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Asthma and Periodontal Disease

[1]Dr. Shrinidhi M S, Prof & Head, Department of Periodontics & Implantology

[2]Dr. Soumya B G, Professor, Department of Periodontics &Implantology

[3]Dr. Gufran Danish Khan, Senior Lecturer, Department of Periodontics &Implantology

[4]Dr. Shivakumar. T P, Reader, Department of Periodontics &Implantology

[5]Dr. Chethana K C, Reader, Department of Periodontics &Implantology

[6]Dr. Usha Chikkaiah, Prof & Head, Department of Oral Medicine & Radiology

[1,2,3,5,6] Sharavathi Dental College and Hospital, Shimoga, Karnataka

[4]Subbaiah Dental College and Hospital, Shimoga, Karnataka

Corresponding Author: Dr. Usha Chikkaiah, Prof & Head, Department of Oral Medicine & Radiology, Sharavathi Dental College & Hospital, TH Road, Alkola, Shimoga 577205.

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Abstract

Aim: Respiratory diseases are responsible for significant morbidity and mortality in human populations. There is evidence that oral diseases and respiratory disorders seem to have an interrelation. Hence, the aim of the present study was to evaluate whether there is any association between the degree of bronchial asthma and severity of periodontal disease.

Material and method: Out of all the asthmatic patients who reported to the General Hospital over a period of one month, 90 were enrolled into the study. 30 non-asthmatic patients with periodontitis were assigned to a control group. Based on GINA criteria and spirometry, asthmatic patients were divided into 3 groups of 30 patients each comprising of mild, moderate and severe bronchial asthma. 30 periodontitis patients from the Out Patient Department of the Dental College were assigned to the control group. The clinical parameters such as OHI(s), Russel's Periodontal Index, Gingival Bleeding Index and Probing pocket depth were recorded in all the groups. Data was analyzed with post-hoc Tukey's test, one way ANOVA using SPSS (software version 17)

Result: The mean OHIS, PD, RPI & GBI were comparatively higher and statistically significant in severe asthmatics, which indicated greater periodontal destruction as well as reduced oral hygiene measures among severe asthmatic group as well as compared to other the other two groups.

Conclusion: The results of the present study demonstrated that bronchial asthma has a positive correlation with the severity of periodontitis.

Keywords: Asthma, periodontitis, spirometry, cytokines, bronchodilators.

Introduction

Respiratory diseases are responsible for significant morbidity and mortality in human populations. Previous studies have quoted potential respiratory pathogens to colonize the dental plaque and get aspirated into the lungs to provoke an infection.[1] These diseases can also be risk indicators and factors for periodontal disease, as few

studies have shown that P.aeruginosa may adhere better to oral epithelial cells that have already been colonized by respiratory pathogens due to a mucosal alteration with regard to fibronectin.[2,3] Patients with meticulous oral hygiene have a lower quantum of enzymes that degrade fibronectin. Hence, destruction of fibronectin leads to better adhesion of respiratory pathogens to these oral epithelial cells, due to the exposure of buried receptors or adhesins on the epithelial cells.

Asthma is a heterogenous disease usually characterized by chronic airway inflammation associated with wheezing, coughing, chest tightness and short episodes of breathlessness with variable expiratory airflow limitation.[4] About 300 million people worldwide are estimated to be at the moment to be suffering from asthma and by 2057 an additional 100 million more will be diagnosed.[5] In India, the prevalence of asthma is 3% of the total population.[6] Pathogenesis of asthma and periodontitis seem to closely resemble each other. The Inflammatory response in asthma is due to release of cytokines like IL-5 (for eosinophil differentiation) IL-4 (Th2 cell differentiation) and IL-13 (IgE formation). However, key cytokines are IL-1β and tumor necrosis factor-α (TNF-α), which amplify the inflammatory response.[7] Further, there is a strong presence of IgE and eosinophils in asthmatic patients.[8]

Considerable evidence show that asthmatic patients exhibit higher plaque scores than non-asthmatics.[9,10,11] Anti-asthmatic medication also has shown evidence of adversely affecting dental health and periodontal health.[12] Hence, this study was carried out to explore the connection between bronchial asthma, anti-asthmatic drugs, and periodontal disease.

Materials and methods

Patients suffering from bronchial asthma and on medication for the past 12 months, in the age range of 30 to 60 years visiting the OPD of General hospital over a period of 30 days, were enrolled for the study. 30 non-asthmatic periodontitis patients were set as controls from the OPD of the dental college. Diagnosed on the basis of Global Initiative[4] and National heart lung and blood institute guidelines,[7] and further based on spirometry results, all the patients were screened for bronchial asthma and enrolled in each of the three groups : mild, moderate and severe asthmatic patients, such that each group comprised of 30 participants each.

Mild asthmatics-Dyspnea only with activity and peak expiratory force (PEF) value of >80%. Moderate asthmatics-Dyspnea interferes with or limits usual activity and PEF value of 60-80%.Severe asthmatics-Dyspnea at rest interferes with conversation and PEF value of < 60%.

Patients who had atleast 20 teeth and who were not diagnosed for the first time to suffer from asthma, and who were taking beta-2 agonist (salbutamol) and inhalational corticosteroids were included in the study. Smokers and patients with other systemic diseases and conditions which could have an effect on the study outcome were excluded.

Clinical parameters such as: Oral hygiene index simplified [OHI(s)] by,[13] Periodontal Index by Russell A L (RPI),[14] Gingival bleeding index (GBI) by Ainamo and Bay,[15] and Probing pocket depth (PPD) using UNC-15 were recorded. The periodontal status was evaluated by two trained examiners who were blinded to the study design.

SPSS (Version 17) was used for statistical analysis. Results are presented as Mean±SD and range values. One Way ANOVA was used for multiple group comparisons followed by post-hoc Tukeys test for groupwise comparisons. A p-value of 0.05 or less was considered for statistical significance.

Results

The mean ± SD age of participants was 45±9.8 years. There was no statistical difference in clinical parameters between age or socio-economic status. The mean OHI(s), PPD, RPI & GBI were comparatively higher in severe asthmatics than mild and moderate asthmatics.

Mean±SD scores for OHI(s) in control, mild moderate and severe asthmatic groups were 0.63±0.16, 1.22±0.46, 1.88±0.61 and 2.10±0.69 respectively(Table.1,Graph.1). The Mean values tended to go up as the severity of asthma increased with groups. However, PPD mean values showed a dip in the moderate asthmatics group only to spike back up again with the severe asthmatic group with values being 4.17±0.39,5.57±0.39,4.98±0.42 and 5.64±0.39 respectively starting from control group(Table.2,Graph.2). Mean GBI increased from control to severe asthmatic group at 27.4±8.3,56.3±12.3,63.4±11.8 and 70.8±12.7 respectively(Table.3,Graph.3). Mean RPI scores however showed a higher value for the moderate group than the severe at 159±0.44 and 1.46± 0.35 respectively, and the control and mild groups exhibited 1.04±0.11 and 1.81±0.53(Table.4,Graph.4).

Intergroup comparison: The control group scores for OHI(s) differed highly significantly from the test groups (p=0.00). However, the difference between moderate and severely asthmatic groups was not statistically different (p=0.39) But, the scores between mild to moderate as well as mild to severe group were statistically highly significant. (p=0.00) (Table. 5)

The difference in PPD values were also statistically highly significant between the control and test groups as well as mild to moderate and mild to severe group (p=0.0 Moderate and severe asthmatics didn't differ significantly in PPD scores. (p=0.92) (Table.6)

While control GBI scores to test group scores difference was highly significant.(p=0.00). and mild to severe asthmatic groups differed statistically highly significantly (p=0.00), mild to moderate and moderate to severe group however, didn't differ significantly (p=0.08,0.06) (Table.7)

Difference in periodontal index scores between control and the test groups were statistically highly significant.(p=0.00) But, between mild to moderate, and moderate to severe groups the difference was not significant. (p=0.14, 0.59) RPI scores were significantly different between mild and severe asthmatic groups. (Table.8)

One way ANOVA test was performed to compare the variation in the group means of all the parameters. The F-values for all groups were high indicating that the OHI(s),PPD,GBI and RPI data were scattered far apart from each other and this difference between the group means was highly statistically significant for each parameter (p=0.00) (Tables.1,2,3,4)

Discussion

This study was conducted to evaluate the effect of asthma on the periodontal status of the asthmatic subjects. The results demonstrated that the parameters for periodontitis were always on the higher end of severity for asthmatic patients. Mean OHI(s) values were the highest for severely asthmatic group and the difference was statistically highly significant in conformation with another study that evaluated the plaque index in asthmatics and non-asthmatics.[16]

Probing pocket depths got progressively deeper as asthma severity increased between groups as compared to controls (non-asthmatic patients with periodontitis) similar to a case control study conducted in Jordan.[17]

Gingival bleeding scores by far showed the highest F-value indicating the statistically highly significant

difference in inflammation in control patients and the severely asthmatics, this was in conformation with a case control study,[11]however the said study did not exhibit the drastic difference that our study exhibited. Increase in gingival bleeding scores are the perfect illustration to the underlying increase in inflammatory cytokines like TNF-α and IL-6 in asthmatic patients[7] rendering them easily susceptible for gingival inflammation and as a result higher bleeding scores.

RPI scores rose from control to mildly asthmatic groups but showed a downward trend through moderate till severe asthmatics, but still remained higher than control despite this freak tendency, difference was statistically significant which is yet again similar to the above mentioned case control study.

The difference in RPI scores and GBI scores could probably be attributed to the fact that chronic periodontitis takes longer to develop[18] and that fact is reflected in the clinical parameters of the same while symptoms of gingival inflammation take much less time to appear, in addition gingival bleeding is one of the very first signs to appear. This could contribute to the RPI scores, despite being significantly higher in severely asthmatics, are not as drastically different when compared to bleeding scores of controls and severely asthmatics.

There is substantial evidence to suggest that asthma/respiratory problems and periodontal disease are probably bi-directional, in the sense that poor oral health could alter the respiratory tract environment and asthmatics probably are predisposed to developing periodontal disease. Poor oral health results in the formation of extensive plaque which may promote colonization of respiratory pathogens in the oral cavity.[12] Several studies have reported that poor oral health alters the quality of respiratory epithelium by secreting hydrolytic enzymes or cytokines which leads to increased susceptibility of respiratory pathogens to adhere and colonize. [19,20,21]

Lee et al [22] have reported a positive correlation between asthma and periodontitis, with the likelihood of asthmatics prone for periodontal disease being 5 times more. In addition, Soledade et al[23] reported patients with periodontitis to have a three-fold increased risk of developing severe asthma. The association is due to colonization of oropharyngeal mucosal surfaces by respiratory pathogens, followed by shedding of these respiratory pathogens leading to contamination of lower respiratory tract.[24,25]

Also, anaerobic bacteria release biologically active products such as lipopolysaccharides and enzymes into the saliva. Aspiration of these bacteria modifies the airway mucosa to stimulate inflammatory cytokine release from epithelial cells.

However, our study is an attempt to investigate the premise of the severity of asthma affecting the levels of periodontal disease parameters. We found that there was a significant increase in the clinical parameters recorded with the rise in severity of the bronchial asthma similar to the findings of Moraschini et al[12] and Bhardhwaj et al[11] The significance noted was more when compared between mild and severe asthmatics rather than between moderate and severe in consort with a similar study.[26] However, a study by Scannapieco did not find any interrelationship between acute respiratory conditions and periodontitis.[27]

Few mechanisms have been reported by which asthma can be an etiological factor for gingivitis and periodontitis.[28] One such pathway is by pathological activation of immune and inflammatory response.[9] Activation of these immune and inflammatory response leads to increase in the release of pro-inflammatory cytokines like IL-1β and TNF-α, which have destructive effects on collagen and bone. +

The second mechanism is because of the adverse effect of drugs such as bronchodilators (β2-agonists) and inhalational corticosteroids used in the treatment of asthma. Long-term use of bronchodilators leads to decrease in salivary flow, which causes dehydration of alveolar mucosa, decrease in IgA, alteration of immune response and increase in calculus formation due to increased level of calcium and phosphorus in sub-maxillary and parotid saliva.[9,10,29, 30]

Calculus is an established etiological factor in initiating gingivitis and sustaining periodontitis.[31] The results of the present study reflect this in the increased OHI(s) scores.

Inhaled corticosteroids are weak organic acids which are not generally metabolized by oral bacteria, and can further pose a threat in pH change if the inhalant used is sugar based.[32] Also, 10-20% of the inhaled drug reaches the lungs, the rest of the drug, which is a considerably significant amount remains in the oropharynx.[33,34]

Systemic circulation of this unutilized corticosteroids leads to reduction in bone mineral density which may act as a predisposing factor for periodontitis.[35]

In health, bone formation and bone resorption are in a delicate balance, bone structure and function deviate from normal once this balance is disrupted.[36] Glucocorticoids play a significant role in this direction by affecting the hypothalamus-pituitary-adrenal axis. The net effect of such an event would be a reduction in osteoblastogenesis, increased osteoblast apoptosis and reduction in bone formation capacity.[37] Osteogenic potential of the bone marrow mesenchymal stem cells is transformed to adipogenic differentiation which causes reduced osteoblastogenesis.[38] Increased production of reactive oxygen species induced by steroids leads to osteoblast apoptosis.This apoptosis is one of the consequences of steroid induced reactive oxygen species production.[39] Animal model studies[40] have demonstrated that activation of glucocorticoid receptors in osteoblasts results in reduced bone mass, trabecular thickness, osteoblast numbers and also impaired osteoblast differentiation. This is the result of steroid-induced suppression of osteoblast-derived cytokines such as interleukin-11.[40] All the above processes lead to a net effect of compromised bone structure and function. Also, the adverse effects of drugs causes xerostomia,[41] which leads to increased plaque accumulation and gingival bleeding.[42] Further a reduced pH of gingival sulcus within as little as 30 minutes after inhalation is noticed.[43] The present study showed that bleeding on probing and plaque scores got progressively higher with the severe asthmatic groups exhibiting the highest scores among the three groups. This could also be the result of increased doses of salbutamol and steroids inhalatives considering the severity of asthma.

Inhalational corticosteroids also compromise the oral immunity and interfere with inflammatory components, as it acts as an anti-inflammatory agent, by downregulation of immune response. Circulating B-cells and T-cells numbers are reduced, and differentiation programmes of progenitor cells are also altered.[44] This creates an environment conducive for the periodontal pathogens to overwhelm host defenses and overgrow the beneficial bacteria and result in periodontal disease.[45] Similar results were obtained by Soledade et al,[23] hence it appears that medications for bronchial asthma, rather than asthma by itself, would play a more prominent, if not the sole etiological role to the detriment of periodontal tissues, however contradictory results have been observed[22] where asthmatics taking anti-asthmatic medications have displayed less periodontitis.

Conclusion

In the present study, considering the limitations, with regard to timeframe and resources, it can be inferred that bronchial asthma does have a positive relationship with

periodontitis, with an increase in the severity of periodontal destruction in severe asthmatics. The evidence suggesting that inhalational cortisteroids might be playing an etiological role, might warrant the need to put the diagnosed asthmatics under a periodic periodontal maintenance care. However, causality cannot be established with this study, because of its retrospective and observational nature. Hence multicenter, prospective studies with large sample size and lengthy follow-up or interventional designs are required to establish the causality to not only treat asthma successfully but treat its offshoots also adequately.

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Legends Tables:

Table.1 : Oral Hygiene Index

Groups		Mean±SD	Min	Max
1.Controls		0.63±0.16	0.3	1.1
2.Mild		1.22±0.46	0.2	1.8
3.Moderate		1.88±0.61	0.5	2.8
4.Severe		2.10±0.69	0.5	3.0
ANOVA	F	48.62	-	
	P	0.00*		

*P value <0.001 HS : highly significant

SD : standard deviation

Min : Minimum

Max : maximum

Table. 2 : Probing Pocket Depths

Groups	Mean±SD	Min	Max
1.Controls	4.17±0.39	3.0	5.0
2.Mild	5.57±0.39	5.0	6.1
3.Moderate	4.98±0.42	4.4	6.2

4.Severe		5.64±0.49	4.8	6.5
ANOVA	F	77.5		
	P	0.00*		

*P value <0.001

HS : highly significant

SD: standard deviation

Min: Minimum

Max : maximum

Table. 3: Gingival Bleeding Index

Groups		Mean±SD	Min	Max
1.Controls		27.4±8.3	9.0	45.0
2.Mild		56.3±12.3	30.0	69.0
3.Moderate		63.4±11.8	36.0	76.0
4.Severe		70.8±12.7	41.0	86.0
ANOVA	F	83.21	-	
	P	0.00*		

*P value<0.001

HS: highly significant

SD : standard deviation

Min : minimum

Max : maximum

Table.4 : Russel's Periodontal Index

Groups		Mean±SD	Min	Max
1.Controls		1.04±0.11	0.9	1.2
2.Mild		1.81±0.53	0.7	2.6
3.Moderate		1.59±0.44	0.8	2.2
4.Severe		1.46±0.35	0.8	1.9
ANOVA	F	20.79		
	P	0.00*		

*P value<0.001

HS: highly significant

SD : standard deviation

Min : minimum

Max : maximum

Table.5 : Difference between groups [OHI(s)]

Groups compared	P value
1 v/s 2	0.00*
1 v/s 3	0.00*
1 v/s 4	0.00*
2 v/s 3	0.00*
2 v/s 4	0.00*
3 v/s 4	0.39, NS

Post-hoc Tukey's test

* P value < 0.001

HS : highly significant

¶ P value > 0.05

NS: not significant

OHI(s): Oral Hygiene Index simplified

Table 6: Difference between groups [PPD]

Groups compared	P value
1 v/s 2	0.00*
1 v/s 3	0.00*
1 v/s 4	0.00*
2 v/s 3	0.00*
2 v/s 4	0.00*
3 v/s 4	0.92, NS

Post-hoc Tukey's test

*P < 0.001

HS: highly significant

¶P < 0.05

S: significant

†P > 0.05

NS: not significant

RPI : Russell’s Periodontal Index

Table.7: Difference between groups [GBI]

Table. 7

Groups compared	P value
1 v/s 2	0.00*
1 v/s 3	0.00*
1 v/s 4	0.00*
2 v/s 3	0.08¶, NS
2 v/s 4	0.00*
3 v/s 4	0.06¶, NS

Post-hoc Tukey's test

* P < 0.001

HS: highly significant

¶P > 0.05

NS: not significant

GBI: gingival bleeding index

Table.8: Difference between groups [RPI]

Table. 8

Groups compared	P value
1 v/s 2	0.00*
1 v/s 3	0.00*
1 v/s 4	0.00*
2 v/s 3	0.14, NS
2 v/s 4	0.01¶
3 v/s 4	0.59†, NS