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Assessment and Correlation of Salivary Ca, Mg, and pH in Smokers and Non-Smokers with Generalized Chronic Periodontitis

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Citation: Alqahtani, S.M.; Gokhale, S.T.; Elagib, M.F.A.; Shrivastava, D.; Nagate, R.R.; Alshmrani, B.A.M.; Alburade, A.M.A.; Alqahtani, F.M.A.; Nagarajappa, A.K.; Natoli, V.; et al. Assessment and Correlation of Salivary Ca, Mg, and pH in Smokers and Non-Smokers with Generalized Chronic Periodontitis. *Medicina* **2023**, *59*, 765. <https://doi.org/10.3390/medicina59040765>

Academic Editor: Gaetano Isola

Received: 6 March 2023

Revised: 12 April 2023

Accepted: 12 April 2023

Published: 14 April 2023



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Abstract: *Background and Objectives:* Diagnostic evaluation with the aid of biomarkers has reached newer heights to assess disease activity. Salivary calcium, magnesium, and pH are one of the biochemical parameters which can be helpful in assessing the progression of periodontal disease. Smokers are at topnotch threat for having oral diseases, predominantly periodontal diseases. The aim of this study was to assess the salivary calcium, magnesium, and pH levels in smokers compared with non-smokers with chronic periodontitis. *Materials and Methods:* The current study was conducted on 210 individuals affected with generalized chronic periodontitis, with the age group between 25 and 55 years. Based on their smoking habit, an equal number of patients were categorized into two groups; namely, group I consisted of non-smokers and group II consisted of smokers. The clinical parameters that were measured included Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL). The biochemical variables that were evaluated in the current study included salivary calcium, magnesium, and pH using an AVL9180 electrolyte analyzer (Roche, Germany). The gathered data were analyzed with an unpaired t test was using SPSS 20.0. *Results:* A statistically significant higher PPD ($p < 0.01$), CAL ($p < 0.05$), and salivary calcium levels ($p < 0.001$) were observed in the smokers' compared with their non-smoking counterparts. Among the biochemical parameters, calcium showed a significantly ($p < 0.001$) higher level in smokers (5.79 ± 1.76) in contrast to non-smokers (3.87 ± 1.03). Additionally, a significant negative correlation ($p < 0.05$) between calcium and PPD was observed in non-smokers, whereas a non-significant inverse relation ($p > 0.05$) was seen in smokers. *Conclusions:* The present study indicates that the salivary calcium level can be a potential biochemical parameter to assess the progression of periodontal disease in smokers and non-smokers. Within the limitations of the current study, the salivary biomarkers appear to have an essential role in the identification and indication of the status of periodontal diseases.

Keywords: periodontology; periodontitis; smokers; salivary calcium; probing pocket depth; salivary biomarkers; salivary magnesium; salivary pH; clinical dentistry

1. Introduction

The metabolic profiles of human biofluids have been used for a long time to evaluate and differentiate an individual's condition in terms of health or disease. Fluctuations have been observed in the volume and compositions of these fluids by virtue of a change in activity, drug usage, nutrition, or disease progression [1].

Oral cavities possess two prominent fluids including gingival crevicular fluid (GCF) and saliva. GCF, being in the closest proximity to gingival tissues, exhibits great potential in detecting periodontal disease and differentiating it from a healthy state [2]. Saliva has an indispensable role in various biological activities in the oral cavity and plays a pivotal role in its defense mechanism [3]. The whole saliva is a combination of fluids consisting of secretions from the major and minor salivary glands; gingival crevicular fluids; and oral mucosa transudate [4]. Thus, saliva is loaded with a variety of molecules and trace elements which make it a promising disease biomarker. Furthermore, it is easy to collect and store, as well as being easily resampled [5].

Ionomics is the study of the ionome, which is defined as a “mineral nutrient and trace element composition of an organism representing the inorganic component of the cellular and organ systems.” In recent years, salivary ionomes have emerged as a promising biomarker and thus have been projected as a vital diagnostic means to observe oral and systemic diseases. As a medium for clinical diagnosis, salivary biomarkers have a number of benefits over serum, such as the non-invasive nature of sample collection and the cost-effective approach, especially when targeting a large population [6,7].

As the main constituent, water comprises 99% of saliva, whereas the remaining 1% is made up of organic and inorganic constituents. The predominant electrolytes present in saliva include calcium, magnesium, potassium, sodium chloride, bicarbonate, and phosphate [8]. Salivary calcium has a close affinity for plaque formation that eventually influences the calculus formation. Since plaque and calculus are considered the main culprit in the etiopathogenesis of periodontal disease [9], the presence of an increased amount of calcium in saliva is known to influence plaque formation and its maturation. It has been observed that periodontally healthy participants with no marginal alveolar bone loss have a lesser potential for plaque and calculus mineralization in contrast to the patients who have been previously treated for periodontitis [10–13]. Magnesium is a known physical antagonist to calcium; however, the exact functional reciprocation in periodontitis or other risk factors associated with periodontitis, such as smoking, have not been explored. Nevertheless, there are a few studies that have shown the association of magnesium with periodontitis [14], and calcium and magnesium with periodontitis [15,16].

According to various cross-sectional and longitudinal studies, smoking is a significant risk factor for the development of periodontal disease [17,18]. Epidemiological as well as clinical studies are in alignment with the detrimental effects of smoking on periodontal tissues and, eventually, in the progression of periodontal disease that manifests as alveolar bone loss, increased probing depth, and tooth loss [19]. Additionally, it has been observed that smokers have poor oral hygiene and increased supragingival calculus formation [20]. It is well-documented that smoking induces a significant increase in the salivary flow rate as a spontaneous reflex action, which may explain the observation of increased calculus in smokers [21]. According to other research, smoking improves the mineralizing potential of saliva thus facilitating calculus formation [18].

Several studies have reported that patients with reduced bone mineral density, heavy smokers, and women in their menopausal ages have greater salivary calcium levels than age-matched peers [8,22,23]. The normal range of salivary calcium is 0.5–2.7 mmol/L [24]. In smokers, a higher level of salivary calcium is produced, which is linked to more bone loss and, accordingly, lower bone mineral density compared with non-smokers [25,26]. Salivary pH is normally between 6.2 and 7.6, with 6.7 being the average [25]. However, the pH of the oral cavity does not dip below 6.3 during rest and it is kept near neutral (6.7–7.3) by saliva [27]. Since smokers have a higher oral pH than non-smokers, there is more room

for this pH to remove calcium and deposit it on teeth, perhaps resulting in high amounts of salivary calcium [28].

There are a few studies that have stated the role of calcium, magnesium, and pH in the progression of periodontal disease [1,11,12,17]. However, there is a lack of data about the appraisal and evaluation of salivary calcium and magnesium levels in smokers and non-smokers with chronic periodontitis. Hence, the present study aims to evaluate the effect of salivary calcium and magnesium in addition to the pH levels in smoking and non-smoking chronic periodontitis patients.

2. Materials and Methods

2.1. Study Characteristics

A cross-sectional study was conducted at the College of Dentistry, King Khalid University, Abha, Saudi Arabia in the year 2019, after approval from the Institutional Ethical committee (SRC/ETH/2018-19/075). This study followed the protocol of the Declaration of Helsinki (1975) revised in 2002.

2.2. Sample Characteristics

A priori sample size calculation was performed using G* power software (Universität Düsseldorf: Psychologie—HHU) [29]. Considering t-test for comparing means of two independent study groups with equal allocation (Allocation ratio $N_2/N = 1$), an effect size (Cohen's d value) of 0.5, and a confidence interval ($1-\beta$ error) of 95% and 0.05 α , a total sample of 210 was calculated. With this sample size, the power of the study was estimated to be 0.95.

Based on the inclusion and exclusion criteria, a total of 210 chronic generalized periodontitis patients were recruited from outpatient department. Later, based on smoking status, an equal number of patients (105) were divided into the two study groups, namely Group I, consisting of non-smokers, and Group II, of smokers. The patients who smoked at least one cigarette per day in the last year were considered active smokers and were included in the study group [14]. After explaining the purpose of the study, informed consent was obtained from all the patients participating in the study.

The patients included were in the age range from 25 to 55 years, with at least 20 permanent teeth. Patients who were clinically diagnosed with chronic periodontitis presented with an evident bone loss on radiographical assessment and with a Probing Pocket Depth (PPD) of ≥ 4 mm with a Clinical Attachment Loss (CAL) of ≥ 1 mm. Patients who gave a history of periodontal therapy in last 6 months and had taken antibiotic coverage in last 3 months were excluded. Along with this, the patients on medications who were affected with a chronic disease which has influence on periodontal parameters were excluded from the study. Patients having xerostomia, either due to systemic or local conditions, were also excluded, as this could influence the periodontal conditions.

2.3. Study Protocol and Clinical Parameters Measured in the Study

A pre-designed data extraction sheet was used to collect information regarding demographic data and details such as medical history and oral hygiene practices. The clinical parameters including Loe and Silness Gingival Index (GI) [30], Bleeding on probing (BOP), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL) were used for the assessment of the clinical condition. To lessen the bias, the measurements of all clinical parameters were documented and taken by a single examiner, who was initially calibrated. The intra-examiner reliability of the examiner for all the coding was 0.88, which was of good agreement. Plaque Index was measured after giving erythrosine in the form of a chewing tablet. BOP and CAL were assessed using a specific periodontal probe (UNC-15, Hu-friedy, Chicago, IL, USA). PPD was recorded from the gingival margin to the gingival sulcus base, while CAL was recorded from cemento-enamel junction (CEJ) to the base of the gingival sulcus.

2.4. Collection of Salivary Sample and Its Laboratory Analysis

A saliva sample was obtained after clinical recordings. A 2 mL of unstimulated whole saliva was collected by the “spitting method” as described by Navazesh M. (1993) [31]. To correspond to the circadian rhythm, salivary samples were collected 2 h after the last meal, after rinsing with water for 5 min. Patient was instructed to spit the saliva gathered in the floor of the mouth into the collecting unit. To avoid time-related alteration in pH of saliva, it was collected immediately. The samples were then sent to the laboratory within 24 h, with temperatures maintained at 2 to 4 degrees Celsius. Salivary pH was measured using pH litmus test paper. AVL9180 electrolyte analyzer (Roche, Germany) was used for measuring calcium and magnesium ions.

2.5. Data Analysis

The data collected were analyzed using statistical package of social sciences (SPSS) 20.0 version (IBM; Chicago, IL, USA). The gathered data were initially checked for normality with Kolmogorov–Smirnov test and visualization methods including histogram and Q-Q plots. All the variables tested in the current study were found to be normally distributed ($p > 0.05$). Results were expressed as means and standard deviation. Based on the normality distribution of the data, parametric test–Unpaired t test was used to compare the clinical and biochemical parameters between the study groups. Correlation among the clinical and biochemical were analyzed using Pearson’s and Spearman correlations for parametric and categorial type of variable, respectively.

3. Results

3.1. Sample Characteristics

There was no significant difference ($p > 0.05$) in the age and gender distribution between the groups, with smokers having a mean age of 42.1 ± 2.3 years and non-smokers having a mean age of 45.8 ± 3.4 years (Table 1).

Table 1. Sample Characteristics.

Variable	Categories	Study Group		p Value
		Group I (Non-Smoker) n = 105	Group II (Smoker) n = 105	
Age (Mean ± SD)		45.80 ± 3.46	42.08 ± 6.19	0.573
Gender †	Male	57 (54)	59 (56)	0.62
	Female	48 (46)	46 (44)	

Note: †—results expressed in Number (%); SD—Standard Deviation.

3.2. Comparative Analysis of Clinical Parameters between the Study Groups

There is a significantly higher PPD ($p < 0.05$) and CAL ($p < 0.01$) in the smoker group compared with the non-smoker patients. However, non-significant ($p > 0.05$) differences in PI and GI were observed in the study group when compared with the control group patients. (Table 2).

Table 2. Comparative analysis of clinical parameters among the study group.

Clinical Parameter	Group I	Group II	p Value
Plaque Index	1.71 ± 0.48	1.51 ± 0.34	0.109
Gingival Index	1.55 ± 0.38	1.67 ± 0.29	0.222
Periodontal Probing Depth	5.57 ± 1.02	6.16 ± 0.77	0.025 *
Clinical Attachment Level	5.08 ± 0.73	5.70 ± 0.67	0.003 **

Note: results expressed in Mean ± Standard Deviation; * $p < 0.05$; ** $p < 0.01$.

3.3. Comparative Analysis of Biochemical Parameters between the Study Groups

Among the biochemical parameters, significantly ($p < 0.001$) raised calcium levels (5.79 ± 1.76 mmol/L) were observed in smokers when compared with non-smokers (3.86 ± 1.03 mmol/L). However, magnesium did not show any difference between the groups. (Table 3)

Table 3. Comparative analysis of biochemical parameters among the study group.

Parameter	Group I	Group II	p Value
pH	6.44 ± 0.86	6.80 ± 0.91	0.160
Calcium	3.86 ± 1.03	5.79 ± 1.76	0.000 ***
Magnesium	0.54 ± 0.18	0.49 ± 0.24	0.413

Note: results expressed in Mean \pm Standard Deviation; *** $p < 0.001$.

3.4. Correlational Analysis of Biochemical and Clinical Parameter in the Study Groups

Depending on the type of variable (parameteric Vs. non-parameteric), Pearson and Spearman correlation analysis was carried out for all variables in both study groups.

The two crucial periodontal clinical indicators, namely PPD and CAL, showed a highly significant ($p < 0.001$) positive correlation in both study groups. Additionally, PPD and CAL were later analyzed with calcium and magnesium. Similarly, the gingival parameters, namely PI and GI, showed a significant ($p < 0.001$) positive correlation in Group I and a positive but non-significant ($p > 0.05$) correlation in Group II. These correlation results reaffirm the presentation of periodontal diseases.

Another key parameter, the pH of the saliva, showed a significant ($p < 0.05$) negative correlation with magnesium in Group I and a significant ($p < 0.05$) negative correlation with calcium in Group II.

The correlation analysis between salivary calcium and periodontal clinical parameters such as CAL and PPD was carried out in each study group. In the control group (Non-smoker), a significant ($p < 0.05$) negative correlation was found between calcium and PPD and CAL. However, a non-significant ($p > 0.05$) positive correlation was seen between the parameters in the study group (Tables 4 and 5) (Figures 1 and 2).

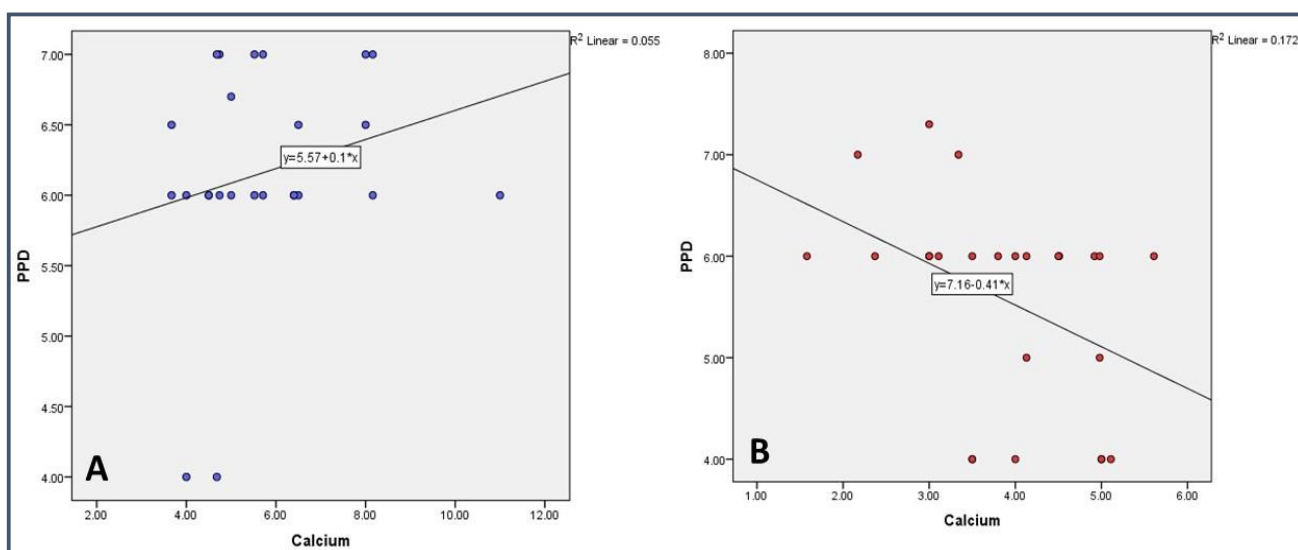


Figure 1. Correlational analysis of Calcium with Periodontal Probing Depth in (A) Smokers and (B) Non-Smokers.

Table 4. Correlation analysis of parameters in Group I.

	Ca	Mg	pH	PI	GI	PPD	CAL	Age	Gender ¶
Ca	-	0.985 (0.004)	0.243 (0.237)	0.978 (−0.006)	0.994 (0.002)	0.039 * (−0.415)	0.043 * (−0.407)	0.415 (−0.171)	0.490 (−0.145)
Mg	0.985 (0.004)	-	0.037 * (−0.419)	0.306 (0.213)	0.671 (0.089)	0.855 (−0.038)	0.789 (−0.056)	0.176 (0.280)	0.853 (−0.039)
pH	0.243 (0.237)	0.037 * (−0.419)	-	0.105 (0.332)	0.068 (0.371)	0.423 (0.168)	0.370 (0.187)	0.616 (0.105)	0.688 (0.084)
PI	0.978 (−0.006)	0.306 (0.213)	0.105 (0.332)	-	0.000 *** (0.783)	0.521 (0.135)	0.570 (0.119)	0.677 (0.088)	0.739 (0.070)
GI	0.994 (0.002)	0.671 (0.089)	0.068 (0.371)	0.000 *** (0.783)	-	0.902 (0.026)	0.865 (−0.036)	0.064 (0.376)	0.711 (0.078)
PPD	0.039 * (−0.415)	0.855 (−0.038)	0.423 (0.168)	0.521 (0.135)	0.902 (0.026)	-	0.000 *** (0.937)	0.834 (0.044)	0.725 (0.074)
CAL	0.043 * (−0.407)	0.789 (−0.056)	0.370 (0.187)	0.570 (0.119)	0.865 (−0.036)	0.000 *** (0.937)	-	0.791 (−0.056)	0.669 (0.090)
Age	0.415 (−0.171)	0.176 (0.280)	0.616 (0.105)	0.677 (0.088)	0.064 (0.376)	0.834 (0.044)	0.791 (−0.056)	-	0.811 (0.050)
Gender	0.490 (−0.145)	0.853 (−0.039)	0.688 (0.084)	0.739 (0.070)	0.711 (0.078)	0.725 (0.074)	0.669 (0.090)	0.811 (0.050)	-

Note: results are expressed as *p* value (correlation coefficient); * *p* < 0.05; *** *p* < 0.001; ¶—Spearman Correlation; Ca—Calcium; Mg—Magnesium; PI—Plaque Index; GI—Gingival Index; PPD—Probing Pocket Depth; CAL—Clinical Attachment Loss.

Table 5. Correlation analysis of parameters in Group II.

	Ca	Mg	pH	PI	GI	PPD	CAL	Age	Gender ¶
Ca	-	0.614 (−0.106)	0.003 ** (−0.572)	0.403 (0.175)	0.343 (0.198)	0.260 (0.234)	0.385 (0.182)	0.058 (0.385)	0.692 (0.083)
Mg	0.614 (−0.106)	-	0.488 (−0.145)	0.580 (0.116)	0.426 (−0.167)	0.667 (−0.090)	0.693 (−0.083)	0.473 (0.151)	0.145 (0.300)
pH	0.003 ** (−0.572)	0.488 (−0.145)	-	0.089 (0.347)	0.201 (0.265)	0.850 (−0.040)	0.976 (−0.006)	0.772 (−0.061)	0.286 (−0.222)
PI	0.403 (0.175)	0.580 (0.116)	0.089 (0.347)	-	0.068 (0.371)	0.444 (0.160)	0.327 (0.204)	0.270 (0.229)	0.453 (0.157)
GI	0.343 (0.198)	0.426 (−0.167)	0.201 (0.265)	0.068 (0.371)	-	0.632 (0.101)	0.492 (0.144)	0.033 * (0.427)	0.689 (0.084)
PPD	0.260 (0.234)	0.667 (−0.090)	0.850 (−0.040)	0.444 (0.160)	0.632 (0.101)	-	0.000 *** (0.963)	0.386 (0.181)	0.098 (−0.338)
CAL	0.385 (0.182)	0.693 (−0.083)	0.976 (−0.006)	0.327 (0.204)	0.492 (0.144)	0.000 *** (0.963)	-	0.388 (0.180)	0.054 (−0.390)
Age	0.058 (0.385)	0.473 (0.151)	0.772 (−0.061)	0.270 (0.229)	0.033 * (0.427)	0.386 (0.181)	0.388 (0.180)	-	0.222 (0.253)
Gender ¶	0.692 (0.083)	0.145 (0.300)	0.286 (−0.222)	0.453 (0.157)	0.689 (0.084)	0.098 (−0.338)	0.054 (−0.390)	0.222 (0.253)	-

Note: results are expressed as *p* value (correlation coefficient); ¶—Spearman Correlation; * *p* < 0.05; ** *p* < 0.01; *** *p* < 0.001; Ca—Calcium; Mg—Magnesium; PI—Plaque Index; GI—Gingival Index; PPD—Probing Pocket Depth; CAL—Clinical Attachment Loss.

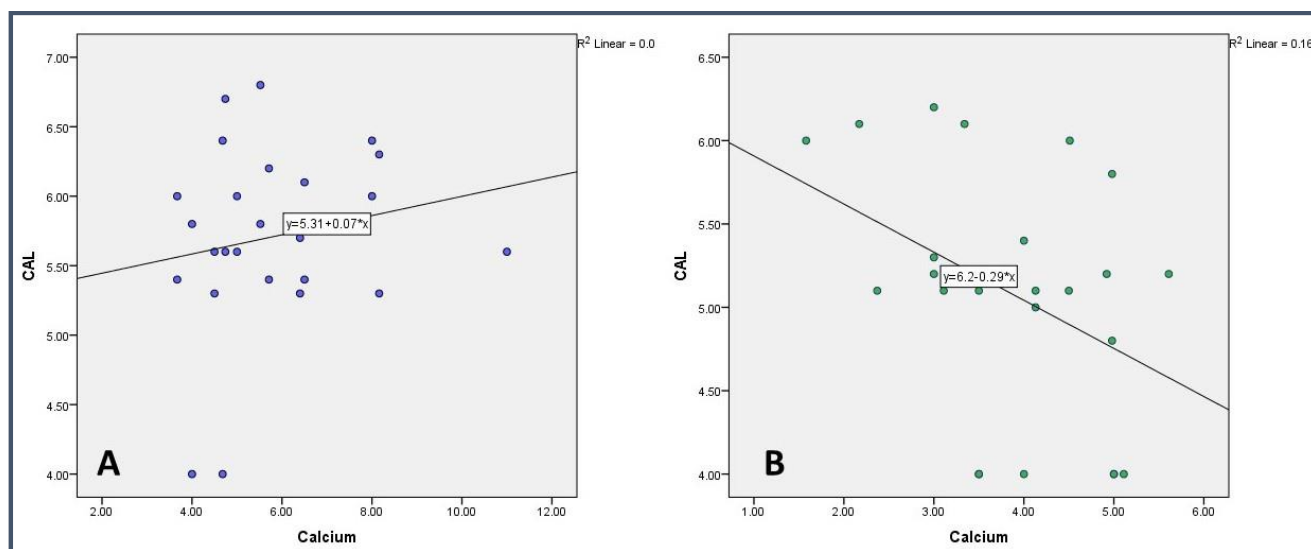


Figure 2. Correlational analysis of Calcium with Clinical Attachment Loss in (A) Smokers and (B) Non-Smokers.

4. Discussion

With the advancement of research, the metabolites profiling of a biological system has been commonly utilized to provide insight into the normal and disconcert metabolic processes [32]. Salivary metabolites can act as a biomarker to understand the complex biochemical interaction of host and bacteria in periodontal diseases [33–35]. It has been proven with various studies that tobacco smoke can alter the biochemical composition, and subsequently the function, of saliva [36,37].

Salivary Ca and Mg can be considered imperative in periodontal health concerning their influence on plaque mineralization. Magnesium may play an important role in preventing periodontal disease as it has a unique ability to reduce inflammation caused by bacterial toxins [18]. A group of studies reported that reduced magnesium concentrations are associated with an enhanced inflammatory response to bacterial challenges, thus promoting periodontitis [15,38]. Conversely, Manea et al. reported that salivary Mg concentrations were significantly higher in the periodontitis group compared with the controls. In another study, it was observed that salivary Mg concentrations were higher in smokers with periodontitis than in non-smokers who were also affected by periodontitis [38]. Although in the present study reduced Mg levels were reported in the smoker group compared with the non-smokers, the difference was non-significant. Similarly, Mg levels showed a non-significant negative correlation with PPD and CAL in both groups. A similar correlation was reported between Mg and periodontal parameters in the study conducted by Erdemir EO et al. [39].

Smokers have been classified as light smokers who smoke one–ten cigarettes a day; moderate smokers who smoke eleven–twenty cigarettes a day; and heavy smokers who smoke more than twenty cigarettes a day [14,40]. Smoking is thought to increase salivary Ca levels independently by reducing skeletal bone density [40]. The literature highlights the increased Ca levels in periodontitis patients [31,35]. However, it is important to note that dietary calcium intake and overall calcium turnover can influence salivary calcium levels [41]. In addition, the continuous exposure of taste receptors to tobacco products such as nicotine probably affects salivary flow rate [42], salivary reflex, and also salivary Ca levels [8]. Smokers have fairly eminent levels of salivary calcium, which is allied with a greater degree of bone loss and lower bone mineral density than non-smokers. The present study showed significantly elevated Ca levels in smokers when compared with non-smokers. A study by Megha Varghese et al. and Kolte et al. reported analogous findings in a sample of periodontitis patients, with salivary calcium ranging higher in the

smoker group than in non-smokers [43,44]. Gupta VV et al. also observed concordant findings in their study, wherein calcium level was increased in smokers diagnosed with aggressive periodontitis [45]. This was contradictory to the study of Ivana Sutej et al. and Shashikanth et al. who found no difference in calcium levels between smokers and non-smokers [28,46]. A study conducted by Zuabi et al. observed a reduction in calcium levels post treatment of periodontitis patients [47]. A higher calcium level was observed in the stimulated saliva of smokers in studies conducted by Sevón et al. [48] and Mc Gregor et al. [25]. According to Sevón et al., the decreased bone mineral density, a side effect of smoking, could be a reason for high salivary calcium [48].

The normal salivary pH ranges from 6.2 to 7.6. The buffering capacities of saliva and salivary flow both have an impact on salivary pH [37]. It was observed in one of the studies that salivary pH was lower in periodontitis patients compared with healthy controls. There was no significant difference in pH readings amongst the groups, although it was more acidic in the smokers' group [27]. Similar findings were observed in a study conducted by Kumar et al. which found a lower pH in smokers with periodontitis [49]. In contrast, the study of Gupta VV et al. showed a significant increase in pH levels in smokers against healthy controls [45] which could be due to the different technique adopted for the collection of saliva. However, the present study did not establish any significant difference in pH between the groups. The current study utilized the unstimulated saliva collection procedure as it bathes the oral cavity predominantly and moistens the oral cavity round the clock. Furthermore, it also represents the pooled sub-gingival plaque sample [50]; whereas, in other studies, stimulated saliva was collected [45]. Additionally, in a study conducted to evaluate the pH of smokers with traditional smoking and e-cigarette smoking and non-smokers, it was found that the traditional smokers and e-cigarette smokers had a lower pH than non-smokers [37].

On comparing the clinical parameters, such as PPD and CAL, smokers had more PPD and CAL compared with non-smokers with periodontitis. A similar observation was noticed by Haffajee AD et al. [51], Shashikanth H et al. [46], and Velidandla S et al. [52]. On comparing the Plaque Index, no difference was found between the groups. A similar observation was noticed in a study conducted by Sreedevi et al. [53]. When the Gingival Index was compared between the groups, no statistically significant difference was found. A similar finding was reported in other studies [54]. However, this result is contradictory to another study conducted by Zuabi et al. [47]. In a study conducted by Erdemir et al., they found that, in smokers, there was a positive correlation between the levels of Ca, Mg, and CAL. Whereas, in the non-smoker group, there was a negative correlation between the mean level of sodium and the Plaque Index ($p < 0.05$) [39]. In our study, we found that, in the non-smokers, there was a significant negative correlation between calcium and PPD and CAL. However, a non-significant ($p > 0.05$) positive correlation was seen between the parameters in the smokers' group. The difference in the study could be because of the assessment method as in the previous study inductively coupled plasma-atomic emission spectrophotometry was used. However, in the present study, an AVL9180 electrolyte analyzer was used for assessment.

Limitations and Future Directions

Within the limitations of this study, confounding factors such as the presence of calcium in the diet and differences in age were not addressed in this study. Therefore, longitudinal studies are recommended for establishing the causal relationship between the parameters. This will also aid the scientific society in winding up the judgment against the role of saliva in the initiation and progression of periodontal disease.

5. Conclusions

Among all the constituents of saliva, salivary calcium is one of the most extensively studied potential markers for the identification of periodontal diseases. The present study draws attention towards the specific risk factors that could influence the pathogenesis of

periodontal disease, amid which smoking is a prompt factor. Smoking also serves as an indirect biomarker for periodontal lesion predilection. The results of the current study indicate that smokers have significantly higher PPD, CAL, and calcium than their non-smoking counterparts. Importantly, salivary calcium was found to be elevated in smokers with chronic generalized periodontitis, thus the attempts to signify that calcium levels in saliva act as both a risk factor and imminent biochemical marker for the assessment of periodontal lesions.

Author Contributions: Conceptualization, S.M.A., S.T.G., M.F.A.E. and R.R.N.; methodology, S.M.A., S.T.G., M.F.A.E., R.R.N. and B.A.M.A.; software, B.A.M.A., A.M.A.A., F.M.A.A. and D.S.; validation, B.A.M.A., A.M.A.A., F.M.A.A. and D.S.; formal analysis, D.S. and K.C.S.; investigation, S.M.A., S.T.G., M.F.A.E., R.R.N. and B.A.M.A.; data curation, K.C.S., V.N. and A.K.N.; writing—original draft preparation, S.M.A., S.T.G., D.S. and K.C.S.; writing—review and editing, S.M.A., S.T.G., M.F.A.E., R.R.N., B.A.M.A., A.M.A.A., F.M.A.A., D.S.; A.K.N., V.N. and K.C.S.; visualization, R.R.N., B.A.M.A., A.M.A.A. and F.M.A.A.; supervision, S.M.A.; project administration, S.M.A., K.C.S. and A.K.N.; funding acquisition, B.A.M.A., A.M.A.A., F.M.A.A., A.K.N., V.N. and K.C.S. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: This study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of King Khalid University, College of Dentistry (SRC/ ETH/2018-19/075). Date of access: 19/3/2019.

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

Data Availability Statement: The data are available on a reasonable request from the corresponding author.

Acknowledgments: The authors extend their appreciation to the Deanship of Scientific Research at King Khalid University for funding this work through Small Groups Project under grant number RGP.1/351/43.

Conflicts of Interest: The authors declare no conflict of interest.

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