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EVALUATION OF THE ASSOCIATION BETWEEN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) AND PERIODONTAL HEALTH AND DISEASE

Dental Science

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ABSTRACT

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is an inflammatory disease characterized by the accelerating deterioration of pulmonary function and increasing airway obstruction including chronic bronchitis and emphysema. Respiratory infection is speculated to build in part, firstly by aspiration of oropharyngeal bacterial flora into the lower respiratory tract where they accumulate and proliferate to develop infection and secondly, failure of host defence system to eradicate these harmful bacteria.

Aim and Objectives: To find out the association between the chronic obstructive pulmonary diseases using pulmonary function test and the periodontal health and disease using various periodontal parameters.

Materials and method: 100 patients diagnosed with COPD were taken as test group and 100 patients diagnosed non-COPD were the relatives of the COPD patients. Periodontal parameters including Oral Hygiene Index Simplified (OHI-S), Pocket Probing Depth (PPD), Gingival Recession (REC) and Clinical Attachment Loss (CAL) were recorded. Statistical analysis was done by using SPSS (Statistical Package for Social Sciences) version 17.

Results: The mean PPD (in mm) among COPD and Non-COPD participants was 5.06±0.86 and 3.61±0.78 respectively. Study result showed statistically highly significant difference in the mean PPD which is more in COPD participants compared to Non-COPD participants. Statistically significant difference was found in the mean REC which is more in COPD participants compared to Non-COPD participants. The mean CAL (in mm) among COPD and Non-COPD participants was 6.89±1.06 and 4.53±1.41 respectively. Statistically highly significant difference was seen in the mean CAL which is more in COPD participants compared to Non-COPD participants. The mean OHI-S (in mm) among COPD and Non-COPD participants was 2.46±0.33 and 2.37±0.32 respectively. Study result showed there wasn't any statistical difference in the mean OHI-S among COPD participants when compared to Non-COPD participants.

Conclusion: From the present study it can be concluded that oral bacteria, poor oral hygiene and periodontitis seem to influence the incidence of COPD in high risk patients. Periodontal disease was significantly associated with reduced lung function and airflow limitation in an adult population. However, large cohort studies with long observation periods evaluating comprehensive lung function parameters are needed to validate the observed associations and to assess causality more closely.

KEYWORDS

COPD, oral hygiene, periodontitis, smoking

Introduction

Periodontium ascribes to the specialized tissues that surround and support the teeth, and maintains them in the maxillary and mandibular jaw bones. Periodontitis leads to loosening of teeth, and subsequent tooth loss occurs, if left untreated, by implicating accelerated loss of alveolar bone that is present around the tooth. The formation and accumulation of sticky, colorless plaque on the teeth occurs by not only the bacterial forth with mucus and other particles but also along with poor oral hygiene. Plaque, which is not extracted by routine oral hygiene measures eventually hardens and forms a "tartar" like mass accumulation that cannot be removed by routine oral hygiene measures. This tartar like accumulation is called calculus. Inflammation from this calculus build-up causes a pocket formation between the teeth and gums, which is filled with plaque and calculus. Soft tissue deepening accessories plaque in the pocket and continuous plaque accumulation and inflammation leads to damage of the tissues and bone surrounding the tooth. As the plaque advances and cultivates beneath the gum line, the body's immune system starts to fight against these bacteria. The bone and the connective tissue, which holds the teeth in place in the oral cavity, start-off to breakdown by the bacterial toxins, which are produced by bacteria, and the body's accustomed response to this infection.[2]

Not alone bacteria and plaque causes periodontal diseases but several other added risk factors include poor oral hygiene habits, smoking, tobacco use, certain medications, stress, diabetes, ill-fitted dentures, older age, hormonal changes, poor nutrition, etc. Smoking and diabetes are absolute dangerous factors for development and progression of periodontal disease. For the majority of the population, healthy periodontal state can be finer maintained by routine oral hygiene practice along with prevention of behavioral and environmental hazardous factors. Because periodontal health and disease is affiliated to an added susceptibility to systemic infection (like, diabetes, cardiovascular disease, low birth weight, bacterial pneumonia, infective endocarditis), it's not alone important for oral health but as well for general health, to control periodontal disease.

The oral cavity perhaps can be a window to long-term health because oral lesions can act as a signal of disease development and progression. It has been found in various investigations that the bacteria that abound in the oral cavity cause respiratory diseases by aspiration into the lungs, adversely in populance with periodontal infections. Respiratory infection is speculated to build in part, firstly by aspiration of oropharyngeal bacterial flora into the lower respiratory tract where they accumulate and proliferate to develop infection and secondly failure of host defence system to eradicate these harmful bacteria. It has been recommended that patients with periodontal infections have large amount of dental plaque, which may act as a reservoir of respiratory pathogens.[1] Respiratory infection that may be caused by pathogenic bacteria, and this pathogenic bacteria may arrive in the dental plaque biofilm in the oral cavity. These pathogenic bacteria can then be taken off in huge quantities into the oral secretions and are in surroundings with cytokines along with other biologically active molecules present in inflamed gingiva.[2] Infection and inflammation of the lung occurs because of repeated aspiration of these oral secretions into the lower airway. Several mechanisms have been suggested to reveal the role of oral pathogens in the pathogenesis of respiratory diseases, which include the following:[1] (1) Aspiration of oral pathogens into the lung to ground infection (2) Periodontal infection related enzymes may abort salivary pellicles on pathogenic bacteria to obstruct their clearance from the mucosal covering (3) Periodontal infection related enzymes in saliva that may adapt mucosal surfaces to advance adherence and colonization by the respiratory pathogens that are again aspirated into the lung (4) Periodontal tissues produce cytokines that may adapt respiratory epithelium to advance disease by respiratory pathogens.

One such respiratory disease is Chronic Obstructive Pulmonary Disease (COPD) which is an inflammatory disease characterized by the accelerating deterioration of pulmonary function and increasing airway obstruction including chronic bronchitis and emphysema.[3] It's not absolutely reversible. COPD is an under-diagnosed deadly lung disease but, it is not directly a "smoker's cough". Different types of

COPD were earlier termed as **Chronic Bronchitis and Emphysema.** Sputum production along with shortness of breath and cough are the main manifestations of COPD. Its pulmonary component is outlined by airflow restriction which is not absolutely reversible. COPD not only centers on the prevalent element of altered lung function, but also recognizes both, the systemic attributes and the discrepancy of COPD. The adverse of COPD can aftereffect from several etiologies; a lot of frequently cigarette smoking that affects the mucociliary barrier and phagocyte activity, triggering an unusual inflammatory response in the lungs.[4] Besides smoking, added accident factors includes ageing, infections, toxic air pollutants, childhood respiratory infections etc.

Till date, scanty evidences are available that have examined the interrelationship between periodontitis and respiratory diseases with selected pulmonary function parameters. Purely on the support of Spirometry (FEV1, FEV1/FVC ratio), several cross-sectional and case-control analysis noted powerful affiliation between history of chronic obstructive pulmonary disease, lung function, and periodontal disease.[5,6,7,8,9] If periodontal infection is assigned as an independent dangerous factor for decreasing lung function, it would accommodate as one of the reason to consider periodontal prevention and treatment as a beneficial choice to spot deteriorating lung function or to at least support regimens for pneumological diseases. There are few recent studies that show weak correlation among periodontal diseases and Chronic Obstructive Pulmonary Disease. Tobacco smoking is the main cause that strongly holds both these altitude. It has also been said that smoking and impaired lung function was associated with impaired quality of life and was not influenced by dental health. Periodontal diseases and smoking are strongly associated with each other, while periodontal disease is weakly affiliated to lung tissue destruction and very weakly affiliated or even not at all affiliated with chronic airflow limitation[10].

Hence to further study the association between COPD and periodontal infection the need of the research is to figure out the association between chronic obstructive pulmonary disease (COPD) using pulmonary function test and periodontal health and disease using various periodontal parameters.

Materials and Method

A descriptive cross-sectional research was governed to figure out the association among the chronic obstructive pulmonary disease using pulmonary function test and the periodontal health and disease using various periodontal parameters.

The study was conducted after Institutional Ethics Committee approval was obtained. The study population consisted of the individuals attending the OPD of Respiratory Medicine, Dhiraj Hospital, Sumandeep Vidyapeeth, Piparia, Vadodara. Study population in the present study was preferred based on the following inclusion and exclusion criteria.

Inclusion criteria:

- Patients should be at least 18 years old.
- Patient should have at least 6 natural teeth.
- Patients who underwent Pulmonary Function Test.
- Patients with Non-COPD should be relatives of the COPD patients.

Exclusion criteria:

- Patient with compromised medical conditions like bleeding disorders, malignancy, etc.
- Pregnancy or lactating women.
- Physically or mentally challenged patients.

The sample size required for the study was 200 (100 per group) to get 0.3 difference in mean CAL between two groups at 95% confidence interval and 90% power. The sample size was obtained using N-Master statistical software. Investigation was fulfilled by a single researcher. The data was recorded by a trained examiner from the department. Intra examiner calibration of the examiner was carried out. The data obtained was evaluated applying Kappa Statistics. The co-efficient of 0.86 was constructed, reflecting a high degree of agreement in the observations.

An entire of 200 participants were enrolled in the research based on the inclusion criteria. Before enrolment of any participants in the study, all the subjects were briefed verbally about the objective of the research. Written information regarding the study was given and participants willing to take part in the investigation were enlisted and written informed consent was procured from tudy subjects.

Diagnosis of COPD was done using Pulmonary Function Test (Spirometry). Information of lung function was estimated by expert physician, by calculating the ratio of Forced Expiratory Volume after 1 second (FeV1)/Forced Vital Capacity (FVC) × 100.

The volume of air which can be blown out by force after one full inspiration is the Forced vital capacity (FVC). In COPD patients it's found to be less than 80%. The volume of air which can be strongly blown out in 1 second succeeding one full inspiration is the Forced expiratory volume in 1 second (FEV1). Average values between 80%-120% are considered normal while below 80% are diagnosed with obstructive diseases such as asthma, COPD, chronic bronchitis, emphysema. The ratio of FEV1 to FVC is FEV1/FVC. In normal healthy adult individuals, FEV1/FVC should be almost 70%-85% declining with age. When the ratio is less than 70%, the patient is said to have obstructive disease.[11] Using Spirometry, 100 patients were diagnosed with COPD taken as a test group and 100 patients diagnosed non-COPD, the relatives of the COPD patients were taken as controls. All the patients who were included in the research were at least 18 years or above and should have minimum of six natural teeth present. The study subjects were asked about smoking history and were divided into Never, former, non and current smokers according to the definitions developed and updated by the US Centers for Disease Control and Prevention. The study subjects were made to sit on the chair and the examination was done using plain mouth mirror, explorer and UNC-15 probe that were used to record the periodontal parameters including Oral Hygiene Index Simplified (OHI-S), Pocket Probing Depth (PPD), Gingival Recession (REC) and Clinical Attachment Loss (CAL).

Probing Pocket Depth was estimated from marginal gingiva to the base of the pocket, Gingival recession was measured from Cementoenamel Junction (CEJ) to the marginal gingiva, and Clinical Attachment Loss (CAL) was measured from the CEJ to the base of the pocket on the mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, disto-lingual sites, for patients with and without COPD.

Data analysis

The data obtained was coded and fed into the SPSS (Statistical Package for Social Sciences) version 16 for analysis. Descriptive statistics was used to account for mean and standard deviation. Independents test was used to compare PPD, REC, CAL and OHI-S among the test group (COPD participants) and Non-COPD participants. The p=0.05 was set as the level of significance.

Results:

The study population consisted of 200 participants in the age range of 18 to 65 years. 100 COPD and 100 Non-COPD participants were included with mean age of 47.31±1.45302 years and 48.74±15.04244 years respectively (Table 1).

Out of 100 COPD participants 69% were males and 31% were females while out of 100 Non-COPD participants 59% were males and 41% were females (Table 2).

The mean FEV1/FVC ratio was 60.7035±8.24184 and 103.5950±14.55632 in COPD and Non-COPD participants respectively (Table 3).

Table 4 shows the distribution of participants by smoking history among COPD and Non-COPD subjects. Maximum numbers of participants 43% were former smokers followed by 22% current smokers, 20% never smokers and 15% non-smoker among COPD participants. Maximum numbers of participants, 31% were non smokers followed by 29% were former smokers, 27% never smokers and 13% current smokers among Non-COPD participants.

Table 5 shows comparison of PPD among COPD and Non-COPD participants. The mean PPD (in mm) among COPD and Non-COPD participants was 5.0680±0.86910 and 3.6180±0.78539 respectively. Study result showed statistically highly significant difference in the mean PPD which is more in COPD participants compared to Non-COPD participants (p=0.000, HS).

Table 6 shows comparison of REC among COPD and Non-COPD participants. The mean REC (in mm) among COPD and Non-COPD participants was 1.8260±0.74191 and 0.9159±0.94445 respectively. Study result showed statistically significant difference in the mean REC which is more in COPD participants compared to Non-COPD participants (p=0.044, S).

Table 7 shows comparison of CAL among COPD and Non-COPD participants. The mean CAL (in mm) among COPD and Non-COPD participants was 6.8940±1.06304 and 4.5330±1.41572 respectively. Study result revealed statistically highly significant variation in the mean CAL which is more in COPD participants compared to Non-COPD participants (p=0.002, HS).

Table 8 shows observation of OHI-S scores among COPD and Non-COPD participants. The mean OHI-S (in mm) among COPD and Non-COPD participants was 2.46±0.33212 and 2.372±0.32352 respectively. Study result showed that there was no statistical variation in the mean OHI-S among COPD participants when compared to Non-COPD participants (p>0.05, NS).

TABLE 1: DISTRIBUTION OF MEAN AGE (IN YEARS) AMONG COPD AND NON COPD SUBJECTS

GROUPS	N	MEAN AGE (IN YEARS)	STANDARD DEVIATION
COPD	100	47.31	1.45302
NON-COPD	100	48.74	15.04244

TABLE 2: DISTRIBUTION OF GENDER AMONG COPD AND NON COPD SUBJECTS

GROUPS	SEX	FREQUENCY(%)
COPD	FEMALES	31(31)
	MALES	69(69)
	TOTAL	100(100)
NON-COPD	FEMALES	41(41)
	MALES	59(59)
	TOTAL	100(100)

TABLE 3: DISTRIBUTION OF MEAN FEV1/FVC RATIO AMONG COPD AND NON COPD SUBJECTS

GROUPS	N	MEAN FEV1/FVC (%)	STANDARD DEVIATION
COPD	100	60.7035	8.24184
NON-COPD	100	103.5950	14.55632

TABLE 4: DISTRIBUTION OF PARTICIPANTS IN RELATION TO SMOKING HISTORY AMONG COPD AND NON COPD SUBJECTS

GROUPS	SMOKING HISTORY	FREQUENCY (%)
COPD	NEVER SMOKER	20(20)
	NON SMOKER	15(15)
	CURRENT SMOKER	22(22)
	FORMER SMOKER	43(43)
	TOTAL	100(100)
NON-COPD	NEVER SMOKER	27(27)
	NON SMOKER	31(31)
	CURRENT SMOKER	13(13)
	FORMER SMOKER	29(29)
	TOTAL	100(100)

TABLE 5: COMPARISON OF PPD AMONG COPD AND NON COPD SUBJECTS

GROUPS	N	MEAN PPD (IN MM)	STANDARD DEVIATION	P VALUE	95% CONFIDENCE INTERVAL OF THE DIFFERENCE	
					LOWER	UPPER
COPD	100	5.0680	0.86910	**0.000 (H.S)**	-1.68100	-1.21900
NON-COPD	100	3.6180	0.78539		-1.68102	-1.21898

TABLE 6: COMPARISON OF REC AMONG COPD AND NON COPD SUBJECTS

GROUPS	N	MEAN REC (IN MM)	STANDARD DEVIATION	P VALUE	95% CONFIDENCE INTERVAL OF THE DIFFERENCE	
					LOWER	UPPER
COPD	100	1.8260	0.74191	**0.044 (S)**	-1.14784	-0.67416
NON-COPD	100	0.9159	0.94445		-1.14792	- 0.67408

TABLE 7: COMPARISON OF CAL AMONG COPD AND NON COPD SUBJECTS

GROUPS	N	MEAN CAL (IN MM)	STANDARD DEVIATION	P VALUE	95% CONFIDENCE INTERVAL OF THE DIFFERENCE	
					LOWER	UPPER
COPD	100	6.8940	1.06304	**0.002 (H.S)**	-2.71013	-2.01187
NON-COPD	100	4.5330	1.41572		-2.71029	-2.01171

TABLE 8: COMPARISON OF OHI-S SCORES AMONG COPD AND NON COPD SUBJECTS

GROUPS	N	MEAN OHIS SCORES	STANDARD DEVIATION	P VALUE	95% CONFIDENCE INTERVAL OF THE DIFFERENCE	
					LOWER	UPPER
COPD	100	2.4600	0.33212	0.059	-0.17943	0.00343
NON-COPD	100	2.3720	0.32352		-0.19943	0.00343

Discussion:

The study population consisted of 200 participants in the age range of 18 to 65 years. Hundred COPD and hundred Non-COPD participants were included with mean age of 47.31±1.45302 years and 48.74±15.04244 years respectively. A study was conducted by Ghani B and Bhattacharya HS where the age of controls and cases ranged from 40 to 65 years where mean was slightly higher in controls which are similar to our study.[3] A similar study was conducted by Deo V et al where the average age of patients with COPD was 41.43 years at the same time the mean age of controls (Non-COPD) was 43.62 years.[8] In contrast to the study conducted by Terashima T et al COPD patients were significantly older than the patients with other group.[12] In a study conducted by Peter K P et al the mean age of patient was more in case group 59.48±11.13years, whereas in the control group it was 49.69±10.16 years which is in contrast to our study.[13]

Out of 100 COPD participants 69% were males and 31% were females while out of 100 Non-COPD participants 59% were males and 41% were females. A study was conducted by Ghani B and Bhattacharya HS where frequencies of males were higher to females similar to our study.[3] Ledic K et al and Deo V et al conducted a similar study where maximum numbers of subjects were males among COPD participants.[14,8]

The mean FEV1/FVC ratio was 60.7035±8.24184 and 103.5950±14.55632 in COPD and Non-COPD participants respectively. In a study conducted by Kucukcoskun M et al the ratio among COPD participants was similar to our study (55.8).[15]

Maximum numbers of participants 43% were former smokers followed by 22% current smokers, 20% never smokers and 15% non smoker among COPD participants. In a study conducted by Ledic K et al maximum number of subjects were former smokers (62.4%) which are similar to our study.[14] In the research conducted by Leuckfeld I et al, COPD group consisted of 99% former smokers.[16] Reports have shown that smoking can be a residual confounding factor which compromises the phagocyte activity and the mucociliary barrier. Researchers have also shown that smokers have worst periodontal condition and they are at a greater risk of developing COPD.[3] Former smokers have almost 4 times higher risk of developing COPD. Many studies have confirmed the negative effect of smoking on increased probing depth, greater recession and significantly increased loss of attachment.[14] In the study conducted by Terashima T et al, proportions of former smokers was significantly higher in patients with COPD which is similar to our study.[12] Cigarette smoking is not the only leading dangerous factor for periodontal infection but also respiratory diseases like emphysema, chronic bronchitis, and lung infection results in the decline of pulmonary function. Smoking plays an important role in the

pathogenesis and progression of periodontal infections. Nicotine and tobacco smoke lowers the administration of oxygen and nourishment to the gingival tissue (Baab and Oberg, 1987),[17] and alters gingival inflammatory response (Bergstrom, 2004)[18] by causing the peripheral vaso-constrictive aftereffect. Cigarette smoking can also impair immunologic function. Cigarette smoking is known to hamper lung clearance straight by interrupting with coughing, compromising the defensive mucociliary action in the airways, and phagocyte activity (Page, 2001).[19] Here, there is more damage to the large airways and to the airway epithelium causing high levels of sputum production in patients, especially in the mornings (known as chronic bronchitis) (Terpenning, 2001).[20] The mechanism behind this increased risk is poorly understood, but it has been suggested that tobacco use depresses the fabrication of defensive IgG2 antibodies and blocks killing of bacteria and phagocytosis by neutrophils (Page, 1998).[21] Periodontal infection was significantly affiliated to the longitudinal deterioration of lung volumes and presence of COPD in current smokers compared to non-smokers, which raises the question whether the observations may actually be the result of residual confounding by smoking.[22,23,6] Since tobacco use can be expressed as a significant independent dangerous factor for both periodontitis and impaired lung function, residual confounding by smoking cannot be ruled out totally and exacerbates interpretation of results.

The mean PPD (in mm) among COPD and Non-COPD participants was 5.0680±0.86910 and 3.6180±0.78539. Study result showed statistically highly significant difference in the mean PPD which is more in COPD participants compared to Non-COPD participants (p=0.000, HS). A study conducted by Terashima T et al mean PD among Non-COPD participants was 3.0±0.9 which was similar to our study.[12] Several studies have recommended that patients with pockets and/or plaque accumulation, favors colonization of respiratory pathogen and make an individual more prone to the risk of developing a respiratory infection or disease. Subjects with periodontal infections and animated levels of proteolytic bacterias decline the non-specific host defence against respiratory pathogens.

The mean REC (in mm) among COPD and Non-COPD participants was 1.8260±0.74191 and 0.9159±0.94445. Study result showed statistically significant difference in the mean REC which is more in COPD participants compared to Non-COPD participants (p=0.044, S). Several mechanisms have been proposed for the association between these two highly common diseases. Respiratory pathogen colonization occurs in the dental plaque, which acts as a reservoir for these pathogens, and is shed into saliva. Pulmonary infection results because of the presence of organisms in the saliva that contaminates the distal portion of the respiratory tree. It is of great significance that the majority of pulmonary diseases are attributable to aerobic bacteria that are found in the oral flora in any oral diseases. On the contrary, some of the facultative anaerobes that are responsible for periodontal breakdown, such as Aggregatibacter actinomycetemcomitans, Fusobacterium nucleatum, Pseudomonas aeruginosa, and Porphyromonas gingivalis, also have been isolated from infected lungs. Furthermore, cytokines which are released by the infected tissue, during the progression of periodontal disease may alter respiratory epithelium. In COPD patient's release of proinflammatory cytokines like interleukin-8, from respiratory epithelium, leads to gross airway epithelial impairment. This subsequently results in release of toxic oxygen radicals and proteolytic enzymes after recruitment and infiltration by neutrophils. It is probable that the oral bacteria secreted in saliva comes in contact with respiratory epithelial surfaces and may adhere to the mucosal surface. Cytokine production is stimulated by these bound oral bacteria. It is again probable that the respiratory epithelial cells get stimulated when the distal respiratory epithelium may get contaminated by these originating cytokines from the oral tissues. Other cytokines are then released by these excited respiratory cells that may recruit inflammatory cells (neutrophils) to the site. The hydrolytic enzymes and other modifying molecules released by these inflammatory cells may result in damage of the epithelium, which may be more sensitive to colonization by respiratory pathogens. Thus, periodontal disease can probably contribute to the exacerbation of COPD.

The mean CAL (in mm) among COPD and Non-COPD participants was 6.8940±1.06304 and 4.5330±1.41572. Study result revealed statistically highly significant variation in the mean CAL which is more in COPD participants compared to Non-COPD participants (p=0.002, HS). The data of the present research recommend that chronic periodontitis as a risk factor for COPD as here PPD, REC and CAL was significantly greater in individuals with COPD as compared to Non-COPD. In a study conducted by Wang Z et al there wasn't any significant variation in mean CAL between COPD and Non-COPD.[9] The mean OHI-S (in mm) among COPD and Non-COPD participants was 2.46±0.33212 and 2.372±0.32352. Study result showed there was no statistical variation in the mean OHI-S among COPD participants when compared to Non-COPD participants (p>0.05, NS). In a study conducted by Peter K T et al (D7) mean OHI-S were significantly worst (p<0.0001) in case group compared to the control group which is similar to our study.[13] Chemical and mechanical plaque control leads to reduction and progression of COPD. There is a decrease in the levels of enzymes that degrade fibronectin when good oral hygiene practices are followed.[24] Aggravation and advancement of COPD depends on basic immigration of microbial pathogens to oral and/or pharyngeal surface. Moreover poor oral hygiene results in high supragingival plaque accumulation and higher concentrations of oral pathogens on both teeth and oral mucosa as well as in the saliva, being jointly responsible for periodontitis with pocketing.[25,26,27,28] 1 mm^3 of plaque, for instance, accommodate more than 10^6 bacteria with 300 different anaerobic and facultative anaerobic species.[29] Under specific conditions, dental plaque can harbour respiratory pathogens and promote their growth. Thus, oral cavity may be a considered as the potential reservoir for respiratory pathogens, which leads to aspiration pneumonia.[30,31,32,33] These pathogens that are shed into the salivary secretion may get contaminated and causes an alteration of respiratory epithelium. The alteration in the environment of the upper airway, caused by these bacteria, favors colonization for respiratory pathogens of the lower respiratory tract. However, even though these bacteria are, mandatory for respiratory infection, alone, they are inadequate to cause disease. Other factors such as the wide variety of confounding factors need to be taken into review as they are ramified.

Conclusion:

Within the limitation of the present research design which is a cross-sectional study design, known to hinder the establishment of a definite cause and effect relationship, the samples included in the present study were taken form only one institution which cannot generalize the whole population. The study should be organized on an array of populance to validate the noted affiliation and to rule their level of generalization. Since tobacco use can be treated as a significant independent dangerous factor for both periodontitis and impaired lung function, residual confounding by smoking cannot be ruled out totally and exacerbates interpretation of results recommended that the periodontitis - a systemic disease relationship should be investigated among healthy never smokers.

It can be concluded that periodontitis, oral bacteria and poor oral hygiene suggest influencing the incidence of COPD in high risk patients. In an adult population periodontal infection was significantly affiliated with reduced lung function and airflow limitation, independently of potential confounders. But, it cannot be argued that only poor oral health, alone, is responsible for respiratory diseases. It can be well said that poor oral health along with other factors (such as environmental pollutants, continued smoking, allergy, genetic factors, viral infections) to encourage the aggravation and advancement of COPD. However, large cohort researches with prolong investigation periods, assessing extensive lung function parameters are essential to certify the marked affiliation and to determine causality more closely. Further to this, it needs to be investigated, if prevention or treatment of periodontitis might have a beneficial impact on lung function, with the help of randomized clinical trials. It is essential to establish permanent collaboration between the pulmonologist and the dentist, and to explain the negative effects of periodontal disease and smoking in the course of COPD.

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