**Original article:**

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

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**Abstract**

**Aim:** Aim of the present study is to evaluate the periodontal health status and oral health behaviours in hospitalized patients with chronic obstructive pulmonary disease (COPD).

**Methods:** A group of 100 cases (hospitalized patients with COPD) and a group of 100 age-, sex-, and race-matched outpatient controls (systemically healthy patients from the outpatient clinic, Department of Periodontics, Government Dental College and Hospital,Ahmedabad) were selected for the study. Detailed case history along with standardized measures of oral health were performed and compared included the gingival index (GI), plaque index (PI), and simplified oral hygiene index (OHI). Data regarding probing depths and clinical attachment levels (CALs) were recorded at four sites per tooth. Also CRP levels in saliva and serum of the patients in both the groups and compared statistically were determined.

**Results:** The comparison of study-population demographics on the basis of age, sex, education, and income showed no significant differences between groups. Patientswith COPD had significantly lower brushing frequency, greater poor periodontal health (OHI and PI), gingival inflammation (GI), deeper pockets, and CALs compared to controls. The difference between COPD patients and controls with regard to serum CRP and salivary CRP was highly significant ($P < 0.001$). There was significant correlation between serum CRP and salivary CRP in the COPD patients and controls.

**Conclusions: L**ower brushing frequency, poor oral health and presence of destructive periodontal diseasewere significantly associated with an increased risk of COPD. Our findings indicate the importance of promoting dental care and oral health knowledge that can be integrated into the prevention and treatment of COPD.

**Key words:** periodontal infection, risk factor, COPD, CRP, poor periodontal health.

## Introduction

Ever since the advent of the term periodontal medicine, research has amazed the world showing the relationship between periodontal diseases and various systemic conditions. Recent reports have implicated that periodontitis is associated with several other diseases including type 2 diabetes mellitus, cardiovascular disease, and respiratory system diseases [1-3.] It can thus be hypothesized that oral health may be an important risk factor or indicator of systemic status, including respiratory diseases like COPD.

Chronic obstructive pulmonary disease (COPD) is a progressive chronic disease which is characterised by an inexorable decline in respiratory function, exercise capacity, and health status [5]. Chronic Obstructive Pulmonary Disease (COPD) kills more than 3 million people every year, making it the 4th largest cause of death in the world[4]. It has been estimated that by the year 2030, COPD will become the third biggest cause of death.

Acute exacerbations of COPD (AECB) are defined as the worsening of COPD symptoms commonly characterized by increase in cough,

sputum production, purulence, and dyspnoea. In 50%–70% of acute exacerbations of COPD, the pathophysiological basis is usually infections [6]. Haemophilusinfluenzae is the most frequent bacterium isolated in all series followed by Streptococcus pneumoniae, and Moraxella catarrhalis, and Pseudomonas aeruginosa (Ball 1995; Monso et al 1995; Soler et al 1998; Miravitlles et al 1999).

The relationship between periodontitis and chronic obstructive pulmonary disease (COPD) has recently received much attention. Periodontitis is a chronic inflammatory reaction to bacterial infections that results in the destruction of the supporting connective tissue and bone of the dentition. Oral pathogens and inflammatory cytokines from periodontal lesions induce systemic inflammation, which may contribute to the pathogenesis of COPD [7]. Lower respiratory infection begins by the contamination of the lower airway epithelium by microorganisms contained in aerosolized droplets or by aspiration of oral secretions containing microorganisms [8.] Host defence systems fail to eliminate these pathogens from the mucosal surface, allowing further multiplication of the microorganisms, and subsequent tissue destruction [8-10.] It has been stated that "Dental plaque may serve as a reservoir for respiratory pathogens, especially in high-risk patients with poor oral hygiene" [8].

Exacerbations of COPD are one of the most common causes for admission in the hospitals globally and in India. Rightly so, identifying and modifying the factors responsible for its aetiology is the need of the day. If maintaining oral hygiene is an important part of preventing such diseases then probably the role of Periodontologist or dentist as a whole might prove to be much rewarding. Abiding thereby following study is planned to establish a substantial association between respitatory diseases and the periodontal status.

**Material and Methods:**

**Study population**

100 hospitalized patients with COPD from the civil hospital Ahmedabad, in the age group 25-75 years and having at least 20 remaining natural teeth were considered for the study. 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 d) hospitalized in intensive care units were excluded from the study. Similarly, 100 age, sex, and race matched systemically healthy subjects (new patients at the outpatient clinic, Department of Periodontics, Government Dental College and Hospital, Ahmedabad, Gujrat, India) were considered as potential controls.

After obtaining institutional and ethics board approval, written consent of patients was also provided. The hospital records pertaining to each patient during the study period in the hospital were screened. Subjects were also screened to ascertain whether they conform to the criteria of the study. All the information regarding the survey was obtained on the basis of the detail case history and included information regarding demographic data, socio economic status, educational qualification and brushing frequency.

**Clinical evaluation:**

The following standardized measures of oral health were performed: the gingival index (GI) ofLoe and Silness, the plaque index (PI) of Silness and Loe, and the simplified oral hygiene index (OHI) of Greene and Vermillion. Probing depth and clinical attachment levels were measured to the nearest mm. The clinical attachment level (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.

An oral examination was done under proper illumination with the case group patient sitting erect on a bed and with a mouth mirror and UNC-15 periodontal probe. The clinical evaluation of the parameters was carried out by single qualified clinician.

Whole unstimulated specimens of saliva were obtained from patients and controls and stored as aliquots at –20 °C for 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 °Cfor estimation of CRP level.CRP level was determined in serum and saliva of both patient and control groups by processing of the samples in the laboratory.

**Statistical Analysis:**

Data collected was tabulated into master sheets and later transferred to the computer spreadsheets. The data was analysed statistically using 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 value <0.05 was considered to be statistically significant.

**Results:**

Demographic data of the population in the study is compared in the Fig 1,2,3,4. There was no significant difference in socio economic status and educational qualification for cases and controls (Fig 3, 4). Mean values of the Brushing frequency, GI, PI, OHIs, Probing depth (PD), CAL and CRP in saliva and blood for patients with COPD were significantly higher than for the control group: brushing frequency (P=4.00268E-10), GI (P =3.80444E-25), PI (P =1.08424E-14 ), OHI (P =7.90204E-19), PD(P=2.17898E-10) and CAL (1.06723E-16). Standard deviation and mean values for brushing frequency, PI score, GI score, OHIS score, Probing depth and CAL are elaborated in Table.1

Mean CRP levels in serum and saliva of COPDpatients [90.08 (SD 17.89) µg/mL and 10.73 (SD 4.77) µg/mL respectively] were significantly higher than those in the controls [1.45 (SD 0.72) µg/mL and 0.74 (SD 0.17) µg/mL respectively]. The difference between COPD patients and controls with regard to serum CRP and salivaryCRP was highly significant ($P < 0.001$). There was significant correlation between serum CRP and saliva CRP in the COPD patients ($r = 0.86$) and controls ($r=0.62$) using Pearson correlation coefficient.

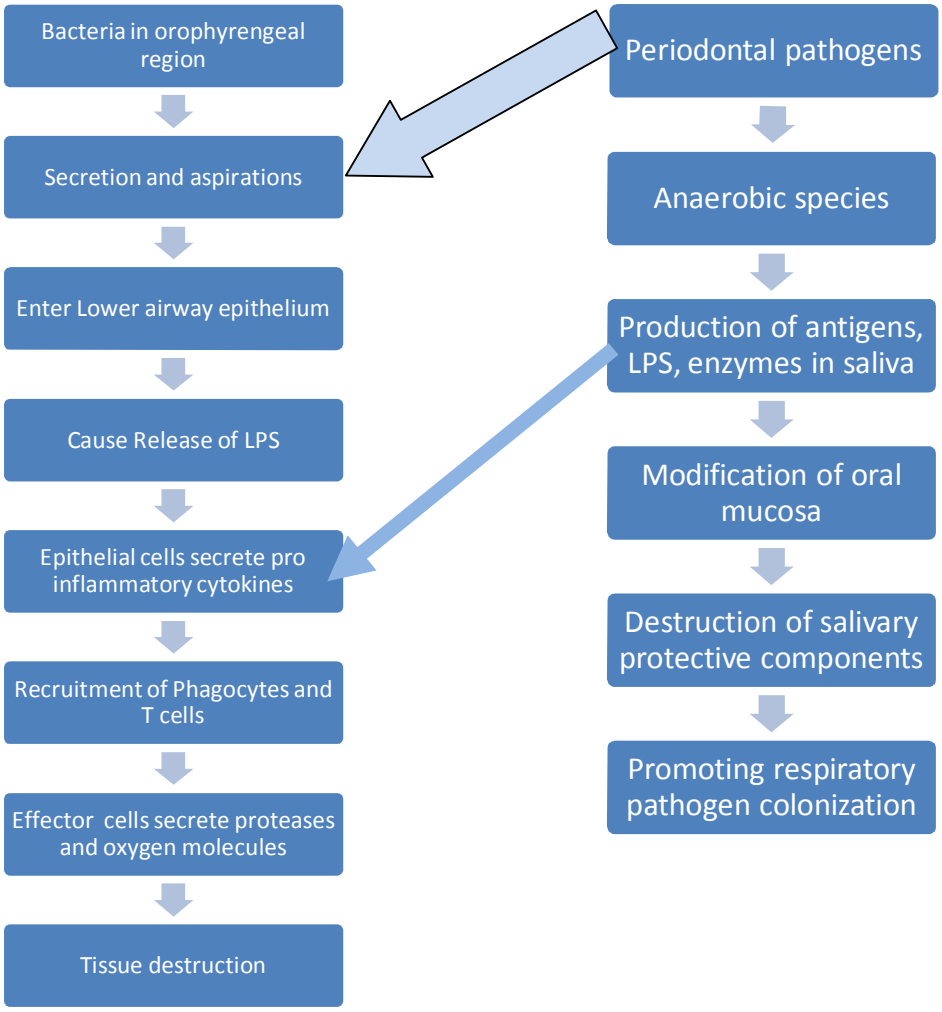
**Discussion:**

Results of our study state that hospitalized patients with respiratory diseases showed consistently poor periodontal status compared to controls. Cases with COPD had significant difference in brushing frequency, PI, GI, OHIS scores, PD and CAL compared to controls ($p<0.05$). Results of our study are in accordance with the results of study by Nikhil Sharma and H. Shamshuddin 2011[12] who stated patients with respiratory disease including COPD had significantly greater poor periodontal health (OHI and PI), gingival inflammation (GI) compared to controls. Significantly greater mean OHI, mean PD, and mean CAL values were also associated with respiratory disease as demonstrated by Scannapiecoet al.[14], Garcia et al.[15], and Hayes et al.[16] However study by Scannapieco and Ho 2001[14] found no significant relationship between gingival bleeding alone and respiratory disease. Study by Wang Z et al 2009.[17]stated that inappropriate tooth brushing method, lower regular supra-gingival scaling, and poorer oral health knowledge remained significantly associated with COPD.

Aspiration of pathogenic bacteria from the Oropharynx into the lungs appears to play an important role in the pathogenesis chronic pulmonary diseases. Subjects with poor oral health may have a

greater risk for colonization by respiratory pathogens and therefore a greater risk for respiratory infection[18]. Patients with periodontal disease and elevated levels of proteolytic bacteria such as Porphyromonasgingivalis and spirochetes produce enzymes (such as proteases). These enzymes and elevated levels of various hydrolytic salivary enzymes that are due to an increased dental-plaque load destroy protective domains of host secretory components (e.g.mucins) and, thus, diminish the non-specific host defense against respiratory pathogens in high-risk patients[10]. Also high salivary concentrations of P. gingivalis enhance the risk for respiratory disease with an oddsratio of 4.2[19].

Oral pathogens and inflammatory cytokines from periodontal lesions induce systemic inflammation, which may contribute to the pathogenesis of COPD[7]. Bacteria enhance pulmonary inflammation in COPD, which could potentially cause increased tissue damage due to leucocyte recruitment and proteinase secretion. The isolation of potentially pathogenic bacteria in bronchoalveolar lavage (BAL) fluid from COPD patients during bronchoscopy is associated with a higher neutrophil count and higher TNFα levels in BAL fluid than in culture negative patients [20]. In vitro studies have demonstrated higher levels of IL-6, CXCL8, and TNFα release following infection with H influenza [21]. Oral bacteria may stimulate periodontal tissues (epithelial cells, endothelial cells, fibroblasts, macrophages, white cells, peripheral mononuclear cells) to release cytokines such as IL-1a, IL-1b, IL-6, IL-8 and TNF-α [22]. These cytokines in turn lead to recruitment of phagocytes as well as T cells followed by release of proteases and oxygen radicals leading to tissue destruction. This makes respiratory epithelium further more susceptible to the bacterial colonization.




**Flowchart showing mechanism for tissue destruction in COPD and role of periodontal pathogens**

Quantitativechangesofspecificsalivarybiomarkerscouldhavesignificanceinthediagnosisandmanagementofbothoralandsystemicdiseases[25].InourstudyCRPlevelsinserumandsalivaweresignificantlyhigherthanthoseinthecontrols.Alsotherewasasignificantcorrelationbetweenserumandsalivary CRPinpatientsandincontrols

Christodoulidesetal.foundsignificantdifferencesinCRPlevelsbetween periodontitispatients with different gradesoforalhealth and normal subjects[28]. Salzberoetal.inhisstudyof93patientswithgeneralizedaggressiveperiodontitisand91healthycontrolsshowedthatserafrompatientswithperiodontalinfectionscontainelevatedlevelsofCRPcomparedwithindividuals with good periodontal health[29]. Y ZHANG et al.[30] suggested that patients with stable COPD had higher serum CRP concentrations than healthy controls and severe COPD was associated with a higher serum CRP concentration than moderate COPD.Several other

studies have shown that patients with COPD have higher systemic concentrations of CRP than healthycontrols [31-33].

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 the COPD is multifactorial and have a complex etiological profile, a demonstration of a dose effect for the association between periodontitis and COPD is rather unlikely. Hence, it is quite obvious that the plaque accumulation or periodontal disease does not directly cause respiratory or other systemic diseases. However poor oral hygiene and poor periodontal status are consistently associated with presence of respiratory disease specially aspiration pneumonia, hospital acquired pneumonia and COPD. Also periodontal pathogens may serve as the reservoir for its microbiological aetiology. 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. The CRP infection link cannot be ignored because CRP spikes during acute exacerbations of COPD.Exacerbations have been linked to accelerated and stepwise decline in lung function and worsening of COPD. It will be very important to understand the effect of increased CRP levels in periodontitis on patients with COPD.

The randomized clinical trials and clinico-pathological study designs may be of long term effectiveness when determining such association. As our study was cross sectional and non interventional with no microbiological evaluation, it limits determining a causal relationship between periodontitis and the COPD.

**Conclusion:**

Presence of consistent poor oral hygiene, lower brushing frequency and presence of destructive periodontal disease may be an aggravating factor for COPD. Hence, oral health care maintenance is to be given equal importance in management of these diseases, thereby emphasizing on maintaining oral hygiene and a healthy periodontium. In the future, large cohort studies and random clinical trials will be needed to investigate the causal effect of oral hygiene and periodontal health with COPD exacerbations and other respiratory diseases.

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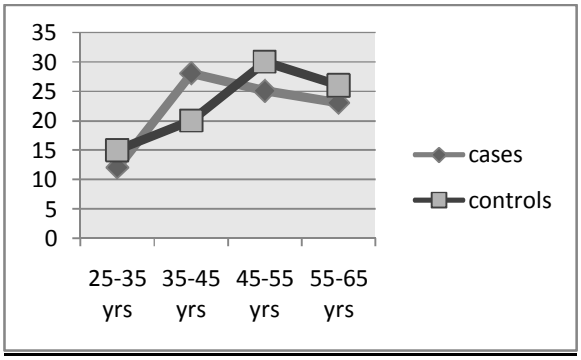
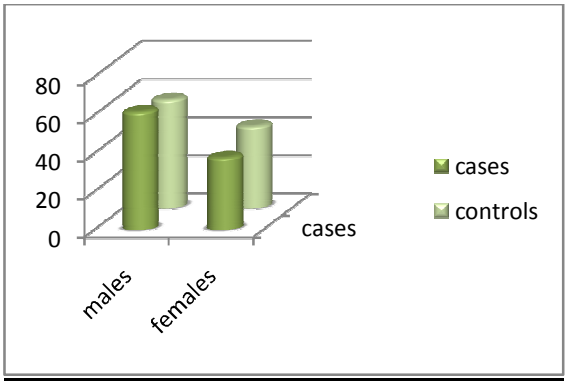
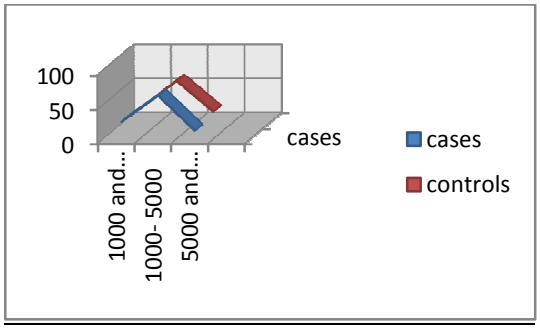
**Comparison between age of cases and controls (Fig 1.)**



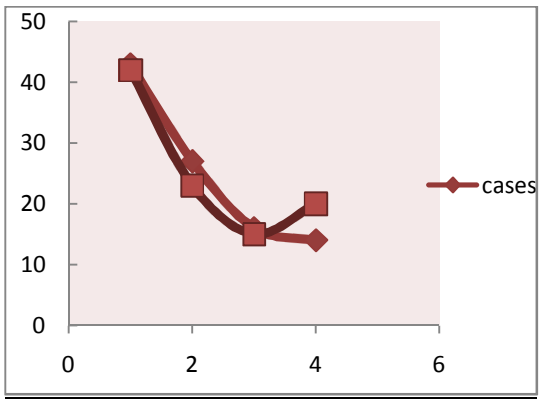

**Comparison of subjects in relation to gender in cases and controls (Fig.2)**



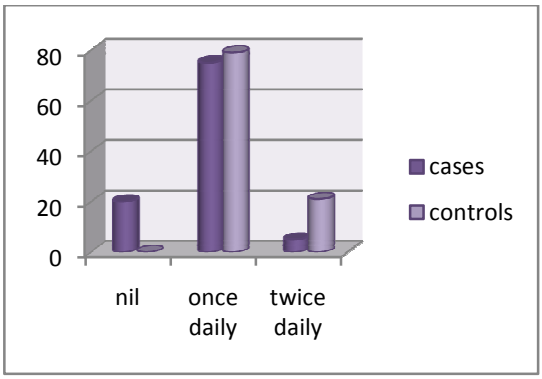
**Comparison of monthly income in cases and controls (Fig.3)**




**Comparison of education qualification in cases and controls (fig.4)**




**Comparison of brushing frequency in cases and controls (fig.5)**




**Comparison of Means and standard deviation of Different Criteria of COPD and Control Group (Table.1)**

| | COPD | | Control Group. | |
|---|---|---|---|---|
| | Mean | S.D. | Mean | S.D. |
| Brushing | 0.84 | 0.49 | 1.21 | 0.41 |
| PI score | 1.6672 | 0.58 | 1.0849 | 0.35 |
| GI score | 1.6866 | 0.4 | 1.054 | 0.3 |
| OHIS score | 3.5697 | 1.22 | 2.1485 | 0.55 |
| Probing depth | 2.57 | 0.85 | 1.76 | 0.70 |
| CAL | 3.16 | 0.53 | 2.12 | 0.82 |

**Comparison of Means and standard deviation of CRP level in serum and saliva of COPD and Control Group (Table.2)**

| | COPD | | Control Group. | |
|---|---|---|---|---|
| | Mean | S.D. | Mean | S.D. |
| CRP (serum) | 90.08 | 17.89 | 1.45 | 0.72 |
| CRP (saliva) | 10.73 | 4.77 | 0.74 | 0.17 |