Original Article

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Association between Periodontal Disease and Dental Caries: An Observational Study

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ABSTRACT

Introduction: Periodontal disease and dental caries are the two most common oral diseases in human. Numerous studies have been carried out in the past to determine the association between occurrence of dental caries and periodontal disease. Evidence on the simultaneous occurrence of these diseases is conflicting. The aim of the study was to evaluate the simultaneous occurrence of periodontal disease and dental caries in Bangalore population.

Methodology: An observational, cross sectional study was conducted among 1001 subjects aged 30 to 50 years for one year. Clinical attachment loss (CAL), DMFT (Decayed, Missing, and Filled Teeth) and Silness-Loe index were recorded.

Results: There was no statistically significant difference among males and females with respect to CAL, plaque index, and DMFT score. There was a positive correlation between plaque index and CAL, r value = 0.575 and a weak negative correlation between DMFT and CAL, r value = -0.063. A weak negative correlation between DMFT and plaque index, r value = -0.120 was observed in the study.

Conclusion: These data indicate that periodontal disease and dental caries do not tend to accumulate in the same subjects. There is a negative association between dental caries and periodontitis.

INTRODUCTION

Two most common oral diseases in humans are periodontal diseases and dental caries, involving teeth and their supporting tissues, Evidence on the simultaneous occurrence of these diseases is conflicting.[1] Periodontitis is an infectious and inflammatory disease of the supporting structures of teeth leading to progressive attachment and bone loss. It is characterized by pocket formation and⁄or gingival reces-sion. There is a complex interaction between plaque microorganisms and host immune system.[2] Immune and inflammatory responses are critical to the pathogenesis of periodontitis and are shaped by a number of host-related factors, both intrinsic (e.g. genetics) and induced (e.g. pollutants).[3]

Dental caries occur because of demineralization of enamel and dentine by organic acids formed by bacteria in plaque through anaerobic metabolism of sugar in diet.[4] Dental caries is a multifactorial disease. The contribution of life style, environmental factors, and hereditary factors has influence on their development. These include the frequent intake of fermentable carbohydrate, poor oral hygiene, high counts of cariogenic microorganisms, inadequate use of fluoride, and impaired salivary functions.[5]

The relationship between periodontitis and dental caries has been the focus of many research groups, as both the destructive diseases can be seen at the time of an oral inspection. There are evidences of positive association[1,6,7], negative association[8], or no association[9] between periodontitis and dental caries. Hence, it is important to study the correlation between these two diseases for a more comprehensive treatment plan and to know till what extent these two common diseases accumulate in same individual. Thus, the aim of the study was to evaluate the simultaneous occurrence of periodontal disease and dental caries in Bangalore population aged 30 to 50 years.

METHODS

The present study was an observational, cross sectional study. The study was conducted in the Department of Periodontics, tertiary care Dental Hospital of Bangalore, Karnataka, after obtaining ethical clearance from the institutional board and ethical committee. The duration of the study was one year. Patients who agreed to participate in the study were selected. The sample size was derived using single proportion formula taking maximum 50% as the disease prevalence, keeping 5% confidence limit, for p=0.05. The inclusion criteria were patients suffering with chronic periodontitis, age between 30 to 50 years[10], and presence of 20 teeth. The exclusion criteria were history of underlying systemic problems, history of tobacco chewing and current or former smoker, pregnant and lactating female, patients on steroid therapy, oral contraceptives, history of periodontal treatment in past six months, and under antibiotic/non-steroidal anti-inflammatory drugs treatment for past six months.

Clinical oral examination was conducted using a dental mirror, University of North Carolina -15(UNC-15). The clinical oral examination included DMFT (Decayed, Missing, Filled teeth), Silness Loe Plaque Index, and Clinical attachment loss (CAL). The teeth were blown dry and the status of each tooth surface was diagnosed.

In oral epidemiology, assessment of dental caries is done by DMFT index.[11] In DMFT, D denotes the number of teeth with decay, M denotes the number of missing teeth that have been extracted due to caries, F denotes the number of teeth being filled due to caries, and T denotes the number of teeth. By adding the four components, DMFT score was given.[12] A tooth was recorded as decayed if there was evidence of caries lesion clearly extending to dentine. The caries lesion was to be cavitated, to have penetrated the fissure and undermined the enamel, or the dentine walls were to have clearly softened.

Loss of attachment is measured as clinical attachment loss (CAL) measured as distance from the cemento-enamel junction (CEJ) to the bottom of the clinical pocket by the UNC-15 probe. Severity of chronic periodontitis was categorized on the basis of amount of clinical attachment loss. Case definitions and severity of periodontitis to be used in epidemiological studies have been proposed: slight=1-2mm, moderate = 3-4 mm, and severe ≥5 mm clinical attachment loss.[13, 14]

Silness Loe Plaque Index was measured on entire dentition. Only plaque of cervical third tooth was evaluated. Plaque extended to the middle or incisal third was not considered. The surfaces examined are distal-facial, mesial-facial, facial, and lingual surfaces. The tooth was dried and examined with explorer. Score 0/1/2/3 was assigned based on scoring criteria.[15]

Score 0: No plaque.

Score 1: A film of plaque adhering to the free gingival margin and adjacent area of the tooth.

Score 2: Moderate accumulation of soft deposit within gingival pocket, tooth, and gingival margin.

Score 3: Abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin.

Data obtained through clinical recording forms (CRF) was compiled onto MS Office excel sheet (v.2010, Microsoft Inc, USA). Statistical analysis of data was performed using statistical package for social sciences (SPSS, v.22.0, IBM). Descriptive data like percentage and frequencies of males and females participating in the present study, mean, and standard deviation of numerical data was expressed. Comparison of means of variables

Table 1: Genderwise comparison of descriptive data of clinical oral examination

	Gender	N	Mean	Std. Deviation	p value
CAL	Male	476	5.04638	1.2623590	0.481 NS
	Female	525	4.98859	1.3241400	
PI	Male	476	0.804712	0.5221613	0.183 NS
	Female	525	0.850827	0.5677789	
DMFT	Male	476	1.26	1.150	0.588 NS
	Female	525	1.22	1.154	

NS: Non significant; CAL: Clinical Attachment Loss ; PI: Plaque Index; DMFT: Decayed Missing Filled Teeth

like CAL, PI, and DMFT among males and females was done using independent t test. Pearson Correlation coefficient (r value) regression curve was used to evaluate the correlation between plaque index/CAL, DMFT/CAL, and DMFT/plaque index.[16]

RESULTS

There were 47.6% (476 out of 1001) males and 52.4% (525 out of 1001) females. No statistically significant difference among males and females with respect to clinical attachment loss, plaque index, and DMFT score (Table 1) was observed in the study. There was a positive correlation between Plaque index and CAL, r value = 0.575 ($r = 0.575$, $p<0.01$). The scatter plot (Figure1) suggested a definite linear relationship between plaque index and CAL, with larger values of plaque tending to be associated with larger values of CAL.

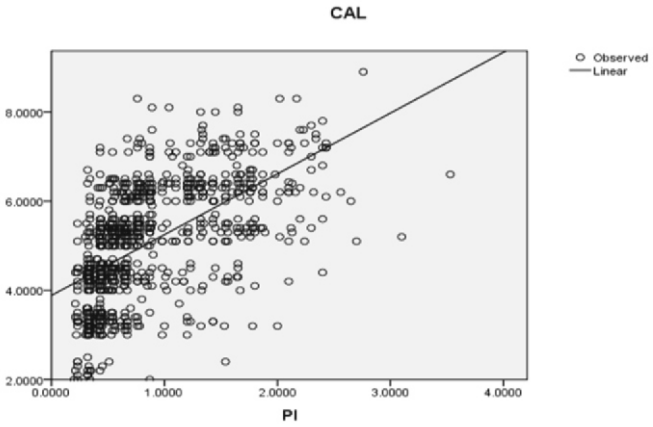

Figure 1: Scatter plot showing correlation between Clinical Attachment Loss and Plaque Index.

There was a negative correlation between DMFT and plaque index ($r = -0.120$, $p < 0.01$). The correlation between DMFT and CAL was found weak having negative pearson correlation coefficient $r = -0.63$ ($p < 0.05$) as depicted in figure 2.

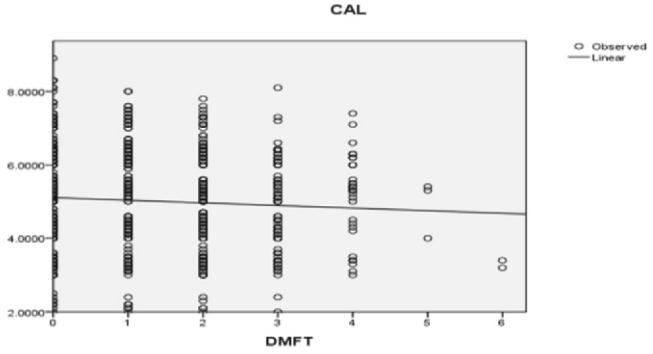

Figure 2: Scatter plot showing correlation between Clinical Attachment Loss and Decayed Missing Filled Teeth.

DISCUSSION

Periodontitis is an inflammatory disease of the periodontium associated with a biofilm. There are two primary causes, a primary microbial shift in the oral micro flora and a primary immunologically mediated destructive host inflammatory response contributing to periodontitis.[17] Loesche proposed both a nonspecific and a specific plaque hypothesis, suggesting that plaque causes periodontal disease initiation and progression.[18] In this study, there was a strong positive correlation between plaque index and clinical attachment loss. The results of this study were in accordance with the previous study.[19]

The results showed a weak negative correlation between DMFT index and CAL. Weak negative association means that average value of DMFT score will change slightly in inverse direction in response to change in CAL, denoting that there is no simultaneous occurrence between dental caries and periodontitis. These results are similar with another study by Sewon et al[8] which reported that concomitant appearance of periodontitis and caries in the same subject is not a generally occurring phenomenon. Although an inverse correlation between proximal caries and juvenile periodontal disease has been suggested by Fine et al.[20] They also suggested an association with some undefined microbiologic or immunologic event. In fact, Lehner[21] found an inverse relation between the immune responses of periodontal disease and caries. The consensus seems to be that caries result from fermentation and periodontal destruction from proteolytic and putrefaction process and these two reactions donot occur simultaneously in the immediate environment of tooth.[22] There is difference in the bacteriological spectrum that may be related to demineralization process seen in the development of caries as opposed to mineralization process seen in calculus formation associated with etiology of periodontal diseases.[1] The results of the study are conflicting with a study conducted by Hyman and Red[23] where a positive association was found between mean loss of attachment and the mean number of decayed surface. The association between periodontitis and dental caries was found positive in another study by Mattila et al[1] stating much possible common background between the diseases.

The result of the present study showed a weak negative correlation between DMFT and plaque index. The amount of dental decay is measured using the DMFT index, a

count of the number of teeth in a person's mouth that are decayed, missing or filled as a result of caries.[24]

Bacteria in dental plaque, through anaerobic metabolism of sugar in diet, produce organic acid which results in demineralization of enamel and dentin leading to dental caries. The development of caries depends not only on presence of plaque but on many other factors like presence of sugar, susceptibility of tooth, quality and quantity of sugar, plaque bacteria, and time for which fermentable dietary carbohydrate are available for bacterial fermentation, frequency of sugar consumption and amount consumed. [25] This could be the reason that the present study showed negative correlation between DMFT and plaque index.

Dental caries and periodontitis are two contrasting diseases which are diagnosable and treatable bacterial infections. While caries mostly involve the supra-gingival surfaces of teeth, periodontitis on the other hand involves in approximation to the sub-gingival plaque near the gingival tissues. The bacteria involved and the patho-physiology of the infections are distinctly different.[26]

The limitation of the study is that it lacks longitudinal follow up of the subjects and biochemical analysis of saliva.

CONCLUSION

The present cross-sectional data support the hypothesis that periodontal disease and dental decay are negatively associated and do not tend to accumulate in the same subject. Hence the treatment plan for these two diseases should be different, after a through clinical examination.

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