Research Article

Impact of oral hygiene instructions on cytokines in smokers and vapers with gingivitis

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Abstract: Background: Cigarette smoking (CS) is a periodontal disease risk factor, affecting clinical parameters such as bleeding on probing (BOP), plaque index (PI), gingival index (GI) and proinflammatory cytokines level. This study examines the impact electronic cigarette use on proinflammatory cytokines and periodontal parameters. Methods: In this non-randomized study, ninety participants diagnosed with gingivitis were assigned into three groups. examined the effect of oral hygiene instructions on periodontal parameters and inflammatory biomarkers. Thirty CS (n=30) vaping electronic-cigarettes (e-cig) (n=29), and non-smoker (NS) (n=31) was included. Clinical parameters including PI, BOP, and GI were recorded at baseline and after 3 weeks. Enzyme-linked immunosorbent assay was used to assess levels of matrix metalloproteinases (MMP)-8 and matrix metalloproteinases (MMP)-9 in saliva at baseline and after 3 weeks. Results: CS and vapers exhibited significantly higher PI, GI, MMP-8 and MMP-9 scores compared to NS before and after the oral hygiene instructions, indicating greater periodontal inflammation. CS as well as individuals who use electronic cigarettes exhibited reduced BOP, likely because nicotine-induced vasoconstriction conceals the actual severity of inflammatory periodontal disease. Conclusion: CS and electronic cigarette use adversely affect periodontal health, increasing PI, GI, MMP-8 and MMP-9 levels while altering BOP. These findings stress the importance of tailored maintenance of periodontium smokers and vapers through integrating vaping cessation into treatment protocols to improve oral health outcomes.

Keywords: gingivitis, vaping, smoking, *MMP8*, *MMP9*.

Introduction

Smoking cigarette has been related to peri-implant and periodontal disease [1]. An abundance of research examining the adverse effects of smoking cigarettes on periodontal and peri-implant tissue showed that there is a negative impact of smoking on periodontal and peri-implant tissue [2-4]. A popular emerging form of smoking is electronic cigarette use (commonly known as e-cigarettes), which is especially widespread among young adults aged 18-25 years. Various companies manufacture these devices, which pose significant health risks. E-cigarettes available in pen-like and other shapes, featuring a battery and a metal heating component within a stainless-steel casing, connected to a cartridge and an atomizer. The cartridge contains a solution of nicotine mixed with ingredients like propylene glycol, glycerin, and other chemicals that create fruity or other flavored tastes [5].A common misunderstanding that vaping e-cigarettes exhibits lesser effect on health as opposed to cigarette smoking; meanwhile, ceasing tobacco usage is the most prevalent reported causes for vaping e- cigarettes [6].

The definition for periodontal diseases arises from inflammatory process between bacterial actions and body response [7]. The reversible form of periodontal disease called gingivitis in which its essential sign is gingival irritation due to plaque accumulation and bacterial settlement [7]. One of the most critical factors affecting periodontal and peri-implant tissue is cigarette smoking [7]. A previous study demonstrated the significant positive association between smoking and the counts of *Streptococcus mutans* and *S. sobrinus* in

the cigarette smoker group compared to waterpipe users [8]. Smoking frequency has direct impact on the disease severity and progression in which heavier smokers tend to have more severe form of disease as compared to lighter smokers [7]. The frequency of smoking have been differently categorized in many researches, one specific study categorized smokers, in which light smoker consume less than 9 cigarette per day, and heavy smokers consume more than 31 cigarette per day [9]. A study found that smoking subjects exhibited elevated salivary IL-8 levels and poorer periodontal conditions compared to non-smokers. These findings have indicated that smoking may disrupt immune responses, potentially increasing smokers' susceptibility to periodontal disease [10]. Smoking frequency has direct impact on the disease severity and progression in which heavier smoker tend to have more severe form of disease as compared to lighter smoker. Additionally, response to periodontal treatment is affected by smoking; in contrast to nonsmoker, smokers showed only 50%–75% of recovery has been achieved in their clinical parameters following scaling and root planning [7]. It is well established fact that proinflammatory effect induced by tobacco smoking through exacerbation of some reactive oxygen species(ROS) and cytokines which has a major contribution on periodontium breakdown [11].

Portable devices powered by batteries, known as electronic cigarettes or e-cigs, a heating component can be used and release aerosol full of chemicals generated by the presence of an e-liquid (which commonly contains nicotine and a variety of flavors such as butter, coffee, candy, and menthol and fruits) and this aerosol can be ultimately inhaled [12]. A study showed that RANKL, probing pocket depth (PPD), and clinical attachment level (CAL) showed statistically significant differences between waterpipe smokers and nonsmokers [13]. Due to the presence of aluminum, copper and lead that are emitted by the device heating process, nicotine free electronic cigarette still poses hazard [14]. Traumatic severe injuries can also be inflicted by electronic cigarette. Further concern that is Blast-related injuries induced by battery explosions, this happened in nation where there is no safety regulation and manufacturing monitoring [15]. A previous report showed the presence of smoking had no significant impact on IL-17 levels, however, suppressive action of smoking against IL-10 was encountered [16]. Another study analyzed the impact of vaping on periodontal parameters and found increased levels of plaque index (PI), PPD, CAL, and marginal bone loss in vaping groups compared to non-smokers [17]. The impact of vaping remains less well established, despite substantial evidence on the harmful effects of smoking on the periodontium. This study aimed to determine whether vaping influences proinflammatory cytokine levels and clinical periodontal parameters in patients diagnosed with gingivitis, both at baseline and following oral hygiene instruction.

Materials and Methods

The present study was non randomized parallel group clinical trial and was conducted from December 2023 to June 2024. The Participants were recruited from the patients' pool in the clinics of Department of Periodontics, College of Dentistry University of Baghdad. The study protocol was reviewed and approved from the ethical committee of College of Dentistry, University of Baghdad (reference no. 856623; 3rd of December 2023). The clinical trial was registered at clinicaltrial.gov (NCT06177158). The present study was performed following guidelines recognized by the Declaration of Helsinki as revised in 2013 for experimentation involving human patients. After a full description of the aim and flow of the study, all participants voluntarily agreed to participate in the study and signed consent forms, which the examiner collected. Ninety-six males, 28±5.2 years, out of 220 participants were diagnosed with gingivitis and were selected to participate in this study. The participants were assigned into three groups: 33 participants in smoker, 31 vaper group and 32 in non-smoker group. Few participants were lost to follow up at the end of the study (figure 1).

Eligibility criteria

The included participants should be gingivitis patients smoking cigarettes for at least 12 months, Patients who use electronic disposable vapes in categories of low to medium dependence according to the Penn

State Electronic Cigarette Dependence Index (one-time consisting of around 15 puffs or last around ten minutes). All participants had generalized gingivitis with an intact periodontium, presenting more than 10% bleeding sites, no probing pocket depth greater than 3 mm, and no clinical attachment loss, based on the criteria proposed by Chapple et al. They also had a minimum of 20 teeth and had never consumed tobacco in any form during their lifetime.. [18]. The study excluded dual smokers, smokeless tobacco users, heavy smokers, patients using high-nicotine formulas (more than 25 mg/ml), individuals with periodontitis or systemic diseases, females, those who had taken antibiotics, steroidal, or non-steroidal anti-inflammatory drugs in the past six months, patients who had undergone periodontal therapy within the last six months, and anyone unwilling to participate..

Interventions

At the baseline visit, before receiving oral hygiene instructions and motivation, salivary samples were collected and clinical periodontal parameters were measured. After a period of three weeks, saliva was collected again, and identical clinical measurements were performed. To ensure patients comprehended the instruction sessions, each participant practiced clinically. Toothbrushing guidance, including standardized motivation and oral hygiene instructions, was demonstrated to all three groups by means of the modified Bass technique and a standardized medium-bristled toothbrush with toothpaste. Participants were instructed to brush twice daily for two minutes using a pea-sized amount of dentifrice..

Study outcomes:

Primary outcome

The primary outcomes were the reduction in periodontal parameter BOP and changes between the baseline and 3 weeks for salivary matrix metalloproteinase (MMP)-8 and -9.

Secondary outcomes:

The secondary outcomes were reduction in periodontal parameters (PI and GI) between the baseline and the endpoints of the study.

Saliva collection

At baseline and end point of the study, saliva was collected as previously described [19]. First, the Participants were instructed to avoid eating, drinking, or carrying out any oral hygiene practices for a minimum of two hours before saliva collection. Subsequently, they were requested to rinse their mouths with tap water for about 30 seconds, approximately 10 minutes before the collection. Afterward, they were instructed to expectorate into sterile tubes while seated in an upright position until a total of 3 ml of unstimulated saliva was obtained. The collected saliva was centrifuged for 20 min at 3000 rpm. A A micropipette was employed to transfer the clear salivary supernatants into plastic Eppendorf tubes containing a premeasured 300 μL solution of protease inhibitor, intended to neutralize the activity of protease enzymes present in the saliva. All collected samples were stored at −20 °C until analysis.

Periodontal parameters

After saliva has been collected, a comprehensive periodontal examination was done using a periodontal probe (UNC-15) probe. The clinical parameters were measured for all the existing dentition included full mouth PI [20] GI[21] and full mouth BOP [22]. All teeth were considered in the measurements except wisdom teeth. These clinical parameters were recorded at baseline as well as at the end point of the study i.e., after three weeks of oral hygiene practices including brushing and flossing.Subsequent to saliva sampling, clinical periodontal recordings, PI was measured by using Modified Quigley-Hein Index (WM-mQHI) on six surfaces (mesiobuccal, buccal, distobuccal, mesiolingual, lingual, and distolingual). With mQHI, each

surface is normally given a score of 0 to 5 as follows: 0 = no plaque, 1 = separate flecks of plaque at the cervical margin; 2 = thin continuous band of plaque (up to 1 mm) at the cervical margin; 3 = band of plaque wider than 1 mm but covering less than one third of the crown; 4 = plaque covering at least one third but less than two thirds of the crown; 5 = plaque covering two thirds or more of the crown [20]. BOP [22] was measured by gently inserting the periodontal probe to the depth of the gingival sulcus \ periodontal pocket then removed coronally and waited for 30 seconds to observe the presence of bleeding (0 = no bleeding, 1 = presence of bleeding). GI was measured by using Löe and Silness index each of the four gingival areas of the tooth is given a score from 0 to 3 as follows: 0 = Normal gingiva, 1 = Mild inflammation slight change in color, slight oedema. No bleeding on probing, 2 = Moderate inflammation—redness, oedema and glazing. Bleeding on probing, 3 = Severe inflammation marked redness and oedema/ Ulceration / Tendency to spontaneous bleeding [22].

Determination of salivary human MMP8 and MMP9

Levels of biomarkers in salivary samples were determined using ELISA which was based on the Biotin double antibody sandwich technology as manufactural instructions. In brief, 40 µl of sample was added to the wells of Coated ELISA Plate followed by 10 µl antibodies and 50 µl streptavidin-HRP. Then after covering the plate with seal membrane and gentle shaking, the plate was incubated at 37°C for 60 min. After incubation, the wells were washed five times. For color development, 50 µl chromogen solution A was added to each well followed by 50 µl chromogen solution B and the plate was incubated for 10 min at 37°C away from light after gentle shaking. An amount of 50 µl of stop solution was added to each well to stop the reaction (blue color changed into yellow immediately). Then, the optical density values of the samples in wells were measured using microplate ELISA reader device (Humareader HS, Germany) under 450 nm wavelength, which should be carried out within the 10 minutes after having added the stop solution. The concentrations of the samples were calculated by using the equation obtained from the standard curve specific for each biomarker.

Statistical analysis

All statistical analyses were done using a statistical software program SPSS (version 27.0 for Windows, IBM, Chicago, IL, USA). Non parametric statistical tests were used for non-normally distribution data revealed by Shapiro-Wilk Normality test. For intragroup differences (between baseline and end point therapy) Wilcoxon signed rank test was used for non- parametric data. To detect any differences among interventions Kruskal Wallis test followed by the Dunn-Bonferroni post hoc test were used. Significant difference was set at p value <5%.

Results

Demographic variables

The demographic characteristics of the participants are illustrated in (table 1). The highest mean age was observed in the CS group (33.3±4.6 years), while the youngest participants were in vapers (23.4 ± 2.3). The mean duration of habit in minutes was higher in vapers (16.7±2.6) than CS (8.5±1.5).

Table 1: Demographic characteristics of the study sample (n=90 male).

Groups	Cig smoker (N=30)	Vapers (N=29)	Non-smokers (N=31)
Age mean ±sd	33.2±4.6	23.4±2.3	27.4±3.0
Mean duration of Habit (in years)	5.8±1.7	2.2±0.7	
Mean duration of session (in minutes)	8.5±1.5	16.7±2.6	

Periodontal parameters

All clinical periodontal parameters including PI, BOP, and GI reduced at baseline and after three weeks of oral hygiene measures. In all groups, there was a significant reduction in GI scores after oral hygiene instructions. For the intergroup differences there was significant differences between the three groups after oral hygiene instructions as shown in table 2. Similarly, mean percentage of BOP was significantly improved in association with all groups with intergroup differences that were observed between the three groups following oral hygiene instructions (Table 2)

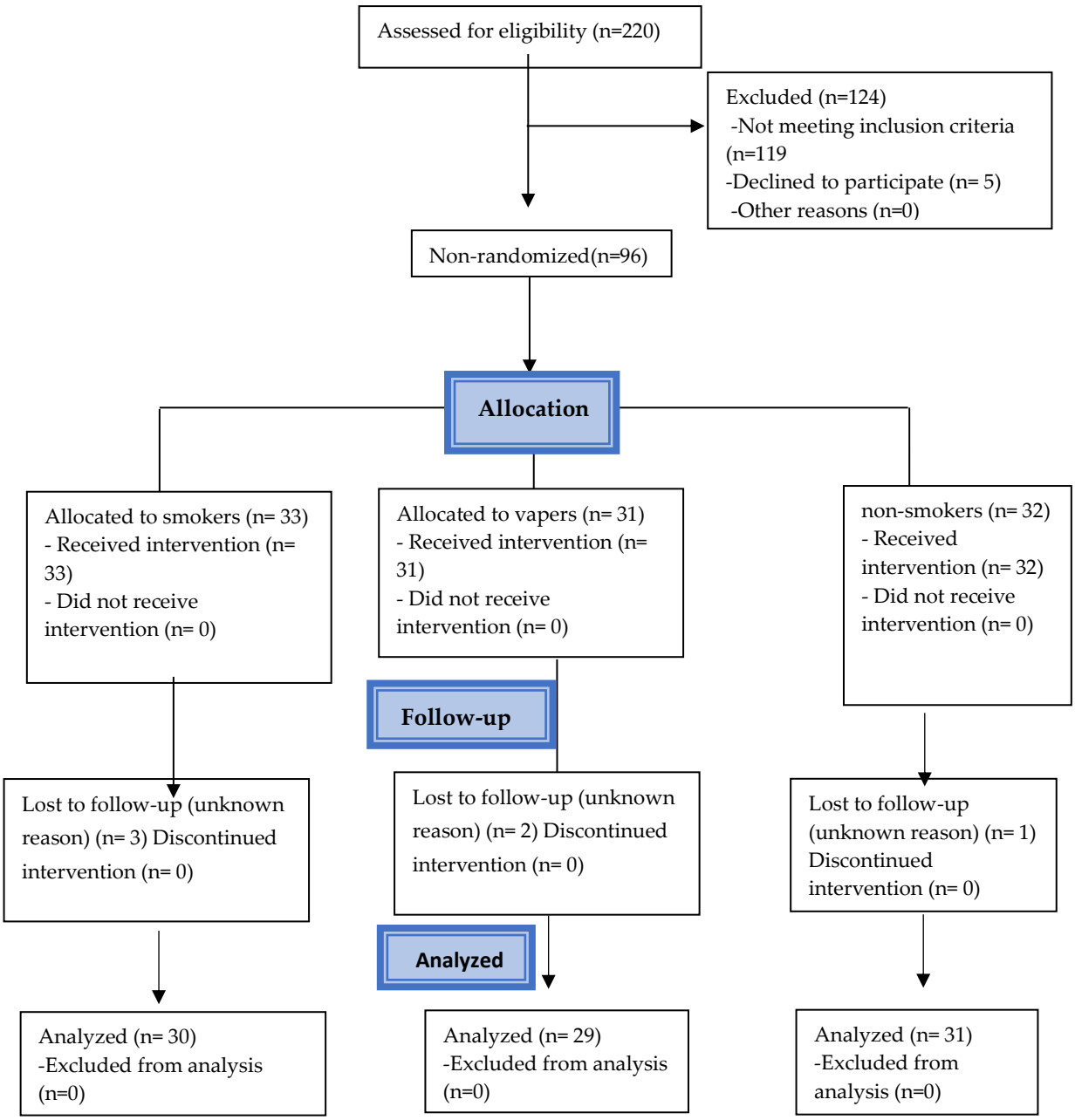

Figure 1 Flow chart of study sample

Table 2 Mean +SDclinical periodontal parameters and mean biomarkers between groups before and after three weeks, in which group 1 is the cigarette smokers, group 2 is E-cigarette smokers, and group 3 is non-smokers.

Periodontal parameters And biomarkers	Cigarette smokers (Group 1) n=30	P Value	E-Cigarette smokers (Group 2) n=29	P Value	Non-smokers (Group 3) n=31	P Value	Post hoc P value (After 3 Weeks)
PI before	4.090±0.29	P >0.001	3.05±0.33	P >0.001	2.04±0.34	P>0.00	<0.001[ac]
PI after	3.203±0.37[ab]		2.15±0.37[c]		0.726±0.27		<0.001[b]
BOP before	37.7%±8.37%	P >0.001	56.4%±8.8%	P >0.001	66.1%±9%	P>0.001	0.681[a]
BOP after	13.7%±2.75%[ab]		16.5%±7.8% [c]		5.9%±1.7%		<0.001[bc]
GI before	0.773±0.09	P >0.001	1.67±0.22	P >0.001	1.97±0.239	P>0.001	1.22[a]
GI after	0.323±0.06[ab]		0.41±0.16[c]		0.21±0.065		<0.001[bc]
MMP8 before	5.6±0.54	P <0.001	4.25±0.95	P >0.001	3.1±0.368	P>0.001	<0.001[ab]
MMP8 after	0.877±0.19[ab]		0.752±0.17[c]		0.452±0.09		<0.001[c]
MMP9 before	4088±332.64	P <0.001	3146±494.5	P >0.001	1624.8±445.7	P>0.001	<0.001[ac]
MMP9 after	2329.73±274.2[ac]		1069±188.6[b]		409.06±53.5		0.0001[b]

group 2 compared to a
group 3 compared to b
group 3 compared to c

Analysis of salivary biomarkers

For MMP8 in smokers there was a significant difference before and after oral hygiene instruction as shown in Figure 2. The P value was <0.001, for E-cigarette smokers there was also significant difference before and after oral hygiene instruction ($p < .001$) and for non-smokers there was a significant difference before and after oral hygiene instruction ($p < .001$).

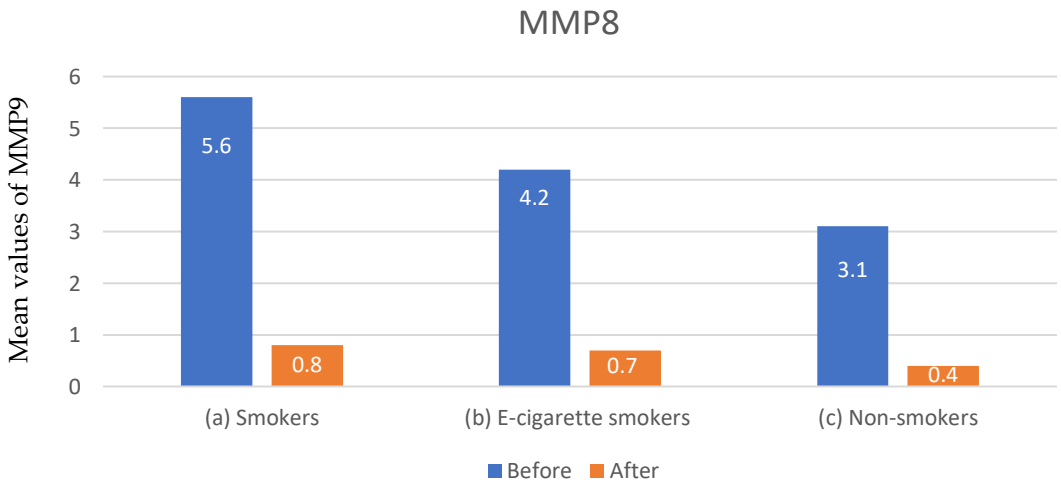

Figure 2: Bar chart representing mean values of MMP8 concentration in y axis before and after oral hygiene instructions (pg/mL) among groups and intervals.

For MMP-9 In all groups, there was a significant reduction in MMP9 values after oral hygiene instructions with P value was < .001.

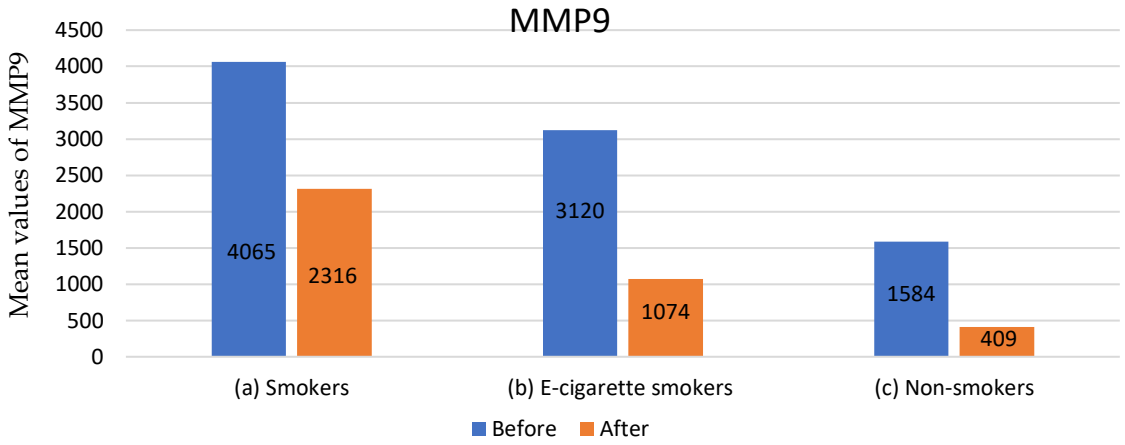

Figure 3: Bar chart representing mean values of MMP9 concentration in y axis before and after oral hygiene instructions (pg/mL) among groups and intervals.

Discussion

Latest research on vaping electronic cigarette and its impact on the clinical, radiographical and periodontal health status showed that individuals who use electronic cigarette exhibit markedly deeper probing depths, more attachment loss, and greater marginal bone loss as opposed to nonsmokers [12]. Nevertheless, to the best of authors' knowledge, this study was the first clinical trial related to the impact of vaping on periodontal health and its effect on proinflammatory cytokines level as compared to cigarette smokers and non-smokers. This study provides the evidence that periodontal parameters and proinflammatory cytokines level are equally worsened in cigarette smokers and individuals using electronic cigarette devices. According to our preliminary results, this study can be reinforced by recent research that show the harmful effect of vaping on oral epithelium and periodontal fibroblast [12]. E-cigarette vapors or aerosol especially that contain flavoring agents, has been related to increase DNA damage and elevate the proinflammatory cytokines level such as cyclooxygenase (COX) 2 and prostaglandin E2 (PGE2) within the cellular context [23]. Additionally it has been hypothesized that both cigarette smoking and vaping can trigger oxidative stress and enhance the levels of advanced glycation end products (AGEs) and their receptors within the cell particularly in gingival and periodontal tissue [12]. it is well established that nicotine in tobacco can diminish the cellular healing response and decrease the BOP tendency [24]. Another study showed that non-smokers experienced significantly higher reduction in BOP than smokers following non-surgical periodontal therapy at sites with moderate and deep bassline PPD [25].

It is remarkable that the percentage of BOP before oral hygiene instruction is significantly lower in smoker and electronic cigarette smoking as compared to non-smokers. Nevertheless, it was significantly higher in smokers and electronic cigarette smokers as compared to non-smoker after oral hygiene instructions. It is recognized that BOP is a traditional parameter that indicates periodontal inflammation [26]. Nicotine is known to cause vasoconstriction leading to decrease in BOP of smokers relative to those who do not smoke [27]. The findings of this study demonstrated that smokers and those who vape show lower bleeding tendency before oral hygiene instructions and higher bleeding scores after oral hygiene instructions as compared to NS suggesting more inflammation occurring in the periodontal tissue in those individuals. Importantly, mean PI was higher in smokers and E-cigarette smokers as compared to non-smokers before oral hygiene instructions. And even after oral hygiene instruction, smokers showed the highest mean in comparison to E-cigarette smokers and non-smokers. Earlier research[28] also proved that cigarette smokers

showed higher plaque accumulation as opposed to non-smokers. Another study found that percentage of PI in smokers were most elevated (~56%) as opposed to E-cigarette users (~43%) [29].

Worth mentioning that GI was lowest in smokers as compared to E-cigarette users and the highest in non-smokers before the oral hygiene instructions. The analysis of previous research showed that non-smokers in all situations have higher values of GI and more prominent clinical inflammatory response in non-smokers group. Higher score of GI in non-smokers group might be related to nicotine vasoconstriction effect [30]. Several articles are consistent with these findings [31,32]. However, we found that the highest GI in E-cigarette users followed by smokers and lowest in non-smokers after the oral hygiene instructions. Earlier research that examined GI were not accounting for mechanical plaque control measures revealed pronounced inflammatory response in smokers [33]. Previous study also explored the effect of smoking on blood flow and reported a higher gingival blood circulation in smokers instead of lower, which contradict the theory that smoking impair bloods gingival blood flow, this findings might be due to the age of the participants who were between 19 and 25 years old [24]. Another research have shown that the level of MMP-8 in saliva and oral fluids is elevated in individuals with periodontitis and these levels decrease after non-surgical periodontal therapy [34].

A recent study found that the most prominent biomarkers in saliva which is doubled than other biomarkers are the MMP-8 and MMP-9 in patient with gingivitis [35]. The main findings of this study declare that MMP-8 before oral hygiene instructions is significantly higher in smokers as compared to E-cigarette smokers and non-smokers. However, after oral hygiene instructions the levels was significantly higher in smokers and E-cigarette smokers as compared to non-smokers. A plausible interpretation for this could be the detrimental effect of smoking that disrupt the vascular and inflammatory processes and affect the granulocyte activity. When facing microbial threats, granulocyte secrete high level of serine proteases, elastase, and MMP-8, which play a crucial role in periodontal tissue destruction [36]. The results of this study are in agreement with findings from other researchers, which showed an elevated levels of MMP-8 in smokers with periodontal disease in comparison to the control group [28]. Conversely, other study reported the lower level of MMP-8 in smokers than non-smokers. Due to high rate of periodontal destruction, it is surprising that smokers exhibit lower level of MMP-8. This anomaly can be explained due to presence of reactive oxygen species can trigger pro-MMPs and impair or break apart MMP-8 [37]. For vaper the only explanation for their lower MMP8 level compared to smokers is that high nicotine level affect the proinflammatory cytokines expression and affect fibroblast growth which was supported by *in vitro* study [38].

MMP-9 has been implicated in bone resorption, aiding in the digestion and breaking down of unfolded type I collagen after it had been cleaved by collagenase [39]. MMP9 has known to regulate various mediators in the beginning of inflammations, including IL-1, IL-6, IL-8, and prostaglandins [40]. The findings of this study showed that MMP-9 levels were significantly higher in cigarette smokers and E-cigarette smokers as compared to non-smokers before and after oral hygiene instructions. One possible reason that is nicotine has been found to elevate the levels of AGEs in oral tissue, such as the gingival and periodontal area [14]. This increase in AGEs enhances their interaction with receptors (RAGEs), leading to the generation of ROS and subsequent oxidative stress in the gingival tissues. This led to diminished movement and engulfing ability of polymorphonuclear cells, lowered production of antibodies, elevated bacterial attachment, and a heightened inflammatory response both systemically and locally due to raised cytokine levels in the blood and crevicular fluid [11]. Increased levels of MMP-8 and MMP-9 in saliva have highlighted localized destruction at specific sites.

To understand the cellular mechanisms behind the greater periodontal breakdown observed in E-cigarette users, additional research is required. This study has several limitations: sample size, only males were included, only 2 markers, salivary markers not crevicular fluid or blood samples. This clinical trial dependence on patient's commitment and ability to perform oral hygiene instructions that it may affect the outcome of the study. Participants' smoking or vaping behaviors may vary in intensity and frequency, making it challenging to standardize exposure levels across the study groups also Vapes are different in

their coils, amperage used, even the nectar used is different in its content which was difficult to standardize. Larger sample size and longer period might be necessary to assess periodontal parameter. Given these limitations, just as cigarette smokers are susceptible to periodontal tissue breakdown, individuals who vape e-cigarettes may also face a risk of poor periodontal tissue health. future studies should focus on Expanding the analysis to include other inflammatory markers (e.g., IL-1β, IL-6) and using additional biological samples like gingival crevicular fluid or blood could provide deeper insights, Investigating the cellular and molecular mechanisms of how vaping influences periodontal tissue breakdown could yield valuable data, such as its effects on oxidative stress, DNA damage, and fibroblast behavior. Clinicians need to educate and inform young adults about the connection between periodontal inflammation and the harmful effects of any form of tobacco use, including e-cigarette vaping. Regular counseling should be provided to help patients quit smoking and vaping. Furthermore, community health programs and anti-tobacco campaigns should actively work to raise public awareness about the adverse effects of tobacco on both oral and overall health.

Conflict of interest

The authors have no conflicts of interest to declare.

Author contributions

AAA; study conception and design. AAA; data collection. AAA and SSS.; Methodology. AAA, and JS; statistical analysis and interpretation of results. AAA, SSS and JS; original draft manuscript preparation. AAA and ASS; Writing & editing. Supervision; SSS and JS. All authors reviewed the results and approved the final version of the manuscript to be published.

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Informed consent

Informed consent was obtained from all individuals (or their guardians) who participated in this study.

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تأثير تعليمات نظافة الفم على السيتوكينات لدى المدخنين ومستخدمي السجائر الإلكترونية المصابين بالتهاب اللثة.

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المستخلص:

الخلفية: تدخين السجائر هو عامل خطر معروف لأمراض اللثة، ويؤثر على المعايير السريرية مثل النزيف عند الجس(BOP) ، ومؤشر اللويحة(PI) ، ومؤشر اللثة (GI)ومستوى السيتوكينات المؤيدة للالتهابات. تدرس هذه الدراسة تأثير استخدام السجائر الإلكترونية على السيتوكينات المؤيدة للالتهابات ومعايير اللثة. الطرق: في هذه الدراسة غير العشوائية، تم توزيع تسعين مشاركًا تم تشخيص إصابتهم بالتهاب اللثة على ثلاث مجموعات. فحص تأثير تعليمات نظافة الفم على معايير اللثة والعلامات الحيوية الالتهابية. تم تضمين ثلاثين CS (المجموعة 1)، و 29 فردًا يستخدمون السجائر الإلكترونية (المجموعة 2)، و 31 NS (المجموعة 3). تم تسجيل مؤشر اللويحة (PI)، والنزيف عند الجس(BOP) ، ومؤشر اللثة (GI) في البداية وبعد 3 أسابيع. تم استخدام اختبار الممتز المناعي المرتبط بالإنزيم لتقييم مستويات MMP-8 و MMP-9في اللعاب في البداية وبعد 3 أسابيع. النتائج: أظهر المدخنون والمستخدمون للسجائر الإلكترونية درجات أعلى بشكل ملحوظ في PI و GIو MMP8وMMP9 مقارنة بغير المدخنين قبل وبعد تعليمات نظافة الفم، مما يشير إلى التهاب دواعم السن بشكل أكبر، كما أظهر المدخنون وكذلك الأفراد الذين يستخدمون السجائر الإلكترونية انخفاضًا فيBOP ، ويرجع ذلك على الأرجح إلى أن تضييق الأوعية الدموية الناجم عن النيكوتين يخفي شدة مرض اللثة الالتهابي الفعلي. الاستنتاج: يؤثر تدخين السجائر

واستخدام السجائر الإلكترونية سلبًا على صحة اللثة، مما يزيد من درجات PI و GIو MMP8وMMP9مع تغيير .BOP تؤكد هذه النتائج على أهمية رعاية اللثة المصممة خصيصًا للمدخنين والمستخدمين للسجائر الإلكترونية من خلال دمج الإقلاع عن التدخين في بروتوكولات العلاج لتحسين نتائج صحة الفم.

الكلمات المفتاحية: التهاب اللثة، التدخين الإلكتروني، التدخين، MMP8، MMP9