participants are. These findings suggest that structural periodontal changes,

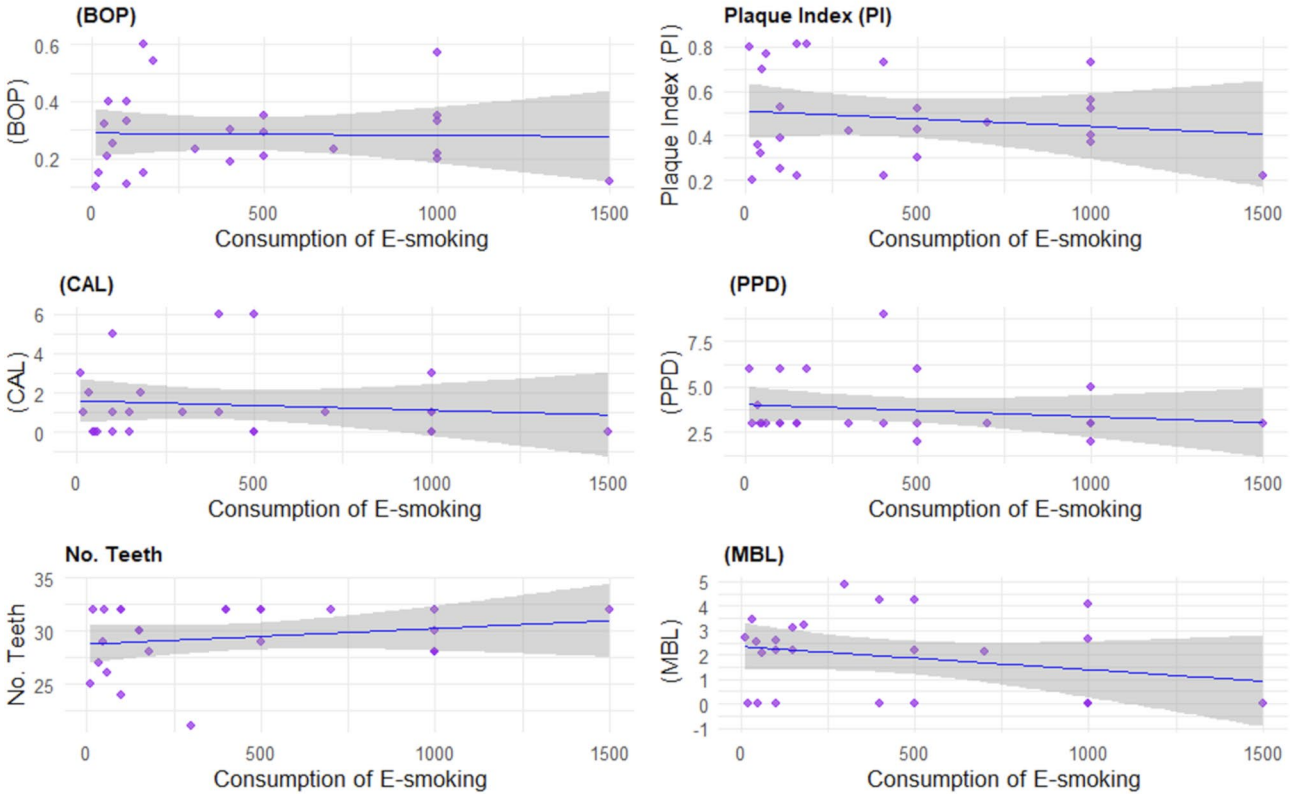

Fig. 3 Correlations between the E-smoking consumption and periodontal parameters of smokers

Table 4 Findings of multivariate analysis with step-back regression for the differences between study groups regarding periodontal indices adjusted for age

Predictors	*P* value	Odds Ratio	Lower limit (95% C.I)	Upper limit (95% C.I)
Age	0.008*	0.78	0.65	0.94
PPD	0.002*	2.07	0.76	3.06
MBL	0.021*	2.15	1.12	4.11

Note: * = significant difference between groups

Table 5 Findings of multivariate regression analysis for the differences between study groups regarding levels of cytokines adjusted for age

Predictors	*P* value	Odds Ratio	Lower limit (95% C.I)	Upper limit (95% C.I)
Age	0.008*	0.85	0.75	0.96
IL-1B	0.194	1.01	1.00	1.02
IL-6	0.435	0.97	0.90	1.04

Note: * = significant difference between groups

particularly MBL and PPD, are critical factors distinguishing E-smokers from non-smokers, when adjusted for age.

The multivariate regression analysis, adjusted for age, revealed that IL-1B and IL-6 levels were no longer significantly associated with E-cig use (Table 5). While IL-1B was significantly different between E-smokers and non-smokers in the bivariate analysis (t test), its association with E-cigs use became nonsignificant in the multivariate model (OR = 1.01, 95% CI: 1.00–1.02, $p = 0.194$). Similarly, IL-6 was not significantly associated with E-cig use (OR = 0.97, 95% CI: 0.90–1.04, $p = 0.435$). In contrast, age was a significant predictor (OR = 0.85, 95% CI: 0.75–0.96, $p = 0.008$), indicating that younger individuals were more likely to be E-smokers.

Discussion

In the present study, the participants' ages were 24.52 ± 2.29 years for E- smokers and 37.14 ± 1.4 years for non-smokers. These results confirm the traditional hypothesis that the mean age of E-cigs users is 19 years, which is younger than that of cigarette smokers, especially among males in (KSA) [11]. Our findings indicate that E-cig users exhibit significantly greater CAL and PPD values than non-smokers do, suggesting more severe periodontal conditions among E-cig users. These results agreed with those of previous studies, which reported that vaping might not be a safer alternative to smoking in terms of tissue health, as both practices appear to promote inflammatory responses in the oral cavity [1, 17, 24, 26, 33, 34]. In addition, e-liquid nicotine acts as a symbiotic factor for periodontal destruction by

affecting the degree of fibroblast collagen and integrin production as well as stimulating cytokine production in periodontal tissues [35–38]. However, similar findings were revealed by other American studies, which revealed a link between the toxicants discharged from E-cigs and poor periodontal conditions [35, 39, 40]. Previous studies have shown that there is a cytotoxic potential of e-liquid additives as flavouring agents for improving gingival oral health. For example, propylene glycol, a common carrier, induces oxidative stress in gingival fibroblasts [35], whereas menthol disrupts epithelial barrier integrity, exacerbating bacterial infiltration [24]. In vitro studies by Sundar et al. (2016) demonstrated that cinnamon and fruit-flavoured aerosols significantly upregulate IL-1B in oral epithelial cells [10]. These agents may synergize with nicotine to amplify periodontal inflammation, underscoring the need for mechanistic research on flavour-specific toxicity.

In this study, we examined two prevalent E-cigs devices: pod systems and vape pens. Pod systems are compact and use prefilled or refillable pods, whereas vape pens are slightly smaller, featuring refillable tanks that allow for more customization in e-liquid. Despite the differences in device design, both systems use e-liquids containing the same nicotine concentration [40]. With respect to BOP, although there was no statistically significant difference between the groups, the mean score of the E-cigs group was lower than that of the control group. This can be explained by the fact that nicotine products and vaping E-cigs can lead to gingival vasoconstriction and a reduction in the tissue blood supply. Similarly, a previous study revealed that BOP was significantly greater in non-smoking patients than in cigarette, waterpipe and E-cig smokers [17]. Therefore, nicotine smokers and ENDS users may not be aware of the ongoing periodontal inflammatory process since gingival bleeding, which is a critical sign of periodontal disease, is reduced [26]. For PI, there was a negative correlation between E-cig use and plaque accumulation, suggesting that E-cig users experience periodontal deterioration independent of plaque accumulation. Research has shown that cigarette smokers are more prone to biofilm formation than E-cigs users are. This may be due to the chemical-thermal degradation of nitric oxide, which regulates salivary secretion, as well as a reduction in saliva production caused by nicotine-induced vasoconstriction. Additionally, inadequate oral hygiene, including shorter brushing durations, is another factor that may contribute to increased plaque accumulation [32, 41, 42]. Our findings indicate that, compared with non-smokers, E-cig smokers exhibit significantly greater MBL. This aligns with the literature suggesting that vaping, similar to traditional smoking, may contribute to destruction of periodontal tissues, including bone loss around teeth [1, 16, 26, 43]. These findings are in accordance with those of a previous study in which the authors reported increased scores of mesial and distal MBL and missing teeth in a group of E-smokers compared with never-smokers with/without periodontitis [44]. A recent meta-analysis assessed the effects of E-cigs on periodontal tissue health in contrast with traditional smoking and non-smoking populations. Their assessment revealed that cigarette smokers had significantly greater PIs, PPDs, CAL and MBL than E-cigs users and non-smokers did. However, they did not provide adequate analysis for E-cigs versus non-smokers or never-smokers [45].

Studies have shown that nicotine smoking causes a proinflammatory reaction by stimulating the release of certain cytokines and reactive oxygen species (ROS). These factors contribute to the degradation of periodontal tissues [23]. However, our investigation revealed greater concentrations of IL-1B in E-cig smokers than in non-smokers via bivariate analysis (t-test). However, the multivariate regression analysis, adjusted for age, revealed that IL-1B levels were no longer significantly associated with E-cig use. These findings suggest that the initial significant difference in IL-1B levels between E-smokers and non-smokers may have been confounded by age, and after adjusting for this variable, the association was no longer significant. This finding varies from that in the literature, highlighting the role of IL-1B in the inflammatory processes associated with nicotine smoking and its impact on periodontal health [32, 41, 42]. The levels of IL-6, which is another proinflammatory cytokine, did not significantly differ between E-cig smokers and non-smokers in our study. This finding contrasts with previous research indicating the upregulation of IL-6 in response to E-cig vapour exposure [2, 10]. This discrepancy could stem from various factors, including the nature of the regulation of IL-1B and IL-6, which are influenced by various systemic and local stimuli, including infections, stress, and environmental factors. The variability in their levels among individuals could be attributed to differences in immune responses or external factors unrelated to smoking habits. Additionally, the duration and intensity of E-cig use may not have been sufficient to induce measurable changes in IL-1B and IL-6 levels, highlighting the need for longitudinal studies to assess the chronic impact of vaping on inflammatory markers [10, 17].

To our knowledge, this study is the first to explore correlations between periodontal parameters and E-cig usage characteristics, such as the duration and frequency of smoking. While no strong correlations were found in this analysis, it was difficult to assess E-cig intensity and standardization thresholds for nicotine frequency due to the product device and liquid heterogeneity [46]. Additionally, the weak, nonsignificant correlations between bleeding on probing (BOP) and the plaque index (PI)

with E-cigs duration and consumption align with recent studies suggesting that the vasoconstrictive effects of nicotine may reduce gingival bleeding, masking periodontal inflammation despite underlying tissue damage [17, 26]. In addition, our findings underscore the complexity of factors influencing periodontal health in E-cigs users, which may extend beyond direct smoking habits to include genetic predispositions and other lifestyle factors, such as stress [3, 47]. Moreover, differences in oral hygiene practices, such as brushing or flossing frequency, could independently affect periodontal health. Dietary habits, particularly sugar and acidic intake, might also contribute to variations in inflammation and plaque levels. Additionally, socioeconomic status (SES) could influence access to dental care and overall oral health, potentially skewing results, and this was considered a limitation. Future studies should control for these factors to better isolate the effects of E-cigs smoking on periodontal health. One of the major limitations of this study is its relatively small sample size, comprising only 53 participants. While this study provides valuable insights into the effects of E-cigs use on periodontal health, the limited number of subjects decreases the generalizability of the findings to the broader population. In addition, an age-matched control group allows for more accurate assessment and minimizes the effect of age as a confounding factor. Additionally, the cross-sectional nature of many studies, including our study, limits the ability to establish causality between E-cigs use and periodontal outcomes. Another limitation of the present study is that the e-cigs status was documented by the participants via self-reports, which may have introduced bias into the findings. Self-reported data can be prone to inaccuracies caused by recall errors, social desirability bias, or deliberate misreporting in which participants might underreport or overreport their vaping habits. Moreover, the absence of standardization of E-cig exposure systems makes comparisons unreliable. In addition, the diversity of E-cigs products and the complexity of their ingredients can lead to variability in studies. Longitudinal studies with larger sample sizes and diverse populations could provide deeper insights into the causal mechanisms and long-term consequences of E-cigs use on oral health outcomes. Furthermore, future studies should consider the use of biochemical markers such as nicotine levels in blood or saliva as objective measures to increase the reliability of smoking data with actual investigations of oral hygiene practices. However, public health initiatives should incorporate evidence-based recommendations to raise awareness about the potential risks associated with E-cigs, especially concerning periodontal health, and promote strategies for smoking cessation and oral hygiene maintenance.

Conclusion

In conclusion, the present study revealed that E-cig smoking had a negative effect on periodontal status. These results may contribute to the pathogenesis of periodontal diseases through the inhalation of nicotine along with different flavouring agents. Efforts must be made to educate and create awareness among patients and the general community regarding the risks associated with e-cig usage, like those associated with nicotine smoking, especially in young populations.

Abbreviations

E-cigs	E-cigarette
E-Smokers	E-cigs smokers
KSA	Saudi Arabia
ENDS	Electronic Nicotine Delivery System
PI	Plaque index
BOP	Bleeding on probing
PPD	Probing pocket depth
CAL	Clinical attachment loss
MBL	Marginal bone loss
ROS	Reactive oxygen species
CEJ	Cemento-enamel junction
SES	Socio-economic status

Supplementary Information

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Supplementary Material 1

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. The standing committee of bioethics research (SCBR) of Prince Sattam Bin Abdulaziz University approved this study protocol (SCBR-153/2023). signed informed consent was obtained from all individuals for participating and agreement for data publication.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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