Examination of Periodontal Status in Chronic Obstructive Pulmonary Disease Greek Adults: A Case – Control Study

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Abstract

Aim: The aim of the current research was to assess the possible differences regarding the periodontal status between individuals suffering from chronic obstructive pulmonary disease (COPD) compared with healthy individuals.

Materials and Methods: 453 individuals with COPD and 997 matching healthy controls were interviewed and oral clinically examined. The indices used to determine the periodontal status for cases and controls included Probing Pocket Depth (PPD), Clinical Attachment Loss (CAL) and Bleeding on Probing (BOP). Chi-square test and logistic regression models were carried out to assess the data.

Results: COPD patients showed worst periodontal parameters among some of the indices examined such as PPD (p = 0.001), tooth-brushing frequency (p = 0.045) and dental check-up (p = 0.048), after adjustment for smoking and socio-economic status.

Conclusions: Probing Pocket Depth and oral hygiene parameters such as tooth-brushing frequency and dental check-up were statistically significant different between individuals who were suffered from COPD and healthy ones.

Keywords: Chronic obstructive pulmonary disease; Periodontal disease; Adults; Risk factors

1. INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is one of the most common respiratory diseases the 4th leading cause of death in the world, and it is predicted that by 2020 will be the 3rd leading cause of death and the 5th leading cause of overall disability worldwide [1]. COPD is a heterogeneous respiratory disease that includes clinical conditions such as emphysema, chronic bronchitis, small airways disease, and non-reversible asthma [2]. Its prevalence in Europe and North America and is 9% to 10% in adults aged 40 years or older, its increased prevalence has been attributed to increased smoking rates, whereas COPD is impaired by exacerbations possibly due to viral or bacterial infections [3].

COPD’s etiology implicates genetic, behavioral and environmental risk factors [4]. The main genetic risk factor is a severe hereditary deficiency of α1-antitrypsin, a circulating serine proteases inhibitor, whereas other possible genetic factors that are involved in COPD pathogenesis have not been yet determined [5, 6]. It is possible that various genes and an interaction between genes and environment are involved in COPD pathogenesis [7]. The environmental and behavioral risk factors which contribute to COPD development are exposure to cigarette and passive cigarette smoke [8, 9], occupational chemicals, such as organic and inorganic chemical fumes and agents [10], indoor and outdoor air and atmospheric pollution, despite the fact that the role of outdoor air pollution in COPD pathogenesis remains unclear [11, 12]. An interesting observation is that not all cigarette smokers develop clinically significant COPD, suggesting that genetic factors may modify each individual’s risk [13]. Moreover, it remains unclear why individuals-never smokers develop...
COPD [14]. Socio-economic status (SES) is involved in COPD pathogenesis as it has been observed that the onset of COPD is inversely associated with SES [15] however, it is unclear whether SES is associated with the mentioned pollutants or other unknown factors that are associated with low SES [16]. As already mentioned COPD is a heterogeneous disease that is characterized by progressive airflow obstruction and chronic airways inflammation, whereas the abnormal inflammatory response of the lung leads to airflow limitation [17]. The chronic airways inflammation is accompanied by increased levels of specific inflammatory cells at different locations of the lung, whereas, it has been detected lung tissue remodeling as a result of the repeated processes of lung tissues injury and repair [18].

Periodontal Disease [PD] and especially periodontitis is a progressive inflammation, which leads to destruction of periodontal tissue. PD is highly prevalent and may affect up to 90% of the world population [19]. Chronic periodontitis is characterized by an increased plasma concentration of inflammatory cytokines and chemokines, such as C reactive protein [Crp], Interleukin [IL]-1 and -6, that are associated with PD severity. The possible role of those inflammatory biomarkers in several systemic diseases development could be attributed to a general inflammatory reaction, a systemic immune reaction to periodontal pathogens or the entry of periodontal pathogens into the blood circulation [20, 21]. Thus, many reports have investigated a possible role of periodontitis as a risk factor for systemic diseases or disorders [22], including COPD [23-37]. A possible link between PD and COPD has been suggested, based on the fact that chronic periodontitis is associated with increased concentration of inflammatory mediators and because of evidence involving chronic inflammation in COPD pathogenesis. Moreover, oral pathogens and inflammatory cytokines and chemokine from damaged periodontal tissue induce a systemic inflammation, which may contribute to COPD pathogenesis [38-40].

COPD and periodontitis have in common pathogenic pathways such as a chronic course with progressive and irreversible tissue destruction caused by neutrophilic inflammation with subsequent proteolytic destruction of connective tissues, gradual loss of normal organ function and share known risk factors such as age, tobacco smoking, SES, and living conditions [38, 39]. It is obvious that an association exists between both pathological conditions as periodontitis appears to be a risk factor for COPD development and periodontal pathogens may play a key role in COPD pathogenesis. In contrast to the mentioned researches, few surveys have investigated whether the periodontal status of individuals who suffer from COPD is worse than that of the non-COPD individuals, where as their findings were inconsistent, some even contradictory [41, 42]. The reasons were that those studies used different population samples and PD indices. The aim of the current report was to assess the possible differences in periodontal condition between individuals who suffered from COPD diagnosed by clinical examinations and healthy ones.

2. Material and Methods
2.1. Study Sample
From March 2019 to October 2020, a case-control study was carried out in which a total of 453 individuals who suffered from COPD, 296 males and 157 females, and 997 controls with normal pulmonary function,409 males and 588 females, aged 35-78 years, were recruited from 3 private practices. All COPD cases were clinically diagnosed and confirmed by lung function examination for more than three years. Criteria used for the diagnosis of COPD based on the Global Initiative for Chronic Obstructive Lung Disease [GOLD] spirometry guidelines. Lung function was examined and assessed using spirometry. The lung function measurements were assessed by the physicians of the patients and recorded on their medical files. During at least 5 forced expirations, the physician attempted to obtain 3 acceptable spiromgrams, at least two of which showed similar results for the Forced Expiratory Volume [FEV]1/1sec [FEV1] and Forced Vital Capacity (FVC). Air limitation was set using the fixed ratiopost-bronchodilatorFEV1/FVC<0.70 [4].COPD patients and controls should meet the following selection criteria: no having fever, worsening of respiratory symptoms, acute pulmonary diseases or medication change within the last month, no having history of lung volume reduction surgery, pneumonectomy, or lung transplantation, no having history of any periodontal treatment in the last 6 months, had not received systemic antibiotic treatment or a systematic treatment with glucocorticoids or immunosuppression agents within the previous
6 months [43], aged 35 years and up with ≥ 15 teeth and periodontitis from stage I to IV [44]. They also should not be suffered from diseases such as cardiovascular disease, diabetes mellitus, rheumatoid arthritis, or any type of malignancies. Those pathological conditions could have potential effects on the periodontal tissues as confounders and could lead to biased secondary associations.

Controlling for the potential confounding effect of smoking and SES, based on the control selection which carried out by the friendly and collegial environment of healthy individuals, and both groups were matched for gender and age. Moreover, cases and controls were selected from the same city population in an effort to select a representative study sample and to avoid or eliminate possible selection biases. Therefore, control group was selected from individuals who were presented to routine health follow-up at the mentioned practices, between 2019 and 2020.

2.2. Dental and Oral Clinical Examination

Cases and controls underwent an oral and dental clinical examination by a well-trained and calibrated dentist regarding the mentioned periodontitis indices PPD, CAL and BOP, and responded to a medical and dental health questionnaire. All indices examined were measured with a millimeter graduated probe [Hu-Friedy PCP 10-SE] at four sites per tooth [mesio-buccal, disto-buccal, mesio-lingual, and disto-lingual] in all quadrants and the worst values of the indices recorded to the nearest 1.0 mm. Remaining roots and 3rd molars were excluded.

The criteria for assessing periodontitis indices, PPD and CAL, were based on the new classification as already mentioned, whereas BOP, was recorded as present or not within 30 seconds following probing with gentle pressure at four sites per tooth [45]. PPD index was dichotomously assessed as code 0: stage I [maximum PPD ≤ 4.0 mm] and code 1: stage II-IV [PPD ≤ 4.0 - ≥ 6.0 mm]. Similarly CAL severity, was assessed as code 0: stage I [CAL: 1.0-2.0 mm], and code 1: stage II-IV [CAL: 3.00 - ≥ 5.0 mm] [44]. BOP absence was assessed as code 0 and presence as code 1, respectively [45]. The same dentist re-examined a random sample of 300 [-20.0%] cases and controls, after a period of 3 weeks for assessing the intra-examiner reliability and no differences were found between both oral clinical examinations [Cohen's Kappa=0.94]] whereas during that time period no oral hygiene instructions were given to the participants.

2.3. Ethical Consideration

The current study was not an experimental study and should not reviewed and approved by authorized committees [Ministry of Health, Dental Associations, etc.] in Greece. The individuals who agreed to participate in the present study signed an informed consent form.

2.4. Statistical Analysis

The worst values of PPD and CAL at four sites per tooth for cases and controls were assessed. Females, non-smokers, low socioeconomic status [income/monthly equivalent to or less than 1,000 €], low educational level [graduated from Elementary/High School], irregular daily tooth-brushing daily [≤1/daily] and annual dental check-up [≤1/year], and controls were coded as 0. Age groups distribution was coded as 0, 1, 2, and 3 for ages 35-49, 50-59, 60-69 and 70+, respectively. The univariate analysis model was carried out to estimate the associations between the independent parameters examined and cases/controls, separately. Multivariate logistic regression analysis was performed to assess the associations between the dependent variable, COPD, and independent ones using the Enter and Stepwise methods. Unadjusted and Adjusted Odds Ratios [OR's] and 95% [Confidence Interval] CI were also estimated. The statistical method Cohran’s and Mantel-Haenszel’s was applied to control possible confounders, such as smoking. Smoking analysis was performed by SPSS statistical package [SPSS PC19.0, SPSS, Inc., Chicago, IL, USA], and a p value less than 5% [p< 0.05] was considered to be statistically significant.

3. RESULTS

Chi-squared test was used to compare characteristics between cases and controls for categorical variables. The results of univariate analysis are presented in Table1, in which low SES, smoking, irregular daily tooth-brushing and annual dental check-up, and deep periodontal pockets were found to be statistically significant different between cases and controls. The same table shows unadjusted OR’s and 95% CI. According to regression model, the 1a step showed that smoking (p=0.000) and deep periodontal pockets (p=0.006) were significantly different between COPD patients and controls, whereas irregular annual dental check-up was marginally significantly
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The examination of periodontal status in chronic obstructive pulmonary disease (COPD) Greek adults showed that different indices were statistically significant different between COPD patients and healthy individuals. PPD was also statistically significant different between cases and controls after adjusting for smoking and SES (Table 3).

Table 1. Univariate analysis of cases and controls regarding each independent variable examined

<table>
<thead>
<tr>
<th>Variables</th>
<th>Cases (no) (%)</th>
<th>Controls (no) (%)</th>
<th>p-value</th>
<th>Odds Ratio (OR)</th>
<th>95% Confidence Interval (CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender: Males</td>
<td>296 (65.3)</td>
<td>409 (41.0)</td>
<td>0.000*</td>
<td>0.37</td>
<td>0.29-0.47</td>
</tr>
<tr>
<td>Females</td>
<td>157 (34.7)</td>
<td>588 (59.0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years): 35-49</td>
<td>45 (9.9)</td>
<td>118 (11.8)</td>
<td>0.661</td>
<td></td>
<td></td>
</tr>
<tr>
<td>55-59</td>
<td>198 (43.7)</td>
<td>409 (41.0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>60-69</td>
<td>152 (33.6)</td>
<td>338 (33.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>70+</td>
<td>58 (12.8)</td>
<td>132 (13.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Socio-economic level: Low</td>
<td>321 (70.9)</td>
<td>506 (50.8)</td>
<td>0.000*</td>
<td>2.36</td>
<td>1.86-3.00</td>
</tr>
<tr>
<td>High</td>
<td>132 (29.1)</td>
<td>491 (49.2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Educational level: Low</td>
<td>260 (57.4)</td>
<td>568 (57.0)</td>
<td>0.880</td>
<td>1.02</td>
<td>0.81-1.27</td>
</tr>
<tr>
<td>High</td>
<td>193 (42.6)</td>
<td>429 (43.0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking: No</td>
<td>91 (20.1)</td>
<td>519 (52.1)</td>
<td>0.000*</td>
<td>0.23</td>
<td>0.18-0.30</td>
</tr>
<tr>
<td>Yes</td>
<td>362 (79.9)</td>
<td>478 (47.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tooth-brush frequency: ≤1/daily</td>
<td>287 (63.4)</td>
<td>488 (48.9)</td>
<td>0.000*</td>
<td>1.80</td>
<td>1.44-1.27</td>
</tr>
<tr>
<td>≥2/daily</td>
<td>166 (36.6)</td>
<td>509 (51.1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dental follow-up: ≤1/annually</td>
<td>306 (67.5)</td>
<td>465 (46.6)</td>
<td>0.000*</td>
<td>2.38</td>
<td>1.89-3.00</td>
</tr>
<tr>
<td>≥2/annually</td>
<td>147 (32.5)</td>
<td>532 (53.4)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Periodontal pockets: ≤4.0 mm</td>
<td>65 (14.3)</td>
<td>381 (38.2)</td>
<td>0.000*</td>
<td>0.27</td>
<td>0.20-0.36</td>
</tr>
<tr>
<td>Depth ≤ 4.0 mm</td>
<td>388 (85.7)</td>
<td>616 (61.8)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAL: 1-2.0 mm</td>
<td>115 (25.4)</td>
<td>301 (30.2)</td>
<td>0.061</td>
<td>0.78</td>
<td>0.61-1.01</td>
</tr>
<tr>
<td>3.00 - ≥ 5.0 mm</td>
<td>338 (74.6)</td>
<td>696 (69.8)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BOP: No</td>
<td>173 (38.2)</td>
<td>364 (36.5)</td>
<td>0.539</td>
<td>1.07</td>
<td>0.85-1.35</td>
</tr>
<tr>
<td>Yes</td>
<td>280 (61.8)</td>
<td>633 (63.5)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p-value : statistically significant

Table 2. Presentation of association between parameters examined and COPD according to Enter (first step-1*) and Wald (final step-6a) methods of Multivariate Logistic Regression Analysis Model

<table>
<thead>
<tr>
<th>Variables in the Equation</th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>df</th>
<th>Sig.</th>
<th>Exp(B)</th>
<th>95% C.I. for EXP (B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>gender</td>
<td>.629</td>
<td>.152</td>
<td>3.224</td>
<td>1</td>
<td>.080</td>
<td>1.876</td>
<td>1.394, 2.525</td>
</tr>
<tr>
<td>socioecon.stat</td>
<td>.271</td>
<td>.171</td>
<td>2.630</td>
<td>1</td>
<td>.105</td>
<td>.758</td>
<td>.543, 1.059</td>
</tr>
<tr>
<td>educat.level</td>
<td>.189</td>
<td>.171</td>
<td>1.231</td>
<td>1</td>
<td>.267</td>
<td>1.208</td>
<td>.865, 1.688</td>
</tr>
<tr>
<td>toothbr.freq</td>
<td>.468</td>
<td>.133</td>
<td>4.398</td>
<td>1</td>
<td>.062</td>
<td>1.026</td>
<td>.464, 1.845</td>
</tr>
<tr>
<td>dent.checkup</td>
<td>.690</td>
<td>.149</td>
<td>7.430</td>
<td>1</td>
<td>.054</td>
<td>1.994</td>
<td>1.489, 2.670</td>
</tr>
<tr>
<td>prob.pock.depth</td>
<td>1.615</td>
<td>.214</td>
<td>9.152</td>
<td>1</td>
<td>.006*</td>
<td>3.026</td>
<td>1.802, 6.651</td>
</tr>
<tr>
<td>clin.attach.loss</td>
<td>.179</td>
<td>.235</td>
<td>.578</td>
<td>1</td>
<td>.447</td>
<td>.836</td>
<td>.528, 1.326</td>
</tr>
<tr>
<td>bleed.on.prob</td>
<td>.281</td>
<td>.209</td>
<td>1.805</td>
<td>1</td>
<td>.179</td>
<td>1.325</td>
<td>.879, 1.996</td>
</tr>
<tr>
<td>Constant</td>
<td>3.996</td>
<td>.270</td>
<td>18.580</td>
<td>1</td>
<td>.000</td>
<td>.018</td>
<td>.018</td>
</tr>
</tbody>
</table>

* Variable(s) entered on step 1: gender, socioeconomic status, educat.level, smoking status, tooth br.freq, dent.checkup, prob.pock.depth, clin.attach.loss, bleed.on.prob

* p-value : statistically significant
Table 3. Presentation of Association between Independent Variables and COPD According to Wald (final step-6) Method of Multivariate Logistic Regression Analysis Model

<table>
<thead>
<tr>
<th>Variables</th>
<th>Exp (B)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Probing pocket depth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non - smokers</td>
<td>2.156</td>
<td>1.533-3.770</td>
</tr>
<tr>
<td>Socioeconomic status: Low</td>
<td>1.385</td>
<td>0.410-1.829</td>
</tr>
<tr>
<td>High</td>
<td>1.106</td>
<td>0.364-1.617</td>
</tr>
<tr>
<td>Daily tooth-brushing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non - smokers</td>
<td>1.088</td>
<td>0.223-1.135</td>
</tr>
<tr>
<td>Smokers</td>
<td>1.117</td>
<td>0.504-1.231</td>
</tr>
<tr>
<td>Annual dental check-up</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non - smokers</td>
<td>1.658</td>
<td>0.778-1.871</td>
</tr>
<tr>
<td>Smokers</td>
<td>1.788</td>
<td>0.823-1.998</td>
</tr>
<tr>
<td>Socioeconomic status: Low</td>
<td>0.849</td>
<td>0.328-1.047</td>
</tr>
<tr>
<td>High</td>
<td>0.772</td>
<td>0.357-1.008</td>
</tr>
</tbody>
</table>

Previous similar studies have confirmed the mentioned significant difference [27, 29, 35, 48-51]. A meta-analysis by Shi et al. [48], analyzed the data of PPD in the COPD and non-COPD group of 8 studies [31, 33, 41, 43, 48, 49, 52-54]. The results showed that probing depth in the COPD patients was deeper than that of the non-COPD group, and the mean difference was 0.261 mm (95% CI: 0.020–0.501, p = 0.033). Because the heterogeneity between the studies
was high (I² = 93.8%), a random effects model was selected. However, the sensitivity analysis revealed that the results were not stable. Despite the last observation the main finding was that periodontitis condition in the COPD group was more severe than that in the non-COPD group.

In contrary, in a similar report Scannapieco et al. [37] recorded that patients with a history of COPD did not show more PPD index than subjects without COPD.

CAL is another index for assessing the severity of periodontitis [47] and also refers to the long term stages of chronic inflammation including destructive processes signs of a chronic inflammatory response. The present report showed no significant difference between cases and controls regarding that index.

The same meta-analysis [48] recorded that the COPD patients suffered from worse periodontal health status, indicated by high level of CAL. Similar findings were confirmed by other researches [35, 50, 51, 55]. Ban et al. [56] were found that oral health of the cases group was worse than that of the controls and in cases group the mean and maximal CAL, were higher. However, it was not clear whether the COPD-associated systemic inflammation impaired the oral status or the chronic periodontitis influenced negatively chronic obstructive pulmonary disease.

Shi et al. [48], also assessed the data of CAL in COPD and non-COPD individuals from 10 studies [29, 31, 33, 37, 41, 49, 52-54, 57], and found that the COPD patients suffered more CAL compared with the non-COPD subjects. The mean difference was found to be 0.480 mm (95% CI: 0.280–0.681, random effect model, I² = 89.7%), which was of statistical significance (p= 0.000), whereas the Egger’s test showed that no publication bias was detected (p= 0.979). Therefore, the sensitivity analysis showed that the pooled results were powerful.

BOP index reflects the host’s vascular response in terms of hyperemia, the capillaries’ dilation and increased blood flow in the inflammation region. BOP is a widely used criterion to diagnose gingival inflammation, however it has been suggested that periodontal pockets with a probing depth of greater than or equal to 5.00 mm showed a significantly higher incidence of BOP [58]. In the current study no significant difference was observed between cases and controls regarding that index. Similarly, Scannapieco et al. [37] found no association between gingival bleeding and chronic respiratory disease. In contrary previous studies revealed that COPD patients suffer from worse periodontal health status, indicated by more inflammation and bleeding in the gingival tissue [48, 56]. Deo et al. [29] found that the percentage of individuals with <20% bleeding sites was significantly greater in non- COPD individuals (12% of non- COPD individuals and 4% of COPD patients). In a meta-analysis [48] which was assessed BOP [41, 54] and Gingival Bleeding Index (GBI), in the COPD and non-COPD groups, contrary results were revealed by comprehensive analysis. For the GBI, the mean difference was 0.241 with no statistical significance (95% CI: 0.106 to 0.588, p=0.173, random effect model, I² = 95.9%), whereas the results of BOP revealed that more bleeding was observed in the COPD (mean difference = 6.878, 95% CI: 5.489– 8.266, p= 0.000, fixed effect model, I² = 0%).

It is possible that these different results may be caused by the limited number of studies which use these three indices, which means that more well-designed studies are needed in the future. At last the number of remaining teeth in both groups, or other PD indices, such as PI, ABL could be used for assessing the periodontal status groups examined.

The outcomes of the current study also showed that oral hygiene habits were significantly different between cases and controls and that COPD-individuals had worse oral hygiene. Such outcomes have confirmed from previous similar studies [31, 35, 48, 49, 54, 59].

Only in a study by Parashar et al. [50], was revealed that the frequency of brushing did not show much difference among both groups.

In Europe and North America the prevalence of COPD is 9-10% in adults aged 40 years or older, observation that has been attributed to increased smoking rates [3]. Despite the fact that older individuals are at higher risk for COPD development [24, 60-67], age is considered as a confounder. Similarly, older individuals are at higher risk for initiation and progression of PD [68]. The difference regarding the parameter of age between COPD individuals and non-COPD ones was not statistically significant in the current report.

COPD, nowadays, mainly affects females because smoking prevalence has increased.
among females and may be more susceptible to the adverse pulmonary function effects of smoking than males. Another reason is that unbiased measures of lung function are underused could lead to under and misdiagnosis of COPD in both genders [69-73]. Females may have a greater predisposition to COPD [74], as the prevalence of smoking in females has increased progressively [75]. Moreover, a growing health burden of COPD among females [76] and non-smokers [77] has been suggested over the last few years, because of their exposure to biomass fuels which could lead to COPD development in younger females [77, 78]. In the current study no statistically significant difference was recorded between cases and controls regarding the gender, which is also considered as a confounder.

SES is a risk factor for COPD development [24, 38, 79-87], however it remains unclear, whether it reflects exposures to cigarette smoke, indoor and outdoor air pollutants, crowding, poor nutrition, or other factors that are related to low SES [16]. COPD patients, have higher age, less income, lower educational level, consumed more tobacco and alcohol, irrespective of whether they suffered from PD or not [88]. The disease is unevenly distributed in the population, as lowest SES individuals who suffer from COPD are more likely to experience poor health outcomes than those of the highest SES COPD patients [87]. The current report showed no statistically difference between COPD and non-COPD individuals regarding SES, which is another crucial confounder.

COPD-patients have lower education level than non-COPD individuals [79, 83, 88, 89]. Moreover, it has also been proposed that high-educated individuals take care of their own oral hygiene more than low-educated ones and could prevent diseases that are associated with PD [90]. No significant difference was recorded between COPD and non-COPD individuals regarding the educational level in the present research. Cigarette smoking and passive cigarette smoking is a risk factor for COPD development [8, 9], despite the fact that not all cigarette smokers develop clinically significant COPD, suggesting that genetic factors may modify each individual’s risk [13], whereas it also remains unclear why individuals-never smokers develop COPD [14]. Smoking is also an important risk factor for PD development and progression [28] and a proven confounder. The association between the two diseases possibly reflects exposure to tobacco smoke [28]. However, it remains unclear whether the susceptibility to tissue destruction that is caused by smoking consists a general individual’s characteristic or whether different individual’s tissues show different reactions to the harmful effects of smoking. If the sensitivity to smoking is not a general individual characteristic the destructive procedures in periodontal tissues will develop independently of changes in the lungs.

SES, and smoking were the main reason why the Cochran and Mantel-Haenszel model was carried out in an effort to clarify if possible significant differences between COPD and non-COPD individuals regarding the PD indices examined could be attributed to those epidemiological parameters. The model showed that smoking and SES were not confounders of PPD and the oral hygiene indices. As mentioned few epidemiological studies, mainly meta-analyses and case control studies have investigated possible differences regarding the periodontal status between individuals suffering from COPD compared with healthy individuals and in some cases have led to contradictory outcomes.

Several limitations must be taken into account in the current retrospective case-control study. It is known that case-controls studies are susceptible to selection, recall, random, referral bias and the their outcomes must be adjusted for known and unknown confounders which can to lead to biased secondary associations regarding the indices examined. In contrary, the prospective cohort studies design can control confounding biases. Other limitations of the current study are the fact that COPD and PD share some common risk factors such as smoking and SES, the fact that PD is considered as a risk factor for COPD pathogenesis, the use of questionnaire as such studies are susceptible to response bias as it is possible that the response given is what the individuals consider the socially adequate response, not the real one.

Some strengths of the current study were the large and the representative study sample and that it was a matched case-control study, as was used randomly selected population-based controls as were selected from non-COPD individuals derived from cases environment,
methodology warrants interval validity. More studies, especially prospective are needed to confirm those findings.

5. CONCLUSION

Probing pocket depth and oral hygiene parameters such as tooth-brushing frequency and dental check-up, as periodontal status indices were statistically significant different between individuals who were suffered from COPD and healthy ones.

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