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Effects of Phase I Periodontal Therapy on the Quality of Life in COPD Patients

Authors
Krishna Das[1,2], Swarga Jyoti Das[1], Jogesh Sarma[3]
[1]Department of Periodontics and Implantology, Regional Dental College, Guwahati-781032, Assam, India
[2]RNB Civil Hospital, Kokrajhar-783370, Assam, India
[3]Department of TB and Pulmonary Medicine, Guwahati Medical College, Guwahati-781032, Assam, India
Corresponding Author
Dr Swarga Jyoti Das
Professor & Head, Department of Periodontics and Implantology, Regional Dental College, Guwahati-781032, Assam, India
Phone no. 091-361-2463050
Fax No. 091-361-2529877, Email: *swargajyoti_das2@rediffmail.com*

Abstract
Quality of life may be severely impaired in patients with chronic obstructive pulmonary disease (COPD). Therefore, goal of its therapy is to minimize the impact of the disease on the patients' quality of life in addition to improve the physiological and functional state. Therefore, the present study was carried out to evaluate the association of periodontal health and COPD, and the effectiveness of phase I periodontal treatment on the quality of life in COPD patients using the St. George's respiratory questionnaire (SGRQ), consisting the spheres of symptoms, activities and impacts.35 subjects with COPD were selected and randomly categorizes into two groups: group A (n=17; received no periodontal therapy) and group B (n=18; received phase I periodontal therapy consisting of oral hygiene instructions, full-mouth scaling and root planing). Oral health status was assessed by evaluating simplified oral hygiene index, gingival index, probing pocket depth and clinical attachment level on day 0 and at the end of 12 months of therapy. Similarly, quality of life was assessed on those two days. A positive correlation between the severity of COPD and oral health was observed. Also, a significant improvement in all the oral health parameters and SGRQ activity score was seen in group B, in contrast of group A, where all the parameters deteriorated. Thus, the findings of the present study may suggest that promotion of oral health care and knowledge through the phase I periodontal therapy may play an important role on modification of the quality of life in COPD patients.
Keywords: *COPD, periodontitis, periodontal therapy, SGRQ.*

Introduction

Periodontal disease is an inflammatory disease caused predominantly by Gram-negative, anaerobic, and microaerophilic bacteria that colonise the subgingival area though modified by environment, physical, social and host stresses. It results in progressive destruction of the periodontal ligament and alveolar bone with increased probing depth, recession, or both (Newman *et al*., 2011)[1]. Throughout the history of

mankind, it has been believed that oral diseases and maladies including periodontal disease, may have an effect on the rest of the body. Over the centuries, writings from the ancient Egyptians, Hebrews, Assyrians, Greeks and Romans, have all noted the importance of the mouth in overall health and well-being. Thus, the concept of linking periodontitis and systemic diseases could be traced back to the beginning of recorded history and medicine (Lindhe *et al*., 2003)[2].

Periodontal disease has been linked to an increased risk of various systemic diseases, including the respiratory diseases *e.g.*pneumonia and chronic obstructive pulmonary disease (COPD) (Fowler *et al.*, 2001)[3]. COPD is a generic term defined by the presence of airflow obstruction with excess production of sputum resulting from chronic bronchitis and/or emphysema. It has been recognized as one of the major causes of death and disability globally and is the fourth leading cause of death in United States (Viegi *et al.,* 2001)[4].The aetiology of COPD is complex and multifactorial, involving multiple genetic and environmental factors (Rabe *et al*., 2007)[5]. An enhanced or abnormal inflammatory response to inhaled particles or gases, beyond the normal protective inflammatory response in the lungs, is a characteristic feature of COPD and is potential to produce lung injury (Saetta, 1999)[6].

In individuals with periodontitis, bacteria present in the gingival sulcus or the subsequently formed periodontal pockets, may have easy access to the blood vessels (Thoden and Abraham, 1984)[7]. Again aspiration of oropharyngeal secretions involving the pathogens present in dental plaque is well-documented cause of COPD. Therefore, it is plausible that oral microorganisms might infect the respiratory tract, causing COPD (Scannapieco, 1999; Scannapieco and Ho, 2001)[8, 9].

Since both the COPD and periodontal disease are inflammatory in nature and the tissue damages occur in both are the result of major inflammatory components (e.g. prostaglandins and cytokines) activated by the host response. Therefore, it is possible that both the disease may have a linking mechanism. Parallel mechanisms for the development of both conditions are given by Travis *et al.*,in 1994[10] (Figure 1).

Again, nearly half of COPD patients are accounted a limitation in daily activities and reduced quality of life. They are in trouble to perform their regular activities and subsequently, leading to depression and hopelessness. Therefore, the goal of COPD therapy should not only be to improve the physiological and functional state, but also to minimize the impact of the disease on the patients' quality of life. Wang *et al*., in 2009[11] noted a positive and important role of promotion of oral health knowledge and dental care on the prevention and treatment of COPD, but its effect on the quality of life in COPD patients have not yet been studied. Keeping this in mind, the present study was proposed to evaluate the association of periodontal health and COPD, and also the effectiveness of phase I periodontal treatment on the quality of life in COPD patients.

Materials and Methods

A total number of thirty five (n=35, 28 male and 7 female) subjects, diagnosed as COPD, were considered for this study. Based on FEV1 values, the patients were classified using global Initiative for chronic obstructive lung disease (GOLD) classification into mild, moderate and severe. The subjects were randomly categorized into two groups: A (n=17) and B (n=18). Group A did not receive any periodontal therapy (control), while group B received phase I periodontal therapy consisting of full-mouth scaling and root planing using hand instruments, and oral hygiene instructions (case).

Inclusion Criteria

- COPD patients diagnosed by the physician
- Subjects above 40 years
- Subjects with not less than 20 teeth

Exclusion Criteria

- Subjects with a history of periodontal treatment in the last 1 year

- Very severe COPD patients with acute exacerbation
- Subjects with a history of smoking and tobacco chewing in the last 1year
- Patients with a history of systemic diseases especially diabetes mellitus, cardiovascular diseases and malignancy
- Immunocompromised patients

Clinical Parameters

The following clinical parameters were recorded using UNC-15 periodontal probe:

- Simplified Oral Hygiene Index (OHI-S) (Debris and Calculus Index)
- Gingival Index (GI)
- Probing Pocket Depth (PPD)
- Clinical Attachment Level (CAL)

Oral Hygiene Index (OHI) (Greene and Vermillion, 1964)[12]

This index is used to classify and assess the oral hygiene status. It comprises of two components, the Debris Index (DI) and the Calculus Index (CI). Six tooth surfaces are examined for DI-Sand CI-S. They are: 16 (buccal), 11 (labial), 26 (buccal), 36 (lingual), 31(labial) and 46 (lingual). The mouth was examined first for DI-S.

Debris index (DI)

The surface area covered by debris is estimated by running the side of a no. 23 explorer along the tooth surfaces to be examined and noting the occlusal or incisal extent of the debris as it is removed from the tooth surface. The scoring criteria is as follows:

- Score 0: No debris or stain present
- Score 1: Soft debris covering not more than one third of the tooth surface, or presence of extrinsic stains without other debris regardless of surface area covered.
- Score 2: Soft debris covering more than one third but not more than two thirds of the exposed tooth surface
- Score 3: Soft debris covering more than two thirds of the exposed tooth surface

The debris score was totalled and divided by the number of tooth surfaces examined to obtain DI-S.

Calculus index (CI)

A no. 5 explorer is used for estimating the amount of supragingival and subgingival calculus. There are a total of 12 scores and a maximum no. of six segments to be examined. For DI and CI, the sequence of the oral hygiene examination should proceed in the following manner: First, the buccal, then the lingual surfaces of the teeth in upper right posterior segment. Next, the labial and lingual surfaces of teeth in the upper anterior segment. And finally, the buccal lingual surfaces of the upper left posterior teeth. Same procedure continues in the lower arch, except from left to right, the lower left posterior segment, the lower anterior segment, and the lower right posterior segment.

The scoring criteria is as follows:

- Score 0: No calculus present
- Score 1: Supragingival calculus covering not more than one third of the exposed tooth surface
- Score 2: Supragingival calculus covering more than one third but not more than two third of the exposed tooth surface or presence of individual flecks of subgingival calculus around cervical portion of tooth or both
- Score 3: Supragingival calculus covering more than two third of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of tooth or both.

The calculus scores are totalled and divided by the number of tooth surfaces examined to obtain CI-S. The DI-S and CI-S scores are referred to as

- Good: 0.0 - 0.6
- Fair: 0.7 - 1.8
- Poor: 1.9 - 3.0

Once the DI-S and CI-S are calculated separately, then they are added together to acquire the OHI-S score, which is interpreted as follows:

- Good: 0.0 - 1.2
- Fair: 1.3 - 3.0
- Poor: 3.1 - 6.0

Gingival index (Loe, 1967)[13]

The scoring criteria is as follows:

- Score 0 - Absence of inflammation / normal gingival
- Score 1 - Mild inflammation; slight change in colour and slight edema; no bleeding on probing.
- Score 2 - Moderate inflammation, redness, edema and glazing, bleeding on probing.
- Score 3 - Severe inflammation, marked redness and edema, ulceration, tendency to spontaneous bleeding.

The scores around each tooth were totalled and divided by four to obtain gingival index (GI) score for the tooth. The gingival score for a subject was obtained by adding the score of each tooth divided by the number of teeth examined. The gingival health was categorized on the basis of scores obtained and evaluated as follows:

- Mild gingivitis - 0.1 – 1.0
- Moderate gingivitis - 1.1 – 2.0
- Severe gingivitis - 2.1 – 3.0

Probing pocket depth (PPD)

PPD was measured from gingival margin to the base of the pocket.

Clinical Attachment level (CAL)

CALwas measured from a fixed point to the base of the pocket.

PPD and CAL were measured using calibrated UNC-15 periodontal probe. It is a 15 mm long probe with millimeter marking at each millimeter and color coding at 5^{th}, 10^{th} and 15^{th} millimeter. The probe is inserted with a firm, gentle pressure (0.75 N) to the bottom of the pocket. The shank should be aligned with the long axis of the tooth surface to be probed. Each tooth was examined at four sites, namely mid-facial, mesio-facial, disto-facial and at centre of lingual surfaces. In the present study an occlusal stent was used as a fixed reference point (Clark et al., 1987)[14]. After placing the stent over the teeth, the distance from the border of the stent to the base of the pocket was measured in mm using UNC -15, keeping the probe on vertical grooves prepared on the occlusal stent as a reference point to avoid clinical variations at different time points of measurement. PPD and CAL of each subject was determined by adding all the individual scores and then dividing this by the total number of surfaces recorded, respectively.

To prepare the occlusal stents, upper and lower casts were made in dental stone from the recorded impressions of upper and lower arch. Occlusal stents were fabricated with cold cure acrylic resin for each patient. It is used to cover the occlusal surfaces of the teeth to be recorded and extended buccally and lingually to cover more than occlusal one third of the crown. Four grooves in relation to a tooth were made in the stent to guide the periodontal probe at the time of recording the pocket depths and clinical attachment levels.

St. George's Respiratory Questionnaire (SGRQ):

Lung function measurements alone do not necessarily reflect the patient's disability. In order to measure the impaired health and quality of life in chronic airway disease, The St. George's Respiratory Questionnaire (SGRQ) was developed by Jones *et al.* (1992, 1997)[15, 16].

SGRQ is a disease-specific, 50-item questionnaire designed to measure impact on overall health, daily life, and perceived well-being (quality of life) in patients with obstructive airways disease. Scores are calculated for three domains: symptoms (distress due to respiratory symptoms), activity (the effects due to impairment of mobility or physical activity) and impacts (the psychosocial effect of the disease on the individual)as well as a total score. The SGRQ has been used in a range of disease groups including asthma, chronic obstructive pulmonary disease (COPD) and bronchiectasis. Repeatability, reliability, validity and sensitivity have been demonstrated in clinical trials. It correlates significantly with other measures of disease activity such as cough, dyspnoea, 6-minuteswalk test and FEV1 as well as the measures of general health such as the sickness impact profile and surfactant factor 36.

A full mouth periodontal examination was performed in all the subjects on day 0. The clinical

parameters and the SGRQ were re-evaluated at the end of 12 months of therapy.

The data were collected and analysed statistically. The Excel and SPSS (SPSS Inc, Chicago) version 17.0 software packages were used for data entry and analysis.

Results & Observations

The study was proposed to evaluate the association of periodontal health and COPD, and to assess the effectiveness of phase I periodontal treatment on the quality of life in COPD patients.

The correlation of FEV_1 values and periodontal parameters on day 0 is shown in Table 1. *r*-values measure the correlation between FEV_1 and periodontal parameters. As shown in the Table 1, a negative correlation between FEV_1 and all the periodontal parameters was observed, which is very highly significant statistically. On plotting, the steepness of the regression line shows the extent of correlation. As the line is slanting towards the right side, thus, it indicates a negative correlation between the FEV_1 and all the periodontal parameters. It refers a subject with decreased FEV_1 (severe COPD) will have higher GI, OHI-S, PPD and CAL compared to that of less severe COPD.

FEV_1 values were correlated with the SGRQ scores involving symptom, activity and impact scores to evaluate the quality of life in COPD subjects, shown in Table 2.A negative correlation between FEV1 and all the SGRQ scores was observed, which is very highly significant statistically. On plotting, the steepness of the regression line shows the extent of correlation. As the line is slanting towards the right side, it indicates a negative correlation between the FEV1 and all the SGRQ scores. It refers that lower the FEV1 (severe COPD), higher is the SGRQ scores compared to that of less severe COPD. These findings suggest that severe COPD patients have poor quality of life.

As shown in Table 3, all the periodontal parameters were increased in group A from day 0 to 1 year. However, the differences were not significant statistically ($p > 0.05$). In contrast, all the periodontal parameters were decreased in group B from day 0 to 1 year, which were found to be statistically significant ($p < 0.05$), except PPD ($p > 0.05$).

As shown in Table 4, all the scores of SGRQ were increased in group A from day 0 to 1 year. However, the differences were not significant statistically ($p > 0.05$). In contrast, all the parameters were decreased in group B from day 0 to 1 year. Amongst these parameters, activity score was found to be statistically significant ($p < 0.05$).

On intra group comparison, the differences in SGRQ activity score of case group was found to be statistically highly significant after one year compared to the control group ($p < 0.01$), but no significant differences in symptom, impact and total scores was observed between day 0 and one year in both the groups (Table 4).

Table 1: Correlation Coefficient of FEV_1 with periodontal parameters

Parameters	Pearson's Correlation Coefficient		
	r value	p value	Significance
FEV_1& GI	-0.939	0.000	***
FEV1 & OHI-S	-0.965	0.000	***
FEV1 & PPD	-0.935	0.000	***
FEV1 & CAL	-0.939	0.000	***

Table 2: Correlation Coefficient of FEV1 with SGRQ scores

Parameters	Pearson's Correlation Coefficient		
	r value	p value	Significance
FEV_1& symptom score	-0.933	0.000	***
FEV1 & activity score	-0.932	0.000	***
FEV1 & impact score	-0.927	0.000	***
FEV1 & total score	-0.947	0.000	***

Table 3 Clinical parameters in control and case at various time points

Parameters	Groups	Day 0	1 year	Day 0 *vs* 1 year	*p* value
		Mean ±SD (Range)	Mean ±SD (Range)		
Simplified oral hygiene index (OHI-S)	A (n=18)	3.57 ± 1.24 (1.66 - 5.16)	3.58 ± 1.29 (1.32 - 5.18)	0.01	0.979^{ns}
	B (n=17)	3.38 ± 1.27 (1.75 – 5.35)	2.32 ± 1.21 (1.21– 4.15)	1.06	0.018*
Gingival Index (GI)	A (n=18)	1.90 ± 0.51 (0.93 - 2.7)	2.03± 0.52 (1.1- 2.8)	0.13	0.43^{ns}
	B (n=17)	1.79 ± 0.54 (0.89 –2.56)	1.19 ± 0.47 (0.5– 2.1)	0.60	0.001***
Probing pocket depth (PPD) (mm)	A (n=18)	3.51 ± 0.56 (2.59- 4.21)	3.55 ± 0.57 (2.59 - 4.56)	0.04	0.841^{ns}
	B (n=17)	3.39 ± 0.78 (2.45 – 4.50)	3.15 ± 0.68 (2.31 - 4.21)	0.15	0.333^{ns}
Clinical Attachment Level (CAL) (mm)	A (n=18)	3.98 ± 0.60 (2.89 – 5.33)	4.01 ± 0.62 (2.89 – 5.41)	0.03	0.87^{ns}
	B (n=17)	4.17 ± 0.75 (2.99 – 5.75)	3.26 ± 0.90 (1.56 – 4.91)	0.91	0.003**

SD = Standard deviation
ns= not significant (p >0.05)
*= Statistically significant (p < 0.05)
**= highly significant (p < 0.01)
***= very highly significant (p < 0.001)

Table 4 SGRQ scores in control and case at various time points

SGRQ scores	Groups	Day 0	1 year	Day 0 vs 1 year	*p* value
		Mean ±SD (Range)	Mean ±SD (Range)		
Symptom	A (n=18)	49.76 ± 21.08 (11.50 - 73.8)	51.40± 20.9 (11.50 - 75.09)	1.64	0.815^{ns}
	B (n=17)	48.32 ± 19.25 (10.18-73.19)	44.65± 19.94 (4.91-69.71)	3.67	0.588^{ns}
Activity	A (n=18)	55.05 ± 18.74 (17.12-79.67)	56.48± 18.31 (23.28-79.67)	1.43	0.817^{ns}
	B (n=17)	53.68± 16.37 (29.43-79.17)	38.20± 13.18 (23.59-54.54)	15.48	0.005**
Impact	A (n=18)	49.78±20.85 (8.75-78.76)	51.07± 19.33 (8.75-83.01)	1.29	0.849^{ns}
	B (n=17)	48.02± 17.30 (13.61-71.53)	36.16± 18.03 (8.84-60.14)	11.86	0.059^{ns}
Total	A (n=18)	51.42 ± 19.97 (12.00 - 78.21)	52.80± 18.89 (14.08-80.47)	1.38	0.833^{ns}
	B (n=17)	49.87± 16.69 (18.37-74.12)	39.38± 14.94 (13.16-60.03)	10.49	0.062^{ns}

SD = Standard deviation ns= not significant (p >0.05) *= Statistically significant (p < 0.05)
= highly significant (p < 0.01) *= very highly significant (p < 0.001)

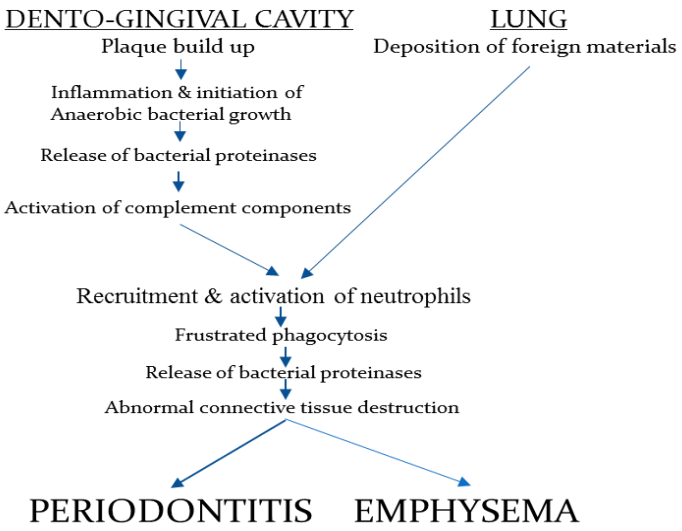

Figure 1: Parallel mechanisms in the development of emphysema and periodontitis (Travis et al., 1994)

Discussion

Systemic health has often been closely linked to the state of the oral cavity. Therefore, in the recent past, there has been greater concern in understanding the association of periodontal disease with many systemic conditions. Indeed, animal- and population-based studies now suggest that periodontal diseases may be linked with systemic diseases including cardiovascular diseases, diabetes, adverse pregnancy outcomes, osteoporosis and respiratory diseases (De Bowes, 1998; Fowler *et al.*, 2001)[17,3]. Both chronic periodontitis and COPD are neutrophilic, inflammatory conditions characterized by the loss of local connective tissue. Evidence suggests an association and perhaps a causal link between the two diseases (Gracia *et al.*, 2001)[18]. However, the nature of any relationship between them is unclear, but if it is established pathophysio-logically, it may help both the dental and medical professionals to determine the best approach to patient care in terms of targeted treatments to improve outcomes and prognosis.

It has been understood that poor oral health (periodontitis) alone is not responsible for COPD, rather poor oral health may work as an adjunct with other factors, namely continued smoking, environmental pollutants, viral infections, allergies and/or genetic factors to promote the progression and/or exacerbation of COPD. However, enhancement of all the parameters of the SGRQ scores with the improvement of periodontal health suggest a possible causative role of poor oral health in COPD. This may support the observation of Wang *et al.*, (2009)[11]and Zhou *et al.* (2011)[19].

The mean differences in CAL, PPD, GI and OHI-S of group B between day 0 and after one year were found to be reduced significantly ($p < 0.05$). In contrast, all the periodontal parameters were observed to be increased in group A from day 0 to the end of 1 year, though not significant statistically ($p > 0.05$). This may due to the phase 1 therapy carried out in group B and progressive nature of the periodontal disease, if not treated and lack of oral health knowledge Wang *et al.*, (2009)[11].In this study, it has been found that the SGRQ symptom score, activity score, impact score and total score are decreased in group B from day 0 to after one year, in contrast of group A where all the parameters are increased. The differences were found to be statistically significant when compared between group B and group A at the end of one year. Therefore, the improvement in the total score obtained in the SGRQ after one year indicate that phase I periodontal treatment significantly influences the quality of life in COPD patients. This supports the findings of Wang *et al.*, (2009)[11]and Zhou *et al.*, (2011)[19], who have suggested that promoting dental care and oral health knowledge may improve the quality of life in COPD patients. Thus, the findings of the present study suggest that phase I periodontal therapy may play an important role in improving the quality of life in COPD patients.

Conclusion

The findings of the present study suggest a positive correlation between the severity of COPD and oral health, *i.e.* oral health status is poor with increased severity of COPD. Again, the observation suggests that phase I periodontal

therapy may play an important role in improvement of the quality of life in COPD patients. It is appropriate to conclude that the maintenance of oral health should receive topmost priority for leading a healthy and qualitative life. Although the evidence of an independent association between chronic periodontitis and COPD grows stronger, there remains a lack of definitive studies designed to establish causality and treatment effects. There is a need for future research to be focused on answering these questions involving larger sample size with long term follow up.

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