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Research Article

Research Article: Are COPD and Periodontal Diseases Associated with Each Other?

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Abstract

Objective: Inhaled corticosteroids can cause periodontal diseases, while periodontal diseases can lead to Chronic obstructive pulmonary disease (COPD) exacerbations. We aimed to investigate the correlation between oral and dental health and COPD exacerbation, and the frequency of periodontal diseases in COPD patients.

Material and methods: A prospective and cross-sectional study was performed involving 28 patients with COPD who had used inhaled corticosteroids for at least one year and 31 non-COPD patients. Dental complaints and dental care habits of the patients were questioned. Plaque index (PI), gingival index (GI), and probing pocket depth (PPD) measurements were performed for all available teeth.

Results: Total tooth loss was high in COPD patients (57.1%, p:0.001). There was no significant difference in exacerbation frequency between the edentulous and dentate subgroups in COPD patients (p: 0.702). There was no difference between the dentate COPD and control groups regarding periodontal disease development, gingival index, dental plaque index, last tooth cleaning, tooth brushing, caries, and probing pocket depth. While the use of inhaler technique (correct/wrong), frequency of exacerbations, frequency of tooth brushing, and presence of diabetes mellitus or reflux were not associated with periodontal disease in dentate COPD patients, no correlation was determined between probing pocket depth, presence of periodontal disease, gingival index, dental plaque index, frequency of tooth brushing and frequency of exacerbations. In the dentate COPD group, the frequency of exacerbations was lower in those with caries (p:0.037).

Conclusion: The correlation between COPD and periodontal disease and the increasing tooth loss in COPD need to be investigated with more extensive studies.

Keywords: COPD; periodontal diseases; inflammation; tooth loss

Running Title: COPD and Periodontal Diseases

Introduction

Chronic obstructive pulmonary disease (COPD) is associated with the chronic inflammatory response in the airways and lungs, characterized by persistent and often progressive airflow limitation. Some risk factors (such as age, smoking, obesity, socioeconomic status, and living conditions) are common in both periodontal diseases and COPD [1]. While drugs used in the treatment of COPD (especially inhaled steroids) may cause periodontal diseases, periodontal diseases may also lead to exacerbations of COPD by microaspiration and passing into the systemic circulation [2]. There are studies examining the correlation between COPD and chronic periodontitis and demonstrating improvement in forced expiratory volume in 1 s/forced vital capacity (FEV1/FVC) ratios through treating chronic periodontitis [3]. Inhaled corticosteroids (ICS), used in the treatment of COPD, pass from the alveoli to the systemic circulation and cause a decrease in BMD in the lumbar spine and femur [4, 5]. In the study of Komerik et al., a decrease in the bone mineral content of the mandible was also reported following inhaled corticosteroid treatment [6)]. Scannapieco et. al emphasized that patients with COPD may be at risk of developing periodontitis and thus teeth loss [7]. Mouth and nose bacteria have been detected in the lung tissue microbiota in COPD, suggesting that oral secretions may have been aspirated [8]. Oral secretions can be aspirated due to decreased laryngotracheal mechanical

sensitivity [9], decreased airway clearance, and impaired mucociliary functions [10] in COPD patients. Based on these data, we aimed to investigate whether there was a correlation between oral and dental health and COPD exacerbation, and the frequency of periodontal diseases in COPD patients.

Material and Methods

Twenty-eight male COPD patients who were followed up in the Chest Diseases Clinic and had used inhaled corticosteroids for at least one year and 31 non-COPD patients were included in the study. A prospective and cross-sectional study was performed. Female patients (for the possibility of being menopausal due to advanced age and taking hormone replacement therapy), patients with a disease that may affect bone metabolism (renal failure, malignancies), or patients using medications that may affect bone metabolism (oral corticosteroid, hormone replacement therapy, vitamin D, calcium) were excluded.

The patients' demographic information, the frequency of COPD exacerbations (requiring hospitalization or emergency admission), how they used inhaled steroids (deep breathing and mouth rinsing), comorbidities, dental complaints, and dental care habits were questioned. Plaque index (PI) [11], gingival index (GI) [12], and probing pocket depth (PPD) measurements were performed by the dentist to evaluate oral hygiene and periodontal status. Approval was sought from Institutional Ethics Committee (date: 07.12.2021, no:2021/280) and written informed consent was obtained from all participants enrolled in the study.

Statistics:

SPSS 20 statistical package program was used to analyze the data obtained from the study. Descriptive statistical methods (frequency, arithmetic mean, standard deviation, median, minimum, maximum, crosstabs) were used. For this study, a power analysis was not performed since periodontal examinations of male COPD patients who applied to the Chest Diseases Clinic for routine control would be performed, and the correlation between oral hygiene and dental care habits and the frequency of exacerbations would be examined. Compliance with normal distribution was evaluated with the Kolmogorov-Smirnov test. The Mann-Whitney U test compared two independent groups by analyzing the medians of the groups that did not show normal distribution. The Independent Sample t-test was used for two independent groups by comparing the arithmetic means of the normally distributed groups. The Chi-square test examined the relationship between categorical variables. The correlation between continuous variables was analyzed using the Pearson correlation coefficient. The statistical significance level was accepted as $p<0.05$.

Results

The mean age was 68.7±8 years for the COPD group and 66±8.5 for the control group (p: 0.202). There was no significant difference between the two groups regarding socioeconomic and education levels. 57.1% of the COPD group and 16.1% of the control group were edentulous (p: 0.001). Of 28 COPD patients, 16 had used an O2 concentrator and 7 bilevel positive airway pressure (BIPAP). 42.9% of COPD patients used inhalers with the wrong technique (not inspiring deeply, not rinsing the mouth) (Table 1).

There was no difference between the groups in terms of the presence of diabetes mellitus (DM), alcohol use, and gastroesophageal reflux. Non-smoker rate was 3.6% in COPD group and 45.2% in the control group (p:0.000). There was no difference in general oral health status, presence of bad breath, last dental examination, and dental scaling. Dryness of mouth was detected with a frequency of 67.9% in the COPD group (p: 0.025). The frequency of toothbrushing and using toothbrushes for cleaning were also significantly lower in the COPD group (p: 0.031, p: 0.000, respectively) (Table 1).

	COPD (n: 28)	Control (n: 31)	Total (n: 59)	p-value
Age	68.7 ± 8	66 ± 8.5	67.3 ± 8.3	0.202
Edentulous	16 (57.1%)	5 (16.1%)	21 (35.6%)	**0.001**
Dentate	12 (42.9%)	26 (83.9%)	38 (64.4%)	
Education				
Primary school	25 (89.3%)	20 (64.5%)	45 (76.3%)	0.085
Middle school	2 (7.1%)	3 (9.7%)	5 (8.5%)	
High School	1 (3.6%)	3 (9.7%)	4 (6.8%)	
University	0 (0%)	5 (16.1%)	5 (8.5%)	
SEL				
Low	0 (0%)	3 (9.7%)	3 (5.1%)	0.140
Moderate	27 (96.4%)	25 (80.6%)	52 (88.1%)	
Good	1 (3.6%)	3 (9.7%)	4 (6.8%)	
Inhaler technique				
Correct	16 (57.1%)			
Incorrect	12 (42.9%)			
With 02 concentrator	16 (57.1%)			
With BIPAP	7 (25%)			
DM	6 (21.4%)	6 (19.4%)	12 (20.3%)	0.843
Non-smoker	1 (3.6%)	14 (45.2%)	15(25.4%)	**0.000**
Ex-smoker	23 (82.1%)	10 (32.3%)	33 (55.9%)	
Smoker	4 (14.3%)	7 (22.6%)	11(18.6%)	
Alcohol use	15 (53.6)	10 (32.3%)	25 (42.4%)	0.098
Gastroesophageal reflux	8 (28.6%)	5 (16.1%)	13 (22%)	0.250

General oral health Good Bad	 6 (21.4%) 22 (78.6%)	 12 (38.7%) 19 (61.3%)	 18 (30.5%) 41 (69.5%)	 0.150
Dryness of mouth	19 (67.9%)	12 (38.7%)	31 (52.5%)	**0.025**
Removable denture	18 (64.3%)	16 (51.6%)	34 (57.6%)	0.325
Halitosis	4 (14.3%)	5 (16.1%)	9(15.3%)	0.844

SEL: socioeconomic level, DM: diabetes mellitus, BIPAP: bilevel positive airway pressure

Table 1. Comparison of demographic and dental findings of COPD and control groups

In dentate patients, there was no difference in periodontal disease, gingival index, dental plaque index, last tooth cleaning, tooth brushing frequency, caries, and probing pocket depth in both groups (Table 2).

	COPD (n: 12)	Control (n: 26)	Total (n: 38)	p-value
Periodontal dis.	7 (58.3%)	17 (65.4%)	24 (63.2%)	0.675
Gingival Index < 3 ≥ 3	 9 (75%) 3(25%)	 16 (61.5%) 10 (38.5%)	 25 (65.8%) 13 (34.2%)	 0.416
Dental plaque index < 3 ≥ 3	 9 (75%) 3(25%)	 14(53.8%) 12 (46.2%)	 23 (60.5%) 15 (39.5%)	 0.215
Latest teeth cleaning < 1 year ≥ 1 year	 0 (0%) 12 (100%)	 1 (3.8%) 25 (96.2%)	 1 (2,6%) 37 (97.4%)	 0.491
Brushing teeth ≥ 1 / day < 1 /day	 3 (25%) 9 (75%)	 13 (50%) 13(50%)	 16 (42.1%) 22 (57.9%)	 0.147
Caries	5 (41.7%)	11 (42.3%)	16 (42.1%)	0.970
Probing pocket depth (mm)	2.5 (0-3)	3 (1-8)	3 (0-8)	0.172
Gingival bleeding	2 (16.7%)	9 (34.6%)	1128.9%)	0.257
DM Yes No	 4 (0.33%) 8 (66.7%)	 6 (23.1%) 20 (76.9%)	 10 (26.3%) 28 (73.7%)	 0.505

Table 2. Comparison of dental examination results of Dentate COPD and control group

It was found that halitosis, total tooth loss, presence of reflux, and DM association did not affect the frequency of exacerbations in the entire COPD group (Table 3).

	Exacerbation frequency/year	p-value
Halitosis Yes (n: 4) No (n: 24)	 0.67 (0-8) 2.17(0-10)	 0.613
Dentate (n: 12) Edentulous (n: 16)	1.67 (0-8) 2 (0-10)	0.702
Gastroesophageal reflux Yes (n: 8) No (n: 20)	 3.67 (0-6) 0.85 (0-10)	 0.67
DM Yes (n: 6) No (n: 22)	 3.67 (1-8) 0.92 (0-10)	 0.057

Table 3. Evaluation of factors affecting exacerbation frequency in COPD patients (n: 28)

In the dentate COPD group, only the presence of diabetes mellitus (DM) among these factors increased the frequency of exacerbations, and the presence of caries was determined to decrease the frequency of exacerbations (Table 4).

	Exacerbation frequency/year	p-value
Probing pocket depth (mm)	*r: -0.042	0.832
Periodontal disease Yes (n: 7) No (n: 5)	 2.2 (0-8) 1 (0-5)	 0.243
Gingival Index < 3 (n: 9)	 1.33 (0-8)	 0.849

≥ 3 (n: 3)	2 (0-3)	
Dental plaque index < 3 (n: 9) ≥ 3 (n:3)	 1,33 (0-8) 2 (0-3)	0.849
Brushing teeth ≥ 1 / day (n: 3) < 1 /day (n: 9)	 2 (0-5) 1.4 (0-8)	0.849
Caries Yes (n: 5) No (n: 7)	 0.5 (0-2) 3 (0-8)	**0.037**
Gastroesophageal reflux Yes (n: 3) No (n: 9)	 3 (2-5) 0.83 (0-8)	0.154
Halitosis Yes (n: 3) No (n: 9)	 1 (0-8) 2 (0-5)	0.924
DM Yes (n: 4) No (n: 8)	 4.33 (3-8) 0.67 (0-3)	**0.009**

* r: Pearson correlation coefficient

Table 4. Factors affecting exacerbation frequency in dentate COPD patients (n: 12)

In the Dentate COPD group, no significant linear correlation was observed between probing pocket depth, presence of periodontal disease, gingival and dental plaque index, frequency of tooth brushing, and frequency of exacerbations. Furthermore, age, inhaler technique, frequency of exacerbations, frequency of tooth brushing, and presence of DM or gastroesophageal reflux were not associated with periodontal disease (Table 5).

	Periodontal disease	p-value
Age	65.4+-7.7	0.529
Inhaler technique Correct (n: 7) Incorrect (n: 5)	 4 (57.1%) 3 (60 %)	0.921
Exacerbation frequency / year	2.2 (0-8)	0.243
Brushing teeth ≥ 1 / day (n: 3) < 1/ day (n: 9)	 0 (0%) 7 (77.8%)	0.018*
DM Yes (n: 4) No (n: 8)	 3 (75%) 4 (50%)	0.408
Gastroesophageal reflux Yes (n: 3) No (n: 9)	 1 (33.3%) 6 (66.7%)	0.310

*: does not meet the assumptions of the chi-square test.

Table 5. Factors affecting the frequency of periodontal disease in Dentate COPD patients

Discussion

Total tooth loss in COPD patients was significantly higher than in the control group. This result can be explained by inhaled corticosteroids causing a decrease in mandibular bone mineral densitometry [6] or by the development of periodontitis in patients with COPD [7]. Chronic periodontitis is a common inflammatory disorder of the oral cavity and has been previously described in patients with COPD [13, 14]. Bacterial pockets form between the tooth and gingiva, which negatively affects the supporting tooth structures and leads to tooth loss. Our study was concluded by supporting studies [15] demonstrating that COPD patients have fewer teeth than controls. It has been suggested that loss of a tooth may prolong the bolus time in the oral cavity, vallecula, and hypopharynx by impairing chewing, bolus formation, and swallowing, and thus, it is predicted that COPD symptoms may increase with the aspiration of oral pathogens [16].

While studies have indicated that the number of remaining teeth is inversely proportional to the frequency of exacerbations [17], there are also studies suggesting the opposite [18]. The presence of diseased teeth may allow for extensive biofilm formation that can then be aspirated into the lungs, leading to a COPD exacerbation [19]. It was determined that full-mouth tooth extraction significantly reduces the periodontal pathogen burden [20]. Another study reported that those without COPD exacerbation had fewer or no teeth [21]. In our study, no significant difference was observed between the edentulous and dentate subgroups in terms of exacerbation frequency in COPD patients. This result indicates the need for further studies with a larger number of patients.

Common risk factors for periodontal disease and systemic diseases include smoking, stress, age, race, and gender [22]. In our study, no significant correlation was determined between age and the development of periodontal disease in the dentate COPD group. Since we included only male patients in the study, the gender difference was not mentioned. While the smoking rate was significantly higher in the COPD group, there was no difference between the COPD group and the control group regarding periodontal disease. Periodontal health indices in COPD patients with frequent exacerbations were better than those in the rare exacerbation group, although their differences were not statistically significant [18]. This result may be due to the frequent use of antibiotics in COPD exacerbations and reducing periodontal inflammation. On the contrary, there are studies [23] showing a higher frequency of periodontal disease in COPD or studies showing no correlation [21]. In our study, however, no significant difference was

observed between the dentate COPD and control groups in terms of the development of periodontal disease. In dentate COPD patients, the use of inhaler technique (correct/incorrect), frequency of attacks, frequency of tooth brushing, and presence of DM or reflux were not associated with periodontal disease.

Pathogenic microorganisms in dental plaque play a critical role in infectious and/or inflammatory processes. The results of plaque index measurements have been variable. Some studies have found it higher in COPD patients [15], while others have not shown a difference (24). Our study revealed no difference between the dentate COPD and control groups regarding the gingival index, dental plaque index, last tooth cleaning, tooth brushing, caries, and probing pocket depth. Some publications find probing pocket depth, gingival index, plaque index [25], and short tooth brushing durations [18] correlated with COPD exacerbation. In our study, no correlation was found in the dentate COPD group between probing pocket depth, presence of periodontal disease, gingival index, dental plaque index, frequency of tooth brushing, and frequency of exacerbations. In the dentate COPD group, the frequency of exacerbations was lower in those with decayed teeth. This result suggested that antibiotic therapy used for carious teeth may also effectively reduce the frequency of COPD exacerbations.

Diabetes mellitus increases the risk of periodontal disease [26]. In our study, when COPD and control groups were compared, although the presence of DM was not associated with the formation of periodontal disease, there was a significant increase in the number of exacerbations in the presence of DM in the dentate COPD group. This result may be due to the direct effects of diabetes on lung physiology, inflammation, or susceptibility to bacterial infection [27].

Incorrect inhaler use rates of up to 94% have been reported in COPD patients. In our study, this rate was 42.9%. In particular, we questioned not making deep inspiration and mouth rinsing after inhalers, which increases tooth and gingival exposure by causing ICS to remain in the mouth. Our study determined no correlation between incorrect inhaler technique and periodontal disease in dentate COPD patients.

In a prospective observational study, Gaeckle et al. demonstrated that people with COPD have poor dental hygiene practices, worse dental indices, and significantly worse oral health-related quality than healthy controls [28]. However, in our study, no significant difference was observed in the dentate COPD group in terms of tooth brushing habits and last tooth cleaning compared to the control group. Dryness of mouth complaints were significantly higher in the COPD group (67.9% / 38.7%, p:0.025). In a meta-analysis, the cumulative incidence of dryness of mouth in COPD patients was 7.4% with tiotropium, 3.9% with ipratropium, 1.6% with salmeterol, and 2.0% with placebo. We also evaluated dryness of the mouth as a side effect of drugs or secondary to the presence of DM.

The limitations of our study are the small number of dentate COPD patients and being a single-center study. However, the results obtained emphasize that periodontal diseases should not be overlooked in COPD patients.

Conclusion

There is need for more extensive studies on the correlation between periodontal disease and respiratory tract and increased tooth loss in COPD patients. Oral and dental health should be given due importance to reduce or prevent COPD exacerbations.

Conflicts of interest

The authors have no conflict of interest.

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Abbreviations

COPD: Chronic obstructive pulmonary disease
BMD: bone mineral densitometry
PI: Plaque index
GI: gingival index
PPD: probing pocket depth
FEV1/FVC: forced expiratory volume in 1 s/forced vital capacity
BIPAP: bilevel positive airway pressure
ICS: Inhaled corticosteroids

References

1. Cullinan MP, Ford PJ, Seymour GJ. Periodontal disease and systemic health: current status. Aust Dent J. 200954 Suppl 1: S62–69
2. Gomes-Filho IS, Passos JS, Seixas da Cruz S. Respiratory disease and the role of oral bacteria. J Oral Microbiol. 2010 Dec 21;2.
3. Sharma S, Gupta A, Verma AK, et al. Impact of Non-surgical Periodontal Therapy on Pulmonary functions, Periodontal Health and Salivary Matrix Metalloproteinase-8 of COPD Patients with Chronic Periodontitis: A Clinico-biochemical Study. Turk Thorac J. 2021 Jul;22(4):324-332.
4. Hanania NA, Chapman KR, Sturtridge WC, et al. Dose-related decrease in bone density among asthmatic patients treated with inhaled corticosteroids. J Allergy Clin Immunol. 1995 Nov;96(5 Pt 1):571-579.
5. Sivri A, Cöplü L. Effect of the long-term use of inhaled corticosteroids on bone mineral density in asthmatic women. Respirology. 2001 Jun;6(2):131-134.
6. Komerik N, Akkaya A, Yildiz M, et al. Oral health in patients on inhaled corticosteroid treatment. Oral Dis. 2005 Sep;11(5):303-308.
7. Scannapieco FA, Ho AW. Potential associations between chronic respiratory disease and periodontal disease: analysis of National Health and Nutrition Examination Survey III. J Periodontol. 2001 Jan;72(1):50-56.
8. Pragman AA, Lyu T, Baller JA, et al. The lung tissue microbiota of mild and moderate chronic obstructive pulmonary disease. Microbiome. 2018 Jan 9;6(1):7.
9. Clayton NA, Carnaby-Mann GD, Peters MJ, et al. The effect of chronic obstructive pulmonary disease on laryngopharyngeal sensitivity. Ear Nose Throat J. 2012 Sep;91(9):370, 372, 374 passim.
10. Smaldone GC, Foster WM, O'Riordan TG, et al. Regional impairment of mucociliary clearance in chronic obstructive pulmonary disease. Chest. 1993 May;103(5):1390-1396.
11. Sılness J, Loe H. Perıodontal Dısease In Pregnancy. II. Correlatıon Between Oral Hygıene And Perıodontal Condtıon. Acta Odontol Scand. 1964 Feb;22:121-135.
12. Loe H, Sılness J. Perıodontal Dısease In Pregnancy. I. Prevalence And Severity. Acta Odontol Scand. 1963 Dec;21:533-551.
13. Costalonga M, Herzberg MC. The oral microbiome and the immunobiology of periodontal disease and caries. Immunol Lett. 2014 Dec;162(2 Pt A):22-38.
14. Eke PI, Dye BA, Wei L, et al; CDC Periodontal Disease Surveillance workgroup: James Beck (University of North

Carolina, Chapel Hill, USA), Gordon Douglass (Past President, American Academy of Periodontology), Roy Page (University of Washin. Prevalence of periodontitis in adults in the United States: 2009 and 2010. J Dent Res. 2012 Oct;91(10):914-920.

15. Ledić K, Marinković S, Puhar I, et al. Periodontal disease increases risk for chronic obstructive pulmonary disease. Coll Antropol. 2013 Sep;37(3):937-942.
16. Cunningham TJ, Eke PI, Ford ES, et al. Cigarette Smoking, Tooth Loss, and Chronic Obstructive Pulmonary Disease: Findings From the Behavioral Risk Factor Surveillance System. J Periodontol. 2016 Apr;87(4):385-394.
17. Wang Z, Zhou X, Zhang J, et al. Periodontal health, oral health behaviours, and chronic obstructive pulmonary disease. J Clin Periodontol. 2009 Sep;36(9):750-755..
18. Liu Z, Zhang W, Zhang J, et al. Oral hygiene, periodontal health and chronic obstructive pulmonary disease exacerbations. J Clin Periodontol. 2012 Jan; 39(1):45-52.
19. Marsh PD, Moter A, Devine DA. Dental plaque biofilms: communities, conflict and control. Periodontol 2000. 2011 Feb;55(1):16-35.
20. Barbosa VL, Angst PD, Finger Stadler A, et al. Clinical attachment loss: estimation by direct and indirect methods. Int Dent J. 2016 Jun; 66(3):144-149.
21. Baldomero AK, Siddiqui M, Lo CY, et al. The relationship between oral health and COPD exacerbations. Int J Chron Obstruct Pulmon Dis. 2019 Apr 23;14:881-892.
22. Page RC. The pathobiology of periodontal diseases may affect systemic diseases: inversion of a paradigm. Ann Periodontol. 1998 Jul;3(1):108-120.
23. Lopez-de-Andrés A, Vazquez-Vazquez L, Martinez-Huedo MA, et al. Is COPD associated with periodontal disease? A population-based study in Spain. Int J Chron Obstruct Pulmon Dis. 2018 Oct 18;13:3435-3445.
24. Katancik JA, Kritchevsky S, Weyant RJ, et al. Periodontitis and airway obstruction. J Periodontol. 2005 Nov;76(11 Suppl):2161-2167.
25. Peter KP, Mute BR, Doiphode SS, et al. Association between periodontal disease and chronic obstructive pulmonary disease: a reality or just a dogma? J Periodontol. 2013 Dec;84(12):1717-1723.
26. Mealey BL, Oates TW. American Academy of Periodontology. Diabetes mellitus and periodontal diseases. J Periodontol. 2006 Aug; 77(8):1289-1303.
27. Gläser S, Krüger S, Merkel M, et al. Chronic obstructive pulmonary disease and diabetes mellitus: a systematic review of the literature. Respiration. 2015; 89(3):253-264.
28. Gaeckle NT, Heyman B, Criner AJ, et al. Markers of Dental Health Correlate with Daily Respiratory Symptoms in COPD. Chronic Obstr Pulm Dis. 2018 Apr 1; 5(2):97-105.