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Original Research Paper

Periodontics

THE RELATIONSHIP BETWEEN PERIODONTAL DISEASE AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE - A CASE CONTROL STUDY IN A TERTIARY CARE CENTRE.

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ABSTRACT

Aims and objectives: This present study was done to investigate the relationship between periodontal disease and chronic obstructive pulmonary disease. **Materials and Methods:** The present cross-sectional study was conducted on 135 patients in which a total of 65 COPD patients and 70 controls with normal pulmonary function were recruited from the Respiratory Medicine Department in association with the Department of Dentistry after taking informed consent to participate in the study. Spirometry was used for the confirmation of lung diseases. We assessed the clinical parameters such as simplified oral hygiene index (OHI-S), plaque index (PI), gingival index (GI), pocket probing depth (PPD), and clinical attachment level (CAL) with the help of SPSS software 21 and compared between cases and controls. **Results:** Individuals in the case group had significantly higher OHI-S, GI, PPD, and CAL ($p < 0.001$) compared with the control group. A significant positive relationship was observed in case group p values, thus suggesting a significant association between COPD and its progression with worsening of periodontal indices. **Conclusion:** A strong association between periodontitis and COPD was found in this study. The patients with COPD showed poor oral hygiene and a higher prevalence of periodontal disease. Both share similar pathophysiology, risk factors and microbial infection. Implementation of proper oral hygiene practices and suitable interventions to control periodontitis should be considered as a vital part of COPD management.

KEYWORDS : Chronic obstructive pulmonary disease, periodontal diseases

BACKGROUND

Periodontitis is defined as a chronic multi-factorial disease associated with dysbiotic plaque biofilms, characterized by loss of periodontal support, clinical attachment, loss, gingival bleeding, periodontal pocketing and alveolar bone loss[1]. Commonly due to bacterial infection and inflammation of the gums, which can lead to the development of periodontitis. It results in a buildup of plaque on the teeth that advances towards the gum and leads to tooth loss and bone damage. Studies suggest oral bacteria and local inflammatory response in periodontal tissues contribute to systemic inflammation which increases the risk for the development of chronic inflammatory conditions including diabetes, cardiovascular and respiratory disease [2].

Chronic obstructive pulmonary disease (COPD) is a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production) due to abnormalities of the airways (bronchitis, bronchiolitis) and /or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction.

(GOLD GUIDELINES 2023)[3].COPD includes a spectrum of pathology that involves damage to the lung parenchyma (emphysema) and abnormalities of the tracheo- bronchial tree (bronchitis).COPD is a significant health problem affecting more than 400 million people worldwide. In 1990, COPD was the sixth most common cause of disease worldwide but now ranks as the third leading cause of death, according to the Global Burden of Disease Study, after cardiovascular disease and stroke(WHO-2019)[4].

About 90% of COPD patients have a history of smoking, and mortality from COPD is about 10 times higher among smokers compared to non-smokers (Snider, 1989). However, since the incidence of COPD is 15%–20% in smokers and also occurs in those who have never smoked (American Thoracic Society, 1996), there may be factors that affect susceptibility to cigarette smoke, as well as risk factors other than tobacco smoke[1].

Periodontitis is associated with an increased risk of development of COPD. Both share several common risk factors such as age, smoking history, stress and ethnicity[5]. These diseases also share similar pathophysiology, characterized by inflammation, neutrophil recruitment and release of proteolytic enzymes destroying the pulmonary alveolus or destruction of the periodontal tissues[6]. Patients with confirmed COPD have lower tooth brushing frequency and poorer oral hygiene periodontal health compared to control groups[7].

The objectives of this paper are to assess the association between periodontal diseases and COPD, controlling the effect of sociodemographic characteristics, status of oral health, lifestyle variables, and comorbidities.

MATERIALS AND METHODS

Study Population

From 1/3/2022 to 1/9/2022, we conducted a case–control study in which a total of 65 COPD patients and 70 controls with normal pulmonary function were recruited from the Respiratory Department of GMC Kota in association with the Department of Dentistry. All are diagnosed to have periodontitis. Controls were frequently matched with COPD patients in age, sex, body mass index, and smoking status. All COPD cases were confirmed by pulmonary function testing (PFT) and they were on medications for more than two years.COPD diagnosis and classification of severity were based on the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines. Physician-diagnosed COPD from stage II to IV (moderate to very severe), post-bronchodilator forced expiratory volume in first second/forced vital capacity (FEV1/FVC) ratio <0.7 and FEV1 < 80% of the predicted value.

Other inclusion criteria for cases and controls included:

(1) aged 45 years and up with ≥ 15 teeth;
(2) Periodontitis from stage II to IV, criteria for diagnosing periodontitis stage were based on the new classification.

Exclusion criteria for cases and controls:

(1) Having features of exacerbation's or respiratory failure
(2) History of other systemic comorbidities
(3) History of any periodontal treatment in the last 6 months
(4) Had received systemic antibiotic therapy within the previous 6 months.

METHODOLOGY

The following parameters were evaluated Community periodontal index (WHO. 1982), plaque index (Loemodification - Silness and Loe., 1967), gingival index (Loe modification - Loe and Silness., 1967), loss of attachment (LOA), and pocket depth measurement were performed using the WHO probe. Spirometry was used to assess the pulmonary function.

Statistical Analysis

Qualitative data will be expressed as percentages and proportions. Statistical analysis will be conducted using SPSS 21 software. A P-value less than 0.05 will be considered as statistically significant and $P < 0.01$ was considered as highly statistically significant.

RESULT

COPD is nowadays considered a systemic disease with implications for all body systems. The relationship between COPD and Periodontal diseases warrants proper attention. After statistical analysis, the following results were obtained.

In the present study, the case group comprised 40 (61.5%) males and 25 (38.5%) females, whereas the control group had 55 (78.5%) males and 15(21.5%) females, and this difference was not statistically significant (p= 0.782, NS). The mean age was higher in the control group (46 ± 12.21 years) compared to the case group (42±10.11 years), and this difference was statistically significant (p< 0.001S) [Table 2].

When smoking status was compared, in the case group, 27(41.6%) were current smokers and 38 (58.4%) were past smokers. However, in the control group, 30 (42.8%) were current smokers, 21(30%) were past smokers and 19 (27.2%) were nonsmokers. There was a statistically significant difference (p=0.002)between the smoking statuses of both groups [Table 2].

The mean OHI-S score, PI score, and GI score were higher in the case group compared to the control group. However, a test of significance of the mean values showed that the case group had statistically no significantly higher values of PI score compared to the control group (p< 0.21). The mean PPD score and CAL score were significantly higher in the case group compared to the control group (p < 0.001) [Table 3].

According to the global initiative for chronic obstructive lung disease (GOLD), the COPD group (case) is subdivided into mild, moderate, severe, and very severe. The COPD intragroup comparison was done to evaluate the effect of the periodontal status in the subgroup of COPD group (case) patients.

The mean score of OHI-S, PI, GI, PPD, and CAL was higher in the moderate and severe COPD group patients compared to the mild COPD group patients; however, this difference was not statistically significant [Table 4].

Table 1:COPD severity classification

GOLD TYPE	SEVERITY	FEV1
GOLD 1	Mild	FEV1 ≥ 80% of predicted
GOLD 2	Moderate	50%<FEV1<80% of predicted
GOLD 3	Severe	30%<FEV1<50% of predicted
GOLD 4	Very severe	FEV1< 30%

Table 2: Demographic details of case and control groups

Demographic details	Case (COPD) (N=65)	Control (N=70)	p
Gender (%)			0.782
Male	61.5%(40)	78.5%(55)	
Female	38.5%(25)	21.5%(15)	
Age in years (Mean+ SD)	42+10.11	46+12.21	<0.001(S)
Smoking status			0.002(S)
Current smoker	41.6%(27)	42.8%(30)	
Past smoker	58.4%(38)	30%(21)	
Nonsmoker	-	27.2%(19)	

Table 3: Comparison of periodontal parameters of case and control groups

Periodontal parameters(Mean+ SD)	Case	Control	p
OHI-S	2.61±0.414	2.304±0.822	0.035 (S)
PI	2.00±0.740	1.66±0.437	0.21(NS)
GI	1.90±0.566	1.10±0.432	<0.001(S)
PPD (mm)	2.55±0.611	2.08±0.544	<0.001(S)
CAL (mm)	3.14±0.884	2.552±0.455	<0.001(S)

OHI-S: Simplified oral hygiene index, PI: Plaque index, GI: Gingival index, PPD: Probing pocket depth, CAL: Clinical attachment level

Table 4: Intragroup comparison of periodontal parameters in mm

COPD	OHI-S	PI	GI	PPD (mm)	CAL (mm)
Mild	2.33±0.503	1.56±0.891	1.26±0.529	2.07±0.742	3.06±0.109
Moderate	2.65±0.873	2.16±0.338	1.76±0.211	1.98±0.727	2.87±0.943
Severe	2.78±0.463	2.20±0.501	1.94±0.397	2.28±0.336	3.64±0.878
p	0.61(NS)	0.001(S)	0.19 (NS)	0.35 (NS)	0.141 (NS)

OHI-S: Simplified oral hygiene index, PI: Plaque index, GI: Gingival index, PPD: Probing pocket depth, CAL: Clinical attachment level, COPD-chronic obstructive pulmonary disease.

DISCUSSION

The relationship between chronic obstructive pulmonary disease, its comorbidities and its correlation with periodontal diseases has become an increasing focus of research.

It has been suggested that periodontitis and COPD are linked through a complex pathological process[8].Including neutrophilic inflammation, protease/anti-protease imbalance, oxidative stress and inflammatory cytokines. Periodontitis is linked to the severity of COPD through the microbial colonization of the respiratory system by dental plaque or airway inflammation caused by periodontal pathogens[8].

Neutrophilic inflammation is seen in both COPD and periodontitis. Neutrophilic lysis is seen in COPD patients and neutrophilic infiltration is a characteristic of periodontitis[9].

Cigarette smoke contains free radicals and reactive oxygen species, which cause oxidative stress and damage lung cells, further leading to tissue inflammation. Plasma antioxidant activities were decreased in patients with periodontitis when compared with a control group[10]. Neutrophil elastase and 1-antitrypsin (AAT) are a common protease/anti-protease pairing. AAT deficiency is an important pathogenic mechanism for COPD. Studies also indicated that the AAT and neutrophil elastase imbalance is also associated with periodontitis[8]. Numerous studies have confirmed that exists

microbial communication between periodontitis and COPD[8].

Periodontal bacteria Aggregatibacter actino mycetemcomitans, Capnocytophaga sputigena, Porphyromonas gingivalis, Tannerella forsythia and Treponema denticola were detected in the tracheal aspirate of severe acute exacerbation COPD[11].

The present study is a case–control study, performed to determine the association between periodontal disease and COPD. A total of 135 patients were included in the study. The control group comprised of 70 healthy patients, whereas the case group included 65 patients suffering from COPD. History and examination of both case and control groups were recorded.COPD cases were confirmed by spirometry. Case groups were further classified into mild, moderate, and severe based on the GOLD standards. All periodontal parameters including OHI-S, PI, GI, PPD, and CAL have been observed to increase as the severity of lung function impairment increases, i.e., FEV, the percentage decreases.

In this study, the age of the study population for both case and control groups is more than 30 years. The mean age of patients in the control group was significantly higher than that of the case group. The smoking tendency is more in the control group. Here significantly higher mean OHI-S score was found in the case group (2.61 ± 0.414) compared to the control group (2.304 ± 0.822). There was no significant change in PI score and significantly higher scores of mean GI were found in the case group (1.90 ± 0.566) compared to the control group (1.10 ± 0.432) and this difference was found to be statistically significant. The mean PPD values for case and control groups were 2.55 ± 0.611 mm and 2.08 ± 0.544 mm, respectively. The mean CAL values for case and control groups were 3.14 ± 0.884 mm and 2.552 ± 0.455 mm, respectively, and this difference was found to be statistically significant.

Plaque accumulation is an essential initial etiological factor in periodontitis. Dental plaque may also provide nutrition to the pathogens in the respiratory tract, especially in patients with poor oral hygiene[12]. Periodontal disease may alter environmental conditions to permit mucosal colonization and infection by respiratory pathogens[13].

It has been concurred that COPD are more prone to periodontal disease because of their poor oral hygiene. However, this study also has some limitations case-control is not the best design of observational study, evidence from these are less accurate and this study cannot completely exclude the possibility of other confounding factors. Future studies are needed to clarify the biological mechanisms underlying the observed association.

CONCLUSION

A strong association between periodontitis and COPD was found in this study. Both share similar pathophysiology, risk factors and microbial infections. Periodontitis leads to systemic inflammation which further causes COPD progression. So the implementation of proper oral hygiene practices and the suitable interventions to control periodontitis should be considered as a vital part of COPD management. It can significantly reduce the burden of COPD and improve the overall quality of life for affected individuals.Future research is needed to clarify the intricate mechanisms underlying the observed association.

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Nil.

Conflicts Of Interest
There are no conflicts of interest.

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