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Original Research Article

Association of salivary pH in patients with dental caries and periodontal disease

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ABSTRACT

Background: The aims of the study was to find out the prevalence of dental caries in healthy and periodontal disease and its association with salivary pH.
Methods: A total of 80 healthy subjects reporting to the department of periodontology were selected. They were divided into four groups (clinically healthy gingival, chronic generalized gingivitis, chronic generalized periodontitis, and aggressive periodontitis) with 20 subjects in each. Periodontal parameters; decayed, missing, and filled teeth (DMFT) index and salivary pH were recorded. Unstimulated whole saliva was collected according to Navazesh method. The pH of saliva was immediately measrued using a single electrode digital pH meter.
Results: Based on statistical test it was seen that caries prevalence and DMFT was found out to be least in aggressive periodontitis group, along with least mean pH value, as compared to the other 3 groups, $p<0.05$.
Conclusions: The low prevalence of caries in aggressive periodontitis can be attributed to the low pH of saliva. Salivary pH plays a vital role in the formation of microbial film, which in turn has a significant role in the pathogenesis of both dental caries and periodontitis.

Keywords: Biofilm, Caries, Gingivitis, Periodontitis, Microflora, Salivary pH

INTRODUCTION

Caries and periodontal disease are the two foremost oral pathologies that remain widely prevalent and affect all populations throughout the lifespan. Caries refers to the localized destruction of susceptible dental hard tissues by acidic by-products from the bacterial fermentation of dietary carbohydrates. It is a chronic multifactorial disease that progresses slowly in most people.

Periodontal disease refers to any disorder of the tissues surrounding and supporting the teeth, i.e. the periodontium. Periodontal disease is considered multifactorial in nature, with a number of factors contributing to its initiation and progression. These include poor oral hygiene, specific plaque bacteria, smoking, systemic conditions (e.g. diabetes), aging and a susceptible host.[1]

Both the diseases being multifactorial in nature, the study of the relationship between dental caries and periodontal disease has been an interesting topic in research. A strong inverse relationship has been shown between aggressive periodontitis and proximal caries. Indeed, loss of periodontal support was the principal cause for tooth loss in the localized and generalized aggressive periodontitis groups, while dental caries was the principal cause for tooth extraction in the incidental attachment loss and control groups.

Little is known about the prevalence of caries and tooth loss in individuals with early-onset periodontitis, and whether the tooth loss in these subjects is mainly due to the loss of periodontal support or dental caries.[7] Information about the prevalence of caries in individuals with different forms of periodontitis would provide an understanding of how caries and different forms of periodontitis are related.

Studies have been done reporting significance of salivary pH and its relation with dental caries and periodontal disease, which conclude a more acidic pH in dental caries, however the converse being true for periodontal disease. Hence, this study aims to find out prevalence of dental caries in healthy and periodontal disease and its association with salivary pH.

METHODS

Study design

A cross-sectional study was conducted on 80 healthy subjects reporting to the department of periodontics, The Oxford Dental College, Bangalore from March 2015-October 2015. Convenience sampling method was used for data collection. The subjects were selected and divided into 4 groups including 20 subjects each, based on the inclusion and exclusion criteria, where: group A - clinically healthy gingiva; group B - chronic generalized gingivitis; group C - chronic generalized periodontitis; and group D - aggressive periodontitis.

Inclusion criteria included subjects taken were systemically healthy, within the age range between 16-40 years of age, with presence of at least 20 natural teeth (excluding third molars).

Exclusion criteria included: any periodontal or dental treatment within last 6 months, pregnancy, long-term medical therapy or medication, smoking and subjects taking tobacco in any form. The diagnosis of chronic generalized periodontitis and aggressive periodontitis was made based criteria given by AAP.[2]

The following parameters were recorded: decayed, missing, and filled teeth (DMFT) score, plaque index, gingival index, probing pocket depth (PD), clinical attachment level (CAL), and salivary pH.

PD and CAL were measured using a William's periodontal probe. Informed consent was taken from the subjects after explaining them about the study. The present study was approved by the ethical committee of the institute.

Saliva collection

Unstimulated whole saliva specimens were collected in the morning, and it was asked from all selected subjects that brush their teeth and do not use any oral stimulation such as eating and drinking for 90 min prior to collection. The subjects were also asked not to cough up mucus as saliva is collected. The subjects spit into the collection tube about once a minute for up to 10 min.[3]

The pH of the saliva was immediately measured in order to prevent any deterioration of the sample. Salivary pH was measured with the help of a single electrode digital pH meter (pHep®). The pH meter was then calibrated using freshly prepared buffers of pH 7 and pH 4. The latter was used for finer adjustment to the pH.[3] Following this, the electrode was kept dipped in double distilled water. Prior to dipping the electrode in the sample, it was gently dried completely using fresh sterile filter papers each time. After analyzing the pH, the electrode tip was again washed with a gentle stream of distilled water and then dipped in the double distilled water.

Statistical analysis

All the analysis was done using statistical package for the social sciences (SPSS) version 18. A p value of <0.05 was considered statistically significant. Comparison of mean DMFT among the groups was done using Kruskal Wallis. Comparison of mean PI, GI, pH among the four groups was done using analysis of variance (ANOVA) with post-hoc test. Comparison of mean PD and CAL between PD and CAL was done using independent sample t test.

RESULTS

Out of 80 study subjects 44 (55%) were males (Figure 1). Mean age was 31.9±4.04. No significant difference was seen in mean DMFT among the four groups (Table 1 and Figure 2).

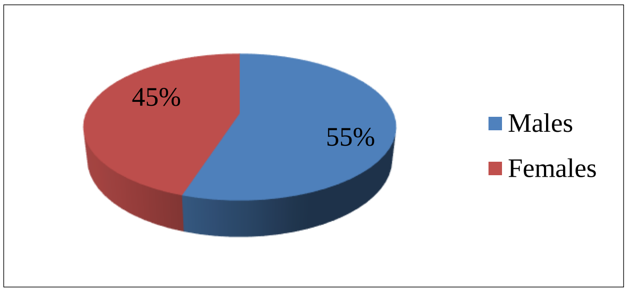

Figure 1: Gender distribution of study subjects.

However a high caries experience was seen to co relate with the group C. There was significant difference in the mean GI among the four groups. Group B, C and D had significantly higher than group A. Similarly, group B and C had higher than group D (Table 1). There was significant difference in the mean pH among the four groups. Group A, B, and C had significantly higher than Group D (Table 1 and Figure 3). A mean pH value of 7.2 in the group B, 7.1 in the group C and 6.3 in the group D was seen. Group D was significantly higher mean CAL than group C (Table 1 and Figure 4).

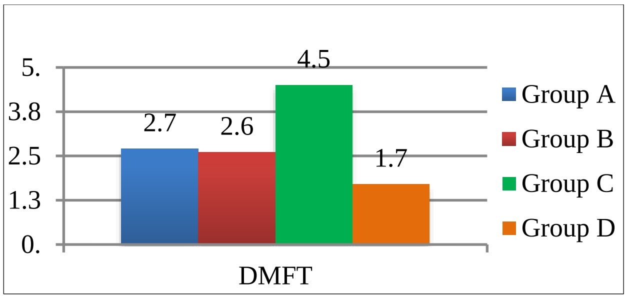

Figure 2: Mean DMFT index values of different groups.

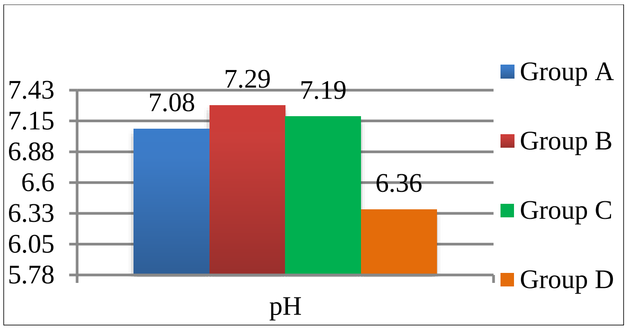

Figure 3: Mean pH values of different groups.

Table 1: Mean, standard deviation, p value and post-hoc test of the study variables.

Parameters	Group (mean±SD)				P value	Post-hoc test
	A	B	C	D		
DMFT^	2.70±2.00	2.60±1.60	4.50±4.54	1.70±1.63	0.075; NS	-
PI†	0.40±0.13	1.21±0.32	1.26±0.36	0.76±.26	<0.001; sig	B, C, D>A B, C>D
GI†	0.39±0.15	1.46±0.39	1.47±0.50	0.97±0.57	<0.001; sig	B, C, D>A B, C>D
pH†	7.08±0.12	7.29±0.39	7.19±0.31	6.36±0.45	<0.001; sig	A, B, C>D
PD‡	.	.	6.48±1.14	7.37±1.12	0.016; sig	-
CAL‡	.	.	7.95±1.57	9.13±1.62	0.025; sig	-

^Kruskal Wallis, †ANOVA with post-hoc Games Howell test, ‡independent sample t test

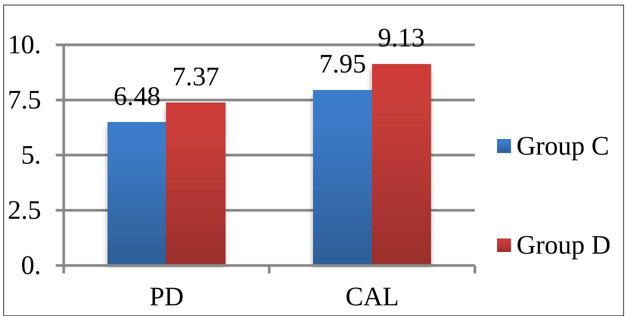

Figure 4: Periodontal pocket dept and clinical attachment loss of group C and group D.

DISCUSSION

The study of the relationship between dental caries and periodontal disease has been an interesting topic in research. The present study attempts to evaluate the relationship between dental caries and periodontal disease, while comparing their simultaneous occurrence in different types of periodontal diseases namely-chronic generalized gingivitis, chronic generalized periodontitis and aggressive periodontitis.

The present results demonstrate a mean pH value of 7.2 in the chronic gingivitis group, 7.1 in the chronic periodontitis and 6.3 in the aggressive periodontitis group, showing a relatively neutral pH in both gingivitis as well as periodontitis, however an acidic pH was seen in the aggressive periodontitis group. The normal salivary pH is from 6 to 7, and varies in accordance with the salivary flow from 5.3 to 7.8. Statistically significant correlations between gingivitis and pH did not exist, however it differed for the periodontits groups.

Findings concerning the simultaneous occurrence of periodontal disease and dental caries are contradictory, showing both positive as well as a negative association. Alabandar et al stated that caries levels were higher in patients with localized juvenile periodontitis, and included a high rate of caries among the clinical features used to identify early onset periodontitis.[7] Contrary to that, studies by Sioson et al and Al Omari et al concluded that patients with AgP had significantly less caries experience.[5,8] The association may relate to the microbiological etiology of both the diseases, along with the similar process of plaque and biofilm formation. Furthermore, both diseases share many social and behavioural background factors in common, which have been related to their etiology.

However, the negative association may be related to the demineralization process, occurring at an acidic pH of 5.5 and below, seen in development of caries as opposed to the mineralization process seen in calculus formation, occurring at an alkaline pH of 6.5 and above.[8]

Thus, changes in the microbial and environmental dynamics in microbial ecosystem may increase the potential for pathogenicity within the microbial ecosystem and subsequently initiate and promote oral diseases.

The present study showed increased caries prevalence in chronic periodontitis patients. The reasons for the same can be attributed to the findings discussed below. *Streptococcus gordini* and *Streptococcus sanguins* (being one of the key stone pathogens for dental caries) generate H_2O_2 in the biofilm during initial colonization.[11] Also, *Streptococcus gordini* is able to recruit *Porphyomonas gingivalis*; the key stone pathogen for chronic periodontitis, into the biofilm via cell signaling through AI-2.[12]

Other possible causes could be due to the friendly relation between *Streptococcus mutans* and *Veilonella spp*, where *Veilonella* uses the lactic acid produced by *Streptococcus* for its growth within the biofilm[13]. Also, it has been shown that large numbers of *S. cristatus* are able to adhere to F. nucleatum, resulting in formations known as corncobs. This co-aggregation is mediated by an arginine-sensitive interaction and actually enables *S. cristatus* to passively invade human epithelial cells.[14]

The host response also has a role to play in the above interaction. It was demonstrated that after invasion into several oral epithelial cell lines, *F. nucleatum* elicits an immediate host response with increased interleukin-8 (IL-8) expression. Association with *S. cristatus* attenuates the induction[15]. Also, *F. nucleatum* is able to increase the production of the cytokines IL-6 and IL-8. Based on the same, it can be inferred that increased cytokine levels can lead to a greater periodontal destruction.

The low caries prevalence seen in the AgP group inspite of having an acidic pH could be attributed to the inter-species interaction in the microbial biofilm. This finding can be attributed to the fact that *Aggregatibacter actinomycetemcomitans* being the key pathogen in aggressive periodontitis, interacts with *Streptococcus spp* to produce H_2O_2. This co-culture stimulates the expression of the complement resistance protein ApiA in *A. actinomycetemcomitans*, significantly increasing its resistance toward host-innate immunity.

It was also observed in the present study that chronic generalized periodontitis was mostly seen to affect males older than 30 years of age; and had significantly increased DMFT scores when compared to healthy and chronic generalized gingivitis; whereas aggressive periodontitis was seen to affect males lying within an age range of 20 to 35 years; however females showed a lower pH value (5.7) when compared to the males (6.4) in the same group.

Compelling reasons exist to use saliva as a diagnostic fluid. It meets the demands for being inexpensive, noninvasive and easy to use diagnostic methods. For patients, the noninvasive collection techniques dramatically reduce anxiety and discomfort and simplify procurement of repeated samples for monitoring over time. Saliva also is easier to handle for diagnostic procedures because it does not clot, thus lessening the manipulations required.

One of the limitations of the study could be Neyman bias. Also the sample size of the study was small; this could lead to voluntary response bias in the study.

CONCLUSION

The low prevalence of caries in aggressive periodontitis can be attributed to the low pH of saliva which could be due to the inter-species interaction in the microbial biofilm. Salivary pH plays a vital role in the formation of microbial biofilm, which in turn has a significant role in pathogenesis of these two most prevalent diseases of the oral cavity. Future studies are warranted to further explore into the dynamics of oral biofilm formation taking salivary pH as a prime component and its effect on microbial interactions.

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Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Albandar JM. Epidemiology and risk factors of periodontal diseases. Dent Clin North Am. 2005;49(3):517-32.
2. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol. 1999;4:1-6.
3. Navazesh M. Methods for collecting saliva. Ann N Y Acad Sci. 1993;694:72-7.
4. Baliga S, Muglikar S, Kale R. Salivary pH: A diagnostic biomarker. J Indian Soc Periodontol. 2013;17(4):461-5.
5. Al Omari MA, Al Habashneh R, Taanni DQ. Dental caries experience in patients with aggressive periodontitis compared to those with chronic periodontitis. Quintessence Int. 2008;39(10):847-51.
6. Takahashi N, Schachtele CF. Effect of pH on the growth and proteolytic activity of Porphyromonas gingivalis and Bacteroides intermedius. J Dent Res. 1990;69:1266-9.
7. Albandar JM, Brown LJ, Loe H. Dental caries and tooth loss in adolescents with early-onset periodontitis. J Periodontol. 1996;67:960-7.
8. Sioson PB, Furgang D, Steinberg LM, Fine DH. Proximal caries in juvenile periodontitis patients. J Periodontol. 2000;71:710-6.
9. Kreth J, Merritt J, Shi W, Qi F. Co-ordinated bacteriocin production and competence development: a possible mechanism for taking up DNA from neighbouring species. Mol Microbiol. 2005;57:392-404.
10. Kuboniwa M, Tribble GD, James CE, Kilic AO, Tao L, Herzberg MC, et al. Streptococcus gordonii utilizes several distinct gene functions to recruit Porphyromonas gingivalis into a mixed community. Mol Microbiol. 2006;60:121-39.

11. Distler W, Kroncke A. The lactate metabolism of the oral bacterium Veillonella from human saliva. Arch Oral Biol. 1981;26:657-61.
12. Edwards AM, Grossman TJ, Rudney JD. Fusobacterium nucleatum transports noninvasive Streptococcus cristatus into human epithelial cells. Infect Immun. 2006;74:654-62.
13. Zhang G, Chen R, Rudney JD. Streptococcus cristatus attenuates Fusobacterium nucleatum-induced interleukin-8 expression in oral epithelial cells. J Periodontal Res. 2008;43:408-16.
14. Hasegawa Y, Mans JJ, Mao S, Lopez MC, Baker HV, Handfield M. Gingival epithelial cell transcriptional responses to commensal and opportunistic oral microbial species. Infect Immun. 2007;75:2540-7.
15. Ramsey MM, Whiteley M. Polymicrobial interactions stimulate resistance to host innate immunity through metabolite perception. Proc Natl Acad Sci USA. 2009;106:1578-83.

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