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Original Research Article

Evaluation of salivary lymphocyte function associated antigen – 1 (LFA-1) level in smokers and non-smokers with periodontitis – A cross-sectional study

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Abstract

Aim and Objective: To assess the salivary levels of LFA-1 in patients with and without smoking who have periodontitis and those who are in good oral health.

Introduction: LFA-1 is a non-specific salivary biomarker, which plays an important role in determining the periodontal health status of an individual.

Materials and Methods: Periodontal screening was done and the subjects were divided into three different groups: Group 1- Periodontally healthy subjects, Group 2 – Periodontitis subjects without smoking habit, and Group 3 – Periodontitis subjects with smoking habit with 24 subjects in each group. Samples of unsimulated saliva were gathered, centrifuged for 12 minutes at 2700 rpm, and the supernatant was collected and kept at -80 °C. The Human LFA-1 Enzyme Linked Immunosorbent Assay (ELISA) kit was used to process the samples. A microplate reader tuned to 450 nm was used to measure each well's optical density.

Result: In contrast to the periodontally healthy group (0.1708 ng/ml) and the periodontitis participants who did not smoke (0.5602 ng/ml), the salivary levels of LFA-1 were considerably greater in the smoking-related group (0.6594 ng/ml). Salivary LFA-1 levels significantly increased by 0.314 ng/ml for every 1 mm rise in Clinical Attachment Level (CAL) in the smoking-associated periodontitis group. This difference was statistically significant at $p<0.001$.

Conclusion: The salivary LFA-1 levels were found to be significantly higher in periodontitis subjects with smoking habits when compared to periodontitis subjects without smoking habits and periodontally healthy subjects which correlate with the clinical attachment level.

Keywords: Periodontitis; Biomarker; Lymphocyte Function-Associated Antigen-1

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1. Introduction

A chronic inflammatory multifactorial illness that affects the teeth's supporting components is called periodontitis. The interplay between the subgingival microbial colonies and the host immune response results in periodontitis. A number of factors, including acquired risk factors and environmental factors, influence the severity of periodontal disease. Smoking has a negative effect on treatment results and is a strong risk factor for the development of periodontal disease.[1] In periodontal health, bacterial colonies and the host immune inflammatory response are in balance; this equilibrium is disrupted in periodontitis.[2] Smoking exacerbates periodontal disease by altering the immune system.

Biomarkers are present in various forms such as ribonucleic acid, Deoxyribonucleic acid, micro-organisms, proteins, etc. Biomarkers are present in saliva, serum, and Gingival crevicular fluid (GCF).[3] Saliva is the most commonly used source for biomarkers than other sources because the collection of saliva is rapid, simple, and painless. Salivary biomarkers are reliable predictors which is used to diagnose the current periodontal health status and facilitate to identify the future plot of periodontal risk. Saliva is considered as a "mirror to the body" because of the presence of similar proteins in both serum and saliva.[4]

Quantitative alteration of biomarkers is associated with the onset, advancement, and resolution of periodontal disease. Salivary biomarker analysis helps in differentiating the periodontal health of an individual from periodontal disease conditions. The neutrophils are the first line of defence cells that reach the site of inflammation or injury by trans-endothelial migration. The adhesion molecules Lymphocyte Function associated Antigen-1 (LFA-1) and Intercellular Adhesion molecule-1 (ICAM-1) are crucial for

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the recruitment of neutrophils during trans-endothelial migration.[5]

A non-specific salivary biomarker called LFA-1 is crucial in assessing a person's level of periodontal health. A transmembrane protein found on lymphocytes, LFA-1 functions as an adhesion molecule that, by connecting with ICAM-1 produced on the endothelium, aids in stopping neutrophil rolling during trans-endothelial migration. LFA-1 expression is observed to be elevated in groups with chronic periodontitis and generalized aggressive periodontitis, downregulated in the group with gingivitis, and marginally higher in the group with periodontally healthy teeth.[6]

With the above-stated findings, we had an interest in evaluating the salivary levels of LFA-1 in periodontally healthy subjects, and periodontitis subjects with and without smoking habit.

2. Materials and Methods

72 participants participated in this cross-sectional study, which was presented to the Institutional Review Board for protocol approval and received ethical clearance from the Institutional Ethics Committee. All subjects provided written informed consent following a thorough explanation of the study's necessity. Totally 72 willing participants were enrolled and divided into three main groups: Group 1 (n=24) - Periodontally healthy subjects, Group 2 (n=24) – Non-smoker patients with Periodontitis, and Group 3 (n=24) – Smoker patients with Periodontitis. Clinical parameters like the Plaque index (PI), Gingival Bleeding Index (GBI), Probing pocket depth (PPD), and Clinical attachment level (CAL) were recorded prior to salivary sample collection.

In group 1, 24 consenting subjects of age ranging from 30 to 60 who have at least 20 natural teeth and are in good general health were enrolled. Inclusion criteria comprised of probing depth not more than 3 mm, bleeding on probing not more than 10% at six sites with no clinical attachment loss. In Group 2, 24 willing subjects of age ranging from 30 to 60 who have at least 20 natural teeth and are in good general health were included. Inclusion criteria include probing depth of >3 mm at six sites surrounding the tooth with clinical attachment loss ≥ 3mm detected at more than or equal to 2 natural teeth and in Group 3, 24 willing subjects of age ranging from 30 to 60 who have at least 20 natural teeth and in good overall health were included. Inclusion criteria include probing depth of ≥ 3 mm at six sites surrounding the tooth with clinical attachment loss ≥ 3mm detected at more than or equal to 2 natural teeth and current smokers according to Centre for Disease Control (CDC) criteria.[7]

Participants those who recently had immunosuppressive medications, antibiotics, and Non – Steroidal anti-inflammatory drugs (NSAIDs) for a period of past 6 months, history of periodontal treatment past 6 months, patients having systemic diseases like diabetes mellitus and Pregnant women and Lactating mothers were excluded from this study.

5 mL unstimulated saliva sample was collected from all subjects involved in this study. The participants were advised to rinse their mouth completely with water and then asked to expectorate unstimulated saliva into a 15 ml disposable centrifuge tube which is made up of Polypropylene (**Table 1**). Collected samples were immediately centrifuged at 2700 rpm for 12 minutes (**Table 2**). The supernatant obtained was collected in a 2 ml plastic storage vials (**Table 3**) and immediately stored at −80° C until biochemical analysis of LFA-1 was performed.

Before processing, every reagent that was kept in the deep freezer was warmed to room temperature. Standard preparation: To create a 6ng/ml standard stock solution, 120µl of the standard (12.8ng/ml) was reconstituted with 120µl of standard diluent. It was then let to sit for 15 minutes with mild stirring before dilutions were made. In order to create 3ng/ml, 1.5ng/ml, 0.75ng/ml, and 0.375ng/ml solutions, duplicate standard points were made by diluting the standard stock solution (6ng/ml) 1:2 with standard diluent. (**Table 4**).

Wash buffer: To obtain 1x wash buffer, 20 ml of concentrated wash buffer (25x) was diluted with 500 ml of distilled water. 0.05ml of the standard was added to the standard well. The sample wells were added with 0.04ml of sample to which 0.01ml of Human LFA-1 antibody was added. To both the standard and sample wells 0.05ml of streptavidin-HRP was added (**Table 5**). The samples were mixed well and covered using a sealer which was incubated at 37 degrees Celsius for one hour. The plate was washed with a minimum of 0.30 ml prepared wash buffer solution for a duration of 1 minute for 5 times. 0.05ml of substrate solution A was added to each well followed by the addition of 0.05ml of substrate solution B. The plate was covered with a new sealer at 37 degrees Celsius for 10 minutes in the dark. 0.05ml of stop solution was added in all the wells and the color changed from blue to yellow immediately.(**Figure 6**) A microplate reader set to 450 nm for each well was used to read the optical density (OD value) within 10 minutes of the color change. (**Figure 7**)

2.1. Statistical analysis

To perform the statistical analysis, the Statistical Package for Social Sciences (SPSS) released in 2003 for Windows version 22.0 was developed by Armonk, NY: IBM Corporation.

2.2. Descriptive statistics

A descriptive analysis of all the explanatory and outcome parameters was performed using frequency and proportions for categorical variables and mean and standard deviation for continuous variables.

2.3. Inferential statistics

The data distribution was examined for normality using the Shapiro-Wilk test, and the results showed that all clinical parameters had a normal distribution while salivary LFA-1

levels had a non-normal distribution. As a result, pertinent parametric and non-parametric tests were used for the inferential testing. The gender distribution of the three groups was compared using the chi-square test. The clinical parameters of the three groups were compared using a one-way ANOVA test and Tukey's post hoc test. The mean CAL for Groups 2 and 3 was compared using the Independent Student t Test. The mean age and salivary LFA-1 levels were compared between the three groups using the Kruskal Wallis test and Dunn's post hoc analysis. To evaluate the association between each group's clinical and salivary LFA-1 levels, the Spearman's Rank correlation test was used. Clinical parameters in each group were utilized to predict the salivary LFA-1 levels using stepwise multiple linear regression analysis. A significance threshold of P<0.05 was established.

3. Results

Table 1 represents the gender distribution and age mean and standard deviation for each of the three groups. The mean values were compared using the Chi-square test and Kruskal-Walli's test, respectively. The mean age of individuals in groups I, II, and III were 43.25, 45.25, and 45.21 respectively. In group 1 (Periodontally Healthy), 19 (79.2%) were males and 5 (20.8%) were females whereas in group 2 (Periodontitis without smoking habit), 18 (75%) were males and 6 (25%) were females. In the case of group 3 (Periodontitis with smoking habit), 24 (100%) were males.

Table 2 denotes the mean and standard deviation of the Plaque index (PI), Gingival Bleeding Index (GBI), Probing depth (PD), and Clinical attachment level (CAL) among the three groups. Comparison of mean values of different clinical parameters between three groups using One-way ANOVA Test followed by Tukey's post hoc Test shows the mean PI score for Group 1 (Periodontally Healthy) was 0.031± 0.16, Group 2 (Periodontitis without smoking habit) was 0.103 ± 1.74 and Group 3 (Periodontitis with smoking habit) was 0.116 ± 1.73. This difference in the mean PI scores between the 3 groups was statistically significant at p<0.001. When comparing the groups, multiple comparisons showed that the smoking group with periodontitis had a considerably higher mean PI score than the Non-smoker and periodontally healthy groups. This difference was statistically significant at p<0.001. Subsequently, the Nonsmoking Periodontitis group demonstrated a considerably higher PI score than the Periodontally Healthy group, with the difference being statistically significant at p<0.001.

Table 3 denotes the mean salivary LFA-1 (ng/ml) levels in groups I, II, and III. The mean Salivary LFA-1 (ng/ml) level was compared between three groups using the Kruskal Wallis Test followed by Dunn's post hoc Analysis. Group III, subjects with periodontitis and a smoking habit had higher salivary levels of LFA-1 (0.6594 ng/ml) than group II, subjects with periodontitis but no smoking habit (0.5602 ng/ml) and group I, subjects with periodontal health (0.1708 ng/ml). With a p-value < 0.001, it was determined that the variations in LFA-1 salivary levels across groups were significant.

Table 4 denotes Spearman's correlation test to assess the relationship between Salivary LFA-1 (ng/ml) levels, and clinical parameters in each group. In Group 2(Periodontitis subjects without smoking habit), Salivary LFA-1 levels showed a significant moderate positive correlation with GI, PPD & CAL at rho=0.48, 0.52 & 0.58 respectively and the findings were statistically significant at p=0.02, p=0.009 & p=0.003. Similarly, in Group 3(Periodontitis subjects with smoking habit), the Salivary LFA-1 levels showed a significant moderate positive correlation with PI & GI at rho= 0.44 & 0.53 and the relationship was statistically significant at p=0.03 & p=0.007 and significant strong positive correlation with PPD & CAL at rho= 0.61 & 0.71 and the relationship was statistically significant at p=0.002 & p<0.001 respectively.

Table 5 denotes stepwise multiple linear regression analysis for predicting Salivary LFA-1 levels using Clinical Parameters in each group. In Group 2(Non-smoker Periodontitis patients), for every 1 mm increase in CAL, there was a significant increase in Salivary LFA-1 levels by 0.218 ng/ml and the finding was statistically significant at p<0.001. The variability in Salivary LFA-1 levels in Group 3(Periodontitis subjects with smoking habit), can be attributed to a 35% variation in CAL levels. In Group 3, for every 1 mm increase in CAL, there was a significant increase in Salivary LFA-1 levels by 0.314 ng/ml and the finding was statistically significant at p<0.001. The variability in Salivary LFA-1 levels in Group 3, can be attributed to 48% variation in CAL levels.

Table 1: Age (years) and gender distribution between the groups

Age and gender distribution among 3 study groups								
Variable	**Category**	**Group 1**		**Group 2**		**Group 3**		**p-value**
		Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	
Age	**Mean**	43.25	1.73	45.25	5.76	45.21	5.60	0.24[a]
	Range	39 - 46		33 - 55		34 - 53		
		n	**%**	**n**	**%**	**n**	**%**	
Sex	**Males**	19	79.2%	18	75.0%	24	100.0%	0.08[b]
	Females	5	20.8%	6	25.0%	0	0.0%	

Note: a. Kruskal Wallis Test; b. Chi Square Test

Table 2: Comparison of the groups' clinical attachment level (CAL), probing pocket depth (PPD), gingival bleeding index (GBI), and plaque index (PI)

Comparison of mean values of different clinical parameters between 03 groups using One-way ANOVA Test followed by Tukey's post hoc Test									
Variables	**Groups**	**N**	**Mean**	**SD**	**Min**	**Max**	**p-value [a]**	**Sig. Diff**	**p-value [b]**
PI	Group 1	0.210	0.031	0.16	0.27	0.210	<0.001*	G1 Vs G2	<0.001*
	Group 2	1.995	0.103	1.74	2.22	1.995		G1 Vs G3	<0.001*
	Group 3	2.000	0.116	1.73	2.21	2.000		G2 Vs G3	0.98
GBI	Group 1	9.04	1.16	7	10	9.04	<0.001*	G1 Vs G2	<0.001*
	Group 2	69.33	6.25	55	79	69.33		G1 Vs G3	<0.001*
	Group 3	69.17	6.43	56	79	69.17		G2 Vs G3	0.99
PPD	Group 1	1.108	0.056	1.01	1.21	1.108	<0.001*	G1 Vs G2	<0.001*
	Group 2	3.139	0.564	1.98	3.92	3.139		G1 Vs G3	<0.001*
	Group 3	3.146	0.280	2.67	3.64	3.146		G2 Vs G3	0.99

* - Statistically Significant, PI – Plaque Index, GBI – Gingival Bleeding Index, PPD – Pocket Probing Depth

Note: a. p-value obtained using One-way ANOVA Test; b. p-value obtained using Tukey's Post hoc Test

Comparison of mean CAL between Group 2 & Group 3 using Independent Student t Test						
Parameter	**Groups**	**N**	**Mean**	**SD**	**Mean Diff**	**p-value**
CAL	Group 2	24	4.025	0.529	-0.137	0.30
	Group 3	24	4.162	0.353		

CAL - Clinical Attachment Level

Table 3: Comparison of salivary LFA-1 levels between the groups

Salivary LFA-1 (ng/ml) mean values in three groups were compared using the Kruskal Wallis Test and Dunn's post hoc analysis.									
Variables	**Groups**	**N**	**Mean**	**SD**	**Min**	**Max**	**p-value[a]**	**Sig. Diff**	**p-value[b]**
Salivary LFA-1 (ng/ml)	Group 1	24	0.1708	0.0573	0.071	0.324	<0.001*	G1 Vs G2	<0.001*
	Group 2	24	0.5602	0.2049	0.233	1.222		G1 Vs G3	<0.001*
	Group 3	24	0.6594	0.1566	0.348	0.924		G2 Vs G3	0.03*

* - Statistically Significant, LFA – Lymphocyte Function associated Antigen

Note: a. p-value derived by Kruskal Wallis Test; b. p-value derived by Dunn's post hoc Test

Table 4: Spearman's correlation test to assess Salivary LFA-1 (ng/ml) levels

The relationship between Salivary LFA-1 (ng/ml) levels, clinical parameters in each group is assessed using Spearman's correlation test						
Group	**Variable**	**Values**	**PI**	**GBI**	**PPD**	**CAL**
Group 1	HIF-1α	rho	-0.04	-0.35	-0.18	.
		p-value	0.85	0.09	0.40	.
Group 2	HIF-1α	rho	0.39	0.48	0.52	0.58
		p-value	0.06	0.02*	0.009*	0.003*
Group 3	HIF-1α	rho	0.44	0.53	0.61	0.71
		p-value	0.03*	0.007*	0.002*	<0.001*

* - Statistically Significant, PI – Plaque Index, GBI – Gingival Bleeding Index, PPD – Pocket Probing Depth, CAL – Clinical Attachment Level

Table 5: Salivary LFA-1 (ng/ml) levels are evaluated using stepwise multiple linear regression.

Stepwise multiple linear regression analysis for predicting Salivary LFA-1 levels using Clinical Parameters in each group							
Group	**DV**	**IV**	**β**	**SE**	**t**	**p-value**	**R^2**
Group 2	Salivary LFA-1	Constant	-0.316	0.277	-1.14	0.27	0.35
		CAL	0.218	0.068	3.187	0.004*	
Group 3	Salivary LFA-1	Constant	-0.646	0.280	-2.305	0.03*	0.48
		CAL	0.314	0.067	4.676	<0.001*	

* - Statistically Significant, LFA – Lymphocyte Function associated Antigen, CAL – Clinical Attachment Level

Note: DV – Dependent Variable & IV – Independent Variable

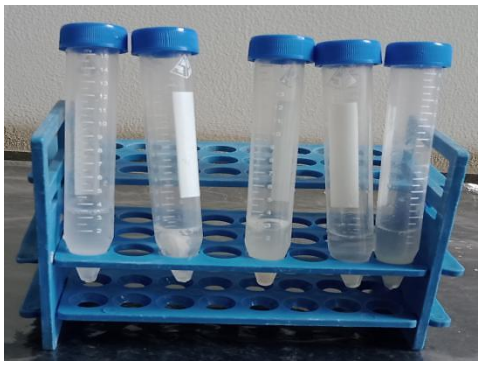

Figure 1: Collected saliva sample

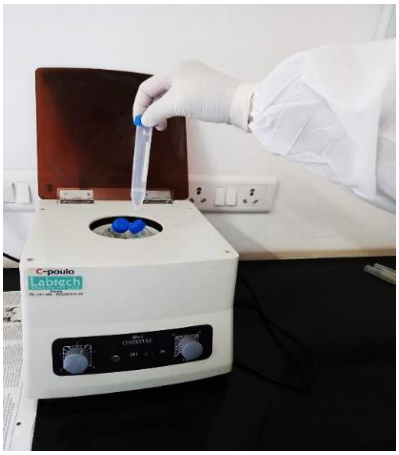

Figure 2: Centrifugation of saliva samples

Figure 3: Collection of supernatants

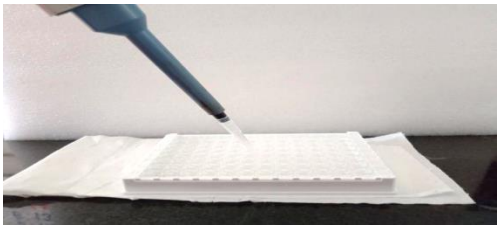

Figure 4: Standard and samples are added to the wells coated with primary antibody.

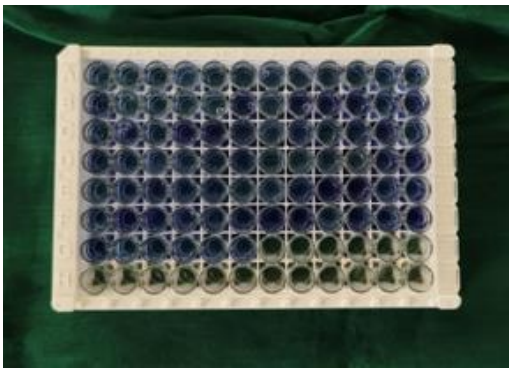

Figure 5: Addition of streptavidin – HRP

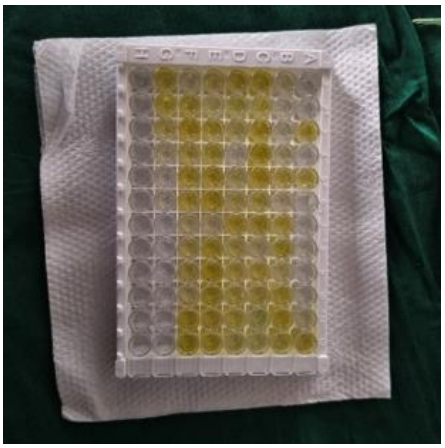

Figure 6: Addition of stop solution (blue color changed to yellow)

Figure 7: Reading of ELISA plates using microplate reader at 450nm

4. Discussion

Periodontitis is a chronic inflammatory disease caused by host-immune inflammatory processes which affect gingiva and tooth-supporting structures. The Inflammatory response to microbial colonies in the dental biofilm is comprehensively reliant on the host defence mechanism which plays a pivotal role in the periodontal disease pathogenesis.[8] Diagnosing periodontal disease helps to differentiate the condition from periodontal health and to determine the appropriate treatment plan.[9] The current clinical diagnostic modalities provide information on the destructive changes due to the disease progression, but the

early disclosure of periodontal disease can be achieved through salivary biomarkers.

The salivary biomarkers have paved the way for the development of surrogate diagnostic techniques for periodontal diseases. Salivary biomarker assay is a molecular diagnostic method that provides information regarding the pathologic changes and biologic response to treatment intervention. Quantitative analysis of salivary biomarkers helps to monitor the host response to periodontal disease and, severity of the disease and also helps in identifying the risk factors associated with periodontal diseases.[10]

Despite the presence of various biomarkers, the non-specific biomarker like Lymphocyte Function associated Antigen – 1 (LFA-1) plays a significant role in determining the periodontal health status of an individual.[11] The expression of the adhesion molecule (ICAM-1) in the pocket epithelium is directly proportional to the migration of LFA-1 in the connective tissue subjacent to the pocket epithelium into the periodontal pocket. Smoking alters the immune-inflammatory response, resulting in increase in the extent and severity of periodontal disease. It downregulates the immune response to microbial challenge. The peripheral blood neutrophils increased after acute smoking, leading to upregulation of adhesion molecules.[12] We aimed to investigate and validate LFA-1 as a potential salivary biomarker for distinguishing periodontal health from periodontitis in individuals with and without smoking habit.

Individuals' mean ages and the variations among the research groups were found to differ significantly. In comparison to all 3 groups, smoker patients with periodontitis had higher mean values of PI, GBI, PD, and CAL. This suggests that smoking-related periodontal disease is more common and severe.

Compared to the periodontally healthy group (0.1708) and the periodontitis participants without smoking habit (0.5602), we discovered that the salivary levels of LFA-1 were considerably higher in the smoking-habitual periodontitis subjects (0.6594). Our study's findings concur with Taymen et al.'s (2020) disclosure that participants with periodontitis had higher levels of LFA-1 than subjects in good periodontal health.[13]

Increased adhesion molecule activation and neutrophil migration may be the cause of the elevated salivary LFA-1 levels we saw in periodontitis patients who smoked. This observation is in accordance to the study conducted by Schaberg et al (1996) in which he analyzed the lung biopsies of 26 patients (15 smoker patients and 11 non-smoker patients), and reported that a significant upregulation of ICAM 1 in smoker patients.[14] Similarly, Overbeek et al (2011) reported increased activation of β2 integrin due to cigarette smoke in the lung of mice.[15]

Additionally, we aimed to evaluate the correlation between the degree of periodontal disease and salivary LFA-1 levels. Salivary LFA-1 levels significantly increased for every 1 mm increase in clinical attachment loss in nonsmoking periodontitis individuals, indicating accelerated disease progression. This finding was consistent with the findings of Inonu et al. (2020), who found that for every millimetre increase in the clinical attachment loss in patients with periodontitis, salivary LFA-1 levels increased.[11]

Our research is the first to report a significant increase in salivary LFA-1 levels for every 1mm increase in clinical attachment loss among smokers with periodontitis.

The strength of our study lies in presenting the role of Salivary LFA-1 as a biomarker and its versatility to predict periodontal health and periodontitis in subjects with and without smoking habits.

The limitations of this study include

1. Our study has a limited sample size and is cross-sectional.
2. The role of LFA-1 as a trustworthy biomarker may be more accurately provided by a varied group of patients with systemic disorders that affect periodontitis.
3. It would have been beneficial to include treatment interventions in order to validate the vital function of salivary LFA-1 as a prognostic indicator.
4. The smoker patients with periodontitis group comprised only male subjects due to the low prevalence of female smokers with periodontitis in our region.

5. Conclusion

The following conclusions were illustrated from our current research:

1. When comparing periodontitis participants with smoking habits to those without smoking (0.5602 ng/ml) and periodontally healthy subjects (0.1708 ng/ml), it was discovered that the salivary LFA-1 levels were considerably higher in the former group (0.6594 ng/ml).
2. Salivary LFA-1 levels significantly increased by 0.218 ng/ml for every 1 mm rise in CAL in the group with periodontitis who did not smoke; this result was statistically significant at $p<0.001$.
3. Salivary LFA-1 levels significantly increased by 0.314 ng/ml for every 1 mm rise in CAL in the smoker with periodontitis group; this result was statistically significant at $p<0.001$.

Based on the findings, salivary LFA-1 might be considered as a diagnostic biomarker in periodontitis and smoking habit is considered a potent risk factor for periodontitis. Hence, further longitudinal studies with therapeutic interventions are required to consider LFA-1 as a predictable biomarker for periodontal health.

6. Ethical Committee Approval

This study has been reviewed and approved by the Institutional Ethics Committee. IEC ref no: 291/KSRIDSR/

IEC/ 2022. Written informed consents were procured from all the participants before enrolling for the study.

7. Conflict of Interest

None.

8. Source of Funding

Self-Funding.

9. Acknowledgements

None.

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