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Original Research

Assessing The Relationship Between Severity of Periodontitis and Chronic Obstructive Pulmonary Disease

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Abstract

Background and Aim: A relationship between poor periodontal health and COPD has been suggested by a number of recent microbiologic and epidemiologic studies. Aim of the study was to evaluate potential association between severity of periodontal disease with chronic obstructive pulmonary disease (COPD)

Material and Methods: The Department of Respiratory Medicine, Shantabaa medical college and general hospital was used to recruit 50 patients without COPD (control group) and 50 patients with COPD (test group) from the Outdoor Patient Department of Respiratory Medicine. The following metrics were used to measure periodontal health: a) probing pocket depth (PPD) (b) mean clinical attachment loss (CAL) and (c) oral hygiene index.

Results: Around 4% of the patients were in severe grading. Majority of participants were in Gold 1 category (52%). The level of exacerbations also increased with severity in the results. Probing Pocket Depth (PPD), Mean Clinical Attachment Loss (CAL) and Oral Hygiene Index (OHI) were measured with significant results. Patients with COPD had deeper pockets (3 ± 0.56), more clinical attachment loss (2 ± 1.45) and poor Oral hygiene status (4 ± 2.23) compared to the control population.

Conclusion: The results showed a positive correlation between periodontitis and Chronic Obstructive Pulmonary Disease (COPD). These findings indicate the potential importance of promoting oral hygiene in the prevention and treatment of COPD, although additional studies are needed to clarify the causal relationship between periodontitis and COPD and explore the biologic mechanisms underlying the observed association.

Key Words: Chronic Obstructive Clinical Attachment Loss, Pulmonary Disease, Oral Hygiene, Periodontitis

I NTRODUCTION

It has long been believed that respiratory infections may be found in the mouth cavity. It is made feasible by the anatomical connection between the mouth cavity and the lungs. However, for an infectious pathogen to enter the lower respiratory system, it must overcome complex mechanical and immunological defenses.[1]

Although the microorganisms can enter the lung through inhalation, aspiration of what pneumologists have long called oropharyngeal secretions is the most frequent way for infections to occur. Thus, it is possible that respiratory tract infections could be caused by oral microorganisms. However, the significance of oral flora in respiratory infection etiology has only lately been thoroughly investigated.[2]

Airflow-affecting pulmonary disorders may potentially be linked to the mouth flora. Chronic obstructive pulmonary disease (COPD) is the most common. Studies show that 14 million individuals suffer from COPD, and a recent survey classified the disease as the sixth biggest cause of death globally, accounting for 2.2 million deaths. Although tobacco use is the primary cause, germs, notably oral bacteria, may be crucial to the disease's development.[3]

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Thus, within the last 10 years, there has been a growing focus on comprehending the connection between periodontal disease and other systemic health consequences, such as chronic obstructive pulmonary disease (COPD).[4]

Chronic obstructive pulmonary disease (COPD) is characterized by excessive sputum production due to emphysema and/or chronic bronchitis. Long-term cigarette smoking is the most significant known risk factor for COPD.[5]

The initial colonization of oral/pharyngeal surfaces by microbial pathogens is critical for the exacerbation and progression of COPD. Along with oral bacteria and pro-inflammatory enzymes, the pathogens are then released into the salivary secretions. As a result, this secretion's contents have the potential to pollute and change the respiratory epithelium.[6]

Supporting bone and connective tissues are destroyed in periodontitis, a localized chronic inflammatory disease brought on by tooth plaque bacteria infecting the periodontal tissues. Mucosal epithelium may attach to oral bacteria in secretions that stick to mucosal surfaces in order to promote the generation of cytokines.[7]

Additionally, bacterial compounds in the aspirate may cause respiratory epithelial cells to produce cytokines, which would attract inflammatory cells. Respiratory pathogens may be more likely to infect the resultant inflammatory mucosal epithelium.[8]

In order to increase the likelihood that respiratory pathogens will colonize the lower respiratory tract, oral bacteria may change the conditions in the upper airway. The aspirate's enzymes may break down mucosal surface macromolecules, revealing receptors that allow respiratory infections to adhere and colonize. [9]

Additionally, cytokines from the oral tissues, such as the gingival crevicular fluids, may infiltrate entire saliva and pollute the distal respiratory epithelium. In order to attract inflammatory cells (such as neutrophils) to the area, these cytokines may cause respiratory epithelial cells to release additional cytokines. These inflammatory cells may harm the epithelium by releasing hydrolytic enzymes, which increases the epithelium's vulnerability to respiratory pathogen infection. [9]

Numerous recent microbiologic and epidemiologic investigations have revealed a connection between poor periodontal health and COPD. Thus, this study is being conducted to analyze the relationship between the severity of periodontitis and COPD.

Aim of the study was to evaluate potential association between severity of periodontal disease with chronic obstructive pulmonary disease (COPD).

MATERIALS AND METHODS

The Department of Respiratory Medicine, Shantabaa medical college and general hospital was used to recruit 50 patients without COPD (control group) and 50 patients with COPD (test group) from the Outdoor Patient Department of Respiratory Medicine. Every patient will have at least six natural teeth and be at least 18 years old. The study was taken into account data on the patient's demographics (gender and age), socioeconomic status (education—classified as illiterate or literate; household income—classified as Rs. <50,000/year or Rs. >50,000/year), and lifestyle (history of smoking—number of cigarettes smoked per day).

Patients were categorized according to the following criteria if a history of COPD is documented:
Classification of airflow limitation severity in COPD (based on post-bronchodilator FEV1)
· In patients with FEV1/FVC <0.70:
- GOLD 1—mild: FEV1≥80% predicted
- GOLD 2—moderate: 50% ≤FEV1 <79% predicted
- GOLD 3—severe: 30% ≤FEV1 <49% predicted
- GOLD 4—very severe: FEV1 <30% predicted
Revised combined COPD assessment

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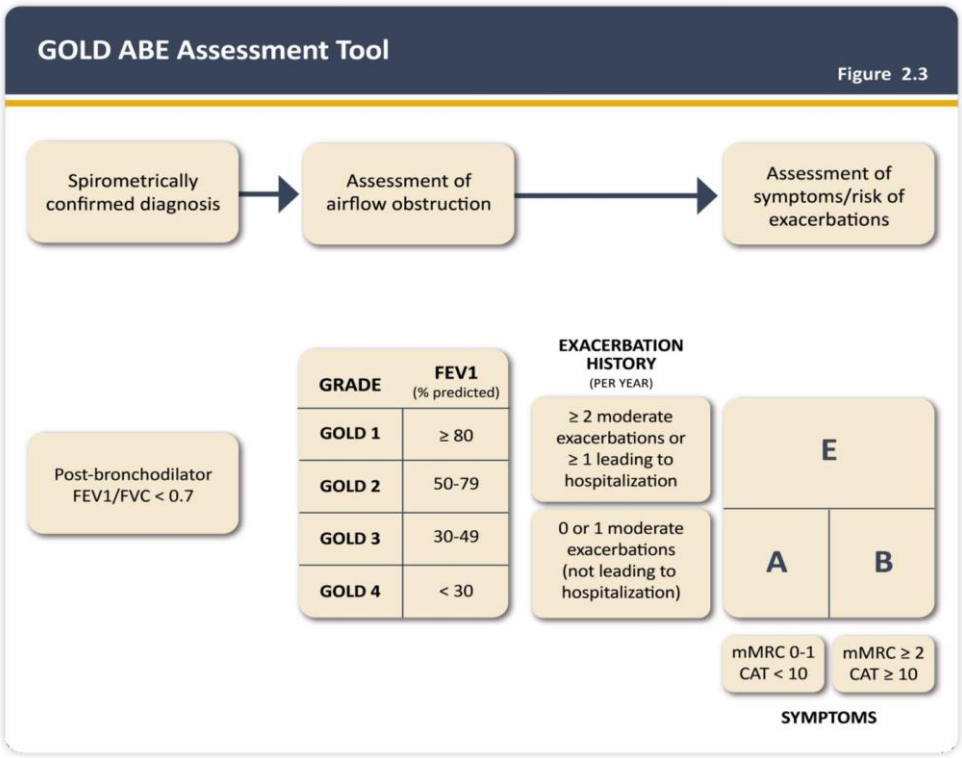

Gold Copd Abe Tool[6]

To ascertain the degree of airflow restriction (i.e., spirometric grade), patients underwent spirometry as part of the updated evaluation plan (above). Additionally, they were evaluated for dyspnea using the modified Medical Research Council (mMRC). Lastly, a record of their past hospitalizations and exacerbations were made.
In order to guide therapy, the letter (groups A to D) offers information about symptom load and exacerbation risk, while the number (spirometric grade 1 to 4) provides information about the degree of airflow limitation.
The following metrics were used to measure periodontal health:
(a) probing pocket depth (PPD) from the gingival margin crest to the periodontal pocket base; (b) mean clinical attachment loss (CAL) using William's graduated periodontal probe from the cement-enamel junction to the base of the periodontal pocket; and
(c) oral hygiene index (OHI, Greene and Vermilion; 1964), (10) which includes the debris index and calculus index.

The scoring for debris index is as follows:
0 - No debris or stain present.
1 - Soft debris covering not more than the gingival third of the tooth surface, or the presence of extrinsic stains without debris regardless of the surface area covered.
2 - Soft debris covering more than one-third but not more than two-third of the exposed tooth surface.
3 - Soft debris covering more than two-third of the exposed tooth surface.
The debris score was totaled and divided by the number of surfaces covered to obtain the simplified debris index.
The calculus index was calculated by using an explorer to estimate surface area covered by supragingival calculus.

The scoring was as follows:
0 - No calculus present.
1 - Supragingival calculus covering not more than one-third of the exposed tooth surface.
2 - Supragingival calculus covering more than one-third but less than two-third of the exposed tooth surface and/or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth.
3 - Supragingival calculus covering more than two-third of the exposed tooth surface and/or the presence of a continuous band of subgingival calculus around the cervical portion of the tooth.
The calculus scores were added and divided by the number of surfaces examined to obtain the simplified calculus index.

The simplified oral hygiene index (OHI) was calculated by adding together the simplified debris index and the simplified calculus index.

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A statistical software package 25 was used for data analysis. A two-sided P value <0.05 was considered to be statistically significant. Independent-samples t test was used to compare continuous variables, and x^2 test was used to compare categorical variables between the COPD and non-COPD groups.

Results

Table 1: Demographic details of target group in the population with COPD. p<0.05

GENDER	SOCIOECONOMIC STATUS	EDUCATION	HISTORY OF SMOKING (ACTIVE SMOKING)	NUMBER OF CIGARETTES SMOKED PER DAY	P value
MALE 30 ± 2.5	URBAN 10 ± 2.1	ILLITERATE 40 ± 2.6	YES 30 ±0.4	>25 PER DAY 40±1.2	0.06
FEMALE 20 ± 1.3	RURAL 40±1.2	LITERATE 10 ± 0.5	NO 20 ±0.6	<25 PER DAY 10±0.2	0.02

Table 2 : Demographic details of control group in the population without COPD. p<0.05

GENDER	SOCIOECONOMIC STATUS	EDUCATION	HISTORY OF SMOKING (ACTIVE SMOKING)	NUMBER OF CIGARETTES SMOKED PER DAY	P value
MALE 34 ± 2.2	URBAN 12 ± 2.1	ILLITERATE 33 ± 2.2	YES 32 ± 0.5	>25 PER DAY 39 ± 1.8	0.08
FEMALE 16 ± 1.5	RURAL 38 ± 1.8	LITERATE 17 ± 0.7	NO 18 ± 0.7	<25 PER DAY 11 ± 0.1	0.10

Table 3: Pulmonary assessment using the Gold ABE assessment tool

Grading	Score	Exacerbation history
Gold 1	Mild 26 (52%)	0
Gold 2	Moderate 12 (24%)	0
Gold 3	Severe 10 (10%)	1
Gold 4	Very severe 2 (4%)	2

Table 4: Mean characteristics of periodontal health among target group and control group (p <0.05)

Category	Probing Pocket Depth (PPD)	Mean Clinical Attachment Loss (CAL)	Oral Hygiene Index (OHI)	p-value (<0.05)
Target group (n=50)	3 ± 0.56	2 ± 1.45	4 ± 2.23	0.001
Control group (n=50)	1 ± 0.06	0 ± 0.06	1 ± 0.87	0.001

Demographic details are being summarized in table 1 and 2. The mean gender of patients with and without COPD was found to be around 30 ± 2.0. Patients with COPD were more likely to be men, current or former smokers, or had lower education. The results were insignificant in control group regarding the demographic details and non-COPD patients.

The GOLD ABE assessment tool was used to assess patients according to their level of symptoms and previous history of examinations. Symptoms were assessed using the mMRC. Exacerbations were also assessed independently of symptoms to highlight their clinical importance.

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Table 3 shows that around 4% of the patients were in severe grading. Majority of participants were in Gold 1 category (52%). The level of exacerbations also increased with severity in the results.

Table 4 shows the correlation between periodontal health and COPD patients as the Probing Pocket Depth (PPD), Mean Clinical Attachment Loss (CAL) and Oral Hygiene Index (OHI) were measured with significant results. Patients with COPD had deeper pockets (3 ± 0.56), more clinical attachment loss (2 ± 1.45) and poor Oral hygiene status (4 ± 2.23) compared to the control population.

DISCUSSION

In this study, a strong association between periodontitis and COPD was shown. The results showed that cases with frequent smoking are often the cases of COPD and have poorer periodontal health. This result can be correlated with the findings of Salmon A et al in 2003[1] who stated the similar results with similar frequencies. This study also showed that periodontal pocket depth was the main periodontal health-related factor for COPD. These findings indicate the importance of promoting oral hygiene in the prevention and treatment of COPD.

The GOLD guideline uses a combined "ABE" approach to assess patients according to their level of symptoms and previous history of exacerbations. *Cigarette smoking* is a key risk factor for COPD. Cigarette smokers have a higher prevalence of respiratory symptoms and lung function abnormalities, a greater annual rate of forced expiratory volume in (FEV) decline and a greater COPD exacerbation rate than nonsmokers, yet fewer than 50% of heavy smokers develop COPD.

Cigarette smoking was also seen to be associated with illiteracy as the findings showed a higher significance in lack of education and prevalence of cigarette smoking. This result can be correlated with the findings of William RC in 2000[2] who stated that lesser socio economic status leads to periodontal complications.

The exacerbation history increases with the increase in GOLD grading. There were more mortalities in very severe group compared to the mild group. This finding is in correlation with Garcia RI et al in 2001[5] where similar mortality findings were observed.

The Probing Pocket Depth (PPD), Mean Clinical Attachment Loss (CAL) and Oral Hygiene Index (OHI) values are on a higher side in target group. They specifically signify the relationship between COPD and periodontal health. These findings are in complete sync with the findings written by Hiremath SS in 2000[6] where the factors related to the periodontal complications are highlighted. It states that systemic complications are highly responsible for poor periodontal health.

The present study expands our previous findings to a larger sample and shows consistent and strong relationship between multiple periodontal indices and COPD status. In this study, cigarette smoking was found the main periodontal health-related factor for COPD. Debris accumulation is clearly an essential initial etiological factor in periodontitis, although the exact mechanism for the relationship between oral hygiene and COPD remains unclear. Some hypotheses suggest that bacteria in oral cavity may be aspirated along with respiratory pathogens and affect adhesion of the later organisms to the respiratory epithelium which subsequently cause lung disease.[11]

The present study is first one-of-a-kind analysis of the association between periodontitis and COPD in a closed population. The findings showed how crucial it is to encourage good dental hygiene in order to prevent and treat both conditions. The planning of oral health care for individuals with COPD might benefit greatly from such data, provided they are validated by subsequent prospective studies. In order to provide fresh perspectives on the pathogenic mechanisms behind the connection between COPD and periodontal health, the authors also hope that this work will encourage and support further function studies.

There are a few restrictions that should be taken into account. First, this study had Berksonian's bias, a form of selection bias that can happen in studies that only include hospital cases and controls. Second, due to the observational nature of this study, causality cannot be deduced.

Conclusion

A strong association between periodontitis and COPD was found. The results showed a positive correlation between periodontitis and Chronic Obstructive Pulmonary Disease (COPD). These findings indicate the potential importance of promoting oral hygiene in the prevention and treatment of COPD, although additional studies are needed to clarify the causal relationship between periodontitis and COPD and explore the biologic mechanisms underlying the observed association.

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