Original Article

Periodontal status and oral health behavior in hospitalized patients with chronic obstructive pulmonary disease

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Abstract

Aim: We evaluated the periodontal health status and oral health behavior among hospitalized patients with chronic obstructive pulmonary disease (COPD) to assess the association of COPD with dental health. **Materials and Methods:** A group of 100 hospitalized patients with COPD and a group of 100 age, sex, and race-matched control patients were included in this study. Detailed case histories along with standardized measures of oral health including gingival index, plaque index (PI), and simplified oral hygiene index (OHI) were estimated and compared. Probing depths and clinical attachment levels (CALs) were recorded at four sites per tooth. C-reactive protein (CRP) levels in saliva and serum were also measured. **Results:** The study subjects had similar demographics and distribution in either group. Patients with COPD had significantly lower brushing frequency, poor periodontal health (OHI and PI), greater gingival inflammation, and deeper pockets/CALs compared to controls. Further COPD patients had significantly higher serum and salivary CRP levels compared to control groups. **Conclusions:** Lower brushing frequency, poor oral health, and presence of destructive periodontal disease were observed among patients with COPD, which warrants promoting dental care and oral health knowledge as an integrated approach to treating COPD patients.

Key words: Chronic obstructive pulmonary disease, C-reactive protein, periodontal infection, poor periodontal health, risk factor

INTRODUCTION

Systemic illness may often impact dental health. Indeed, several studies have associated periodontitis with other systemic diseases such as type 2 diabetes mellitus, cardiovascular and pulmonary diseases.[1-3] Hence, it can be hypothesized that oral health may be an important indicator of systemic status, including respiratory diseases such as chronic obstructive pulmonary disease (COPD).

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COPD is a progressive chronic disease which is characterized by an inexorable decline in respiratory function, exercise capacity, and health status.[4] Over 3 million deaths globally every year are due to COPD, making it the fourth highest cause of death worldwide, which is progressively increasing.[5] Acute exacerbations of COPD are defined as the worsening of COPD symptoms, which are commonly observed as an increase in cough,

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sputum production, purulence, and dyspnea. In 50-70% of acute exacerbations of COPD, the pathophysiological basis is usually infectious.[6] Haemophilus influenzae is the most frequent bacterium isolated in most cases, followed by *Streptococcus pneumoniae*, *Moraxella catarrhalis*, and *Pseudomonas aeruginosa*.[6-9]

The relationship between periodontitis and COPD has recently received much attention. Periodontitis is a chronic inflammatory condition consequence to bacterial infections that results in the destruction of the tooth-supporting connective tissue and bone. Oral pathogens and inflammatory cytokines from periodontal lesions induce systemic inflammation, which may contribute to the pathogenesis of COPD.[10] Poor host defense may further exacerbate microbial growth and promote subsequent tissue destruction.[11-13] Indeed, dental plaque may serve as a reservoir for systemic pathogens, which may be evident as poor oral hygiene.[11]

Exacerbations of COPD are one of the most common causes for hospital admission globally and in India. Hence to evaluate the role of periodontal status contributing to exacerbations of COPD, we assessed the oral health among COPD inpatients.

MATERIALS AND METHODS

Study population

One-hundred hospitalized patients with COPD at Department of General Medicine, Civil Hospital, Ahmedabad, Gujarat, India were included in the study. Similarly, 100 age, sex, and race matched systemically healthy subjects from the outpatient clinic, Department of Periodontics, Government Dental College and Hospital, Ahmedabad, Gujarat, India were considered as potential controls. Inclusion exclusion criteria were set and systematic random sampling was used to select cases and controls.

Inclusion criteria

(1) Patients who were hospitalized for >3 days and diagnosed with acute respiratory disease (i.e., pneumonia, acute bronchitis, or a lung abscess) or an exacerbation of chronic respiratory disease (i.e., COPD, which included chronic bronchitis and emphysema);
(2) Patients with no past or present history of respiratory disease were considered potential controls; and
(3) Patients were aged 25-75 years and having at least 20 remaining natural teeth were considered for the study.

All COPD patients were clinically checked for barrel-shaped chest, hyper resonant note on percussion and bilateral bronchi. Criteria used for diagnosis were based on the Global Initiative for Chronic Obstructive Lung Disease spirometry guidelines.[32,36]

Exclusion criteria

Patients with:

(a) History of systemic diseases other than respiratory diseases.
(b) Under any medication known to influence periodontal status.
(c) Having history of any periodontal treatment in past 6 months (day) hospitalized in intensive care units were excluded from the study.

Following institutional and ethical board approval, written consent of patients was sought. The hospital records pertaining to each patient during the study period were reviewed to match inclusion criteria. All the data of the survey were obtained on the basis of the detailed case history and included information regarding patient's age, sex, socioeconomic status (SES), and brushing frequency. SES variables included educational qualification and monthly income. All patients who had a history of smoking were heavy smokers (>20 cigarettes/day).

Clinical evaluation

The following standardized measures of oral health were performed: The gingival index (GI),[14] the plaque index (PI),[15] and the simplified oral hygiene index (OHIS).[16] Probing depth and clinical attachment level (CAL) were measured to the nearest mm. The CAL was obtained by subtracting the distance from the free gingival margin (FGM) to the cement-enamel junction as a reference point of each tooth from the distance from the FGM to the bottom of the sulcus in the absence of recession. In the presence of recession, CAL was calculated by adding distance from FGM to the cementoenamel junction to the pocket depth. An oral examination was performed under proper illumination with the case group patient sitting erect on a bed and with a mouth mirror and University of North Carolina-15 periodontal probe. A single qualified clinician performed the clinical evaluation of the parameters.

A whole unstimulated saliva sample was obtained from the patients and controls and stored as aliquots at −20°C for C-reactive protein (CRP) determination. A sample of 2 mL blood from patients and controls was collected into vacutainer tubes, centrifuged, and the serum was separated and stored at −20°C for the estimation of CRP level.

Statistical Analysis

The data (mean [standard deviation [SD]) were analyzed by SPSS statistical package (version 12.0, SPSS Inc., Chicago, IL, USA) using un-paired *t*-test to compare means between cases and controls for brushing frequency,

PI score, GI score, OHIS score, PD, CAL, and CRP values in saliva and blood. $P < 0.05$ were considered to be statistically significant. Correlation between serum and saliva CRP was assessed by Pearson's correlation coefficient.

RESULTS

Demographic data of the study population are compared in Table 1. There was no significant difference in SES and educational qualification between COPD and control group. Mean values of the brushing frequency were significantly ($P < 00001$) lesser while GI, PI, OHIS, probing depth, and CAL were higher for patients with COPD compared to control group [Table 2].

Mean CRP levels in serum and saliva of COPD patients (90.08 [SD 17.89] μg/mL and 10.73 [SD 4.77] μg/mL, respectively) were significantly ($P < 0.0001$) higher than those in the controls (1.45 [SD 0.72] μg/mL and 0.74 [SD 0.17] μg/mL, respectively) [Table 3]. A significant and positive correlation was observed between serum and saliva CRP levels in the COPD patients ($r = 0.86$) and control subjects ($r = 0.62$).

DISCUSSION

Hospitalized patients with respiratory diseases consistently show poor periodontal status compared to controls. Cases with COPD had a significant difference in brushing frequency, PI, GI, OHIS scores, PD, and CAL compared to controls, which is consistent with previous reports.[17-19] Significantly greater mean OHI, mean PD, and mean CAL values were also reported to be associated with respiratory disease.[20-22] Although lack of association between gingival bleeding alone and respiratory disease is also reported.[20] Nevertheless, inappropriate tooth brushing method, lower regular supra-gingival scaling, and poor oral health knowledge remain significantly associated with COPD.[23]

Aspiration of pathogenic bacteria from the oropharynx into the lungs appears to play an important role in the pathogenesis of chronic pulmonary diseases.[11] Patients with periodontal disease have elevated levels of proteolytic bacteria (*Porphyromonas gingivalis* and *Spirochetes*) which produce proteases and together with elevated levels of various hydrolytic salivary enzymes destroy protective domains of host secretory components (e.g., mucins) and thus, diminish the nonspecific host defense against respiratory pathogens.[13] In light of complex and multifactorial nature of the respiratory disease, demonstration of a dose effect for such association between periodontitis and respiratory disease was not possible. However, high salivary concentrations of *P. gingivalis* were reported to enhance the risk of developing respiratory diseases.[24]

Oral pathogens and inflammatory cytokines from periodontal lesions induce systemic inflammation, which may contribute to the pathogenesis of COPD [Figure 1].[10] Bacteria can enhance pulmonary inflammation in COPD, which could potentially cause increased tissue damage due to leucocyte recruitment and proteinase secretion.[25-27] In periodontal disease, host response via immune defense system is considered

Table 1: Study population demographic

Variables	COPD (*n*)	Controls (*n*)
Age groups (years)		
25-35	12	15
35-45	28	20
45-55	25	30
55-65	23	26
65-75	12	9
Sex		
Male	62	57
Female	38	43
Education		
Nil	43	42
Till 5th class	27	23
5th-10th class	16	15
Above 10th class	14	20
Monthly income (INR)		
1000 and below	25	21
1000-5000	63	62
5000 and above	12	17
Smoking		
Smokers	53	11
Nonsmokers	47	89

Table 2: Comparison of means and SD of different criteria of COPD and control group

Parameter	COPD		Control group		*P*
	Mean	SD	Mean	SD	
Brushing	0.84	0.49	1.21	0.41	<0.0001
PI score	1.6672	0.58	1.0849	0.35	<0.0001
GI score	1.6866	0.4	1.054	0.3	<0.0001
OHIS score	3.5697	1.22	2.1485	0.55	<0.0001
PD	2.57	0.85	1.76	0.70	<0.0001
CAL	3.16	0.53	2.12	0.82	<0.0001

PI: Plaque index, GI: Gingival index, OHIS: Simplified oral hygiene index, CAL: Clinical attachment level, SD: Standard deviation, COPD: Chronic obstructive pulmonary disease, PD: Probing depth

Table 3: Comparison of means and SD of CRP level in serum and saliva of COPD and control group

Parameter	COPD		Control group		*P*
	Mean	SD	Mean	SD	
CRP (serum)	90.08	17.89	1.45	0.72	<0.0001
CRP (saliva)	10.73	4.77	0.74	0.17	<0.0001

CRP: C-reactive protein, SD: Standard deviation, COPD: Chronic obstructive pulmonary disease

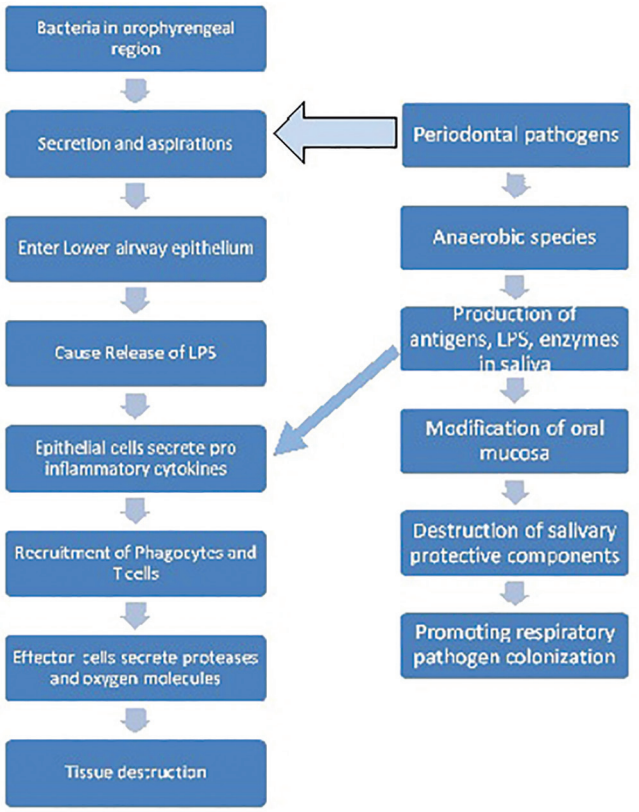

Figure 1: Mechanism for tissue destruction in COPD and role of periodontal pathogens

important, specifically, the cellular response. Thus, the higher prevalence of periodontal diseases reported among COPD patients may possibly be also due to activation of the immune system, inhaled drugs or an interaction between them.[28]

Quantitative changes of specific salivary biomarkers could have significance in the diagnosis and management of both oral and systemic diseases.[29] In our study, CRP levels in serum and saliva were significantly higher compared to patients in control group. Moreover, a significant and positive correlation was also observed between serum and salivary CRP levels of COPD and control patients. Indeed, significant differences in CRP levels between periodontitis patients with different grades of oral health are also reported.[30-35]

The association between periodontitis and COPD may have been confounded by shared risk factors especially smoking, which is the leading risk factor for periodontitis, emphysema, chronic bronchitis, and lung infections. As COPD is multifactorial and has a complex etiological profile, a demonstration of a dose effect for the association between periodontitis and COPD is challenging. Nevertheless, poor oral hygiene and poor periodontal status is consistently associated with the presence of respiratory disease specially aspiration pneumonia, hospital-acquired pneumonia, and COPD. It is, however, likely that periodontal pathogens may serve as the reservoir of microbiological etiology of COPD. Considering the pathogenesis of the periodontal and COPD, it seems possible that the inflammatory process underneath the periodontal disease may modify the respiratory epithelium and increase the risk of COPD. Hence, the CRP infection link cannot be ignored because CRP spikes during acute exacerbations of COPD and it will be very important to understand the effect of increased CRP levels in periodontitis on patients with COPD. The randomized clinical trials and clinicopathological study designs may be of long-term effectiveness when determining such association. As our study was cross-sectional and noninterventional with no microbiological evaluation, it limits determining a causal relationship between periodontitis and the COPD.

We conclude that presence of consistent poor oral hygiene, lower brushing frequency, and presence of destructive periodontal disease among COPD patients may be aggravating factors. Hence, oral health care maintenance must be given equal importance in the management of COPD.

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Conflicts of interest
There are no conflicts of interest.

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