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Periodontal Disease Increases Risk for Chronic Obstructive Pulmonary Disease

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A B S T R A C T

The aim of this study was to explore whether a periodontal disease could be a risk indicator for a chronic obstructive pulmonary disease (COPD). The examined group comprised 93 patients with COPD (mean age 65.8 years). The control group comprised 43 systemically healthy individuals (mean age 62.1 years). Respiratory and periodontal conditions were examined in both groups. COPB subjects had significantly worse periodontal conditions than controls ($p<0.05$) with regard to each parameter of periodontal condition, except for gingival inflammation. COPD patients had higher Plaque Index than control patients (82.84±22.81 vs. 57.15±26.96; $p<0.001$), higher periodontal depth (3.02±0.92 vs. 2.57±0.79 mm; $p=0.007$), higher gingival recession (1.97±1.09 vs. 0.91±0.79 mm; $p<0.001$), and higher mean clinical attachment loss (CAL) (4.12±1.74 vs. 2.91±1.27 mm; $p<0.001$). Multiple logistic regression model, after controlling for other risk indicators, showed that periodontal disease, presented as CAL ≥4 mm at ≥60% sites, was associated with odds ratio of 3.2 (95% CI 1.0–9.8) for the COPB group. Data suggest that periodontal disease could be a risk indicator for COPD.

***Key words**: chronic obstructive pulmonary disease, smoking, periodontitis, clinical attachment loss, case-control study, Croatia*

Introduction

During the early 1990's many researchers collected evidence, indicating that periodontal disease can represent a risk for certain systemic diseases, such as pulmonary diseases[1–3], cardiovascular diseases[4], premature birth of a child with low birth weight[5] and diabetes mellitus[6,7]. In certain risk populations it has been demonstrated that poor oral health and periodontitis can be associated with several pulmonary conditions[8,9]. Nowadays respiratory diseases significantly contribute to the morbidity and mortality of people worldwide. At the beginning of the 1990's infections of the lower respiratory pathways were the third common cause of death in the world, and chronic obstructive pulmonary disease (COPD) was the sixth[10,11].

COPD, a progressive and irreversible disease, is one of the main causes of death in the world[12,13]. The most significant risk factor for COPD is long-term cigarette smoking, pipe smoking, cigar smoking and people exposed to large amounts of secondhand smoke also are at risk. Occupational exposure to dusts and chemicals, long-term exposure to chemical fumes, vapors and dusts can irritate and inflame your lungs. COPD develops slowly over years, so most people are at least 40 years old when symptoms begin. A rare genetic disorder known as alpha-1-antitrypsin deficiency is the source of a few cases of COPD[14].

Scannapieco's group hypothesised that the oral cavity can have an important role in respiratory infections, i.e. that oral infection and periodontal infection can increase the risk of the occurrence of bacterial pneumonia or COPD[15]. A study by Hayes also indicated correlation between oral hygiene and numerous respiratory diseases[16]. Many anaerobic and aerobic oral bacteria from a periodontal pocket can be aspirated into the lungs where they can induce aspiration pneumonia. The surface of

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the teeth covered with dental plaque can also be a source of colonisation of respiratory pathogens and cause nosocomial pneumonia. Therefore mentioned pathogens can be aspirated into the lungs and cause infection and exacerbation of COPD[10].

The aetiology of periodontal diseases indicates a whole range of local, systemic and congenital factors. Local and extraneous stimuli are bacterial plaque (a result of poor oral hygiene), drugs, smoking, stress etc. Systemic factors include AIDS, acquired endocrine diseases, acquired inflammatory diseases, nutritional deficit, pregnancy, osteoporosis and menopause. Congenital factors are age, gender, inheritance, congenital immunodeficiency, Down's syndrome, Pappilon-Lefevre syndrome, etc[17,18]. Contemporary dentistry has new responsibility and understanding for the care of patients with established periodontitis because not only are the patient's teeth at risk, but also the whole state of health and the entire organism[18–20].

Subjects and Methods

Study population

The retrospective case – control study was performed on 136 dentate subjects, 93 in COPD group, mean age 65.8 years, and 43 in control group of systemically healthy subjects, mean age 62.1 years. Data were collected on their age, body mass index (BMI), education and activities, smoking habits, associated diseases, drugs and the stage and symptoms of COPD. Subjects (age 50–75 years) included in this study were outpatients in the University Hospital Jordanovac, Zagreb with a diagnosis of COPD, all in stable stage and without exacerbations of symptoms in the past 2 months at recruitment. Control subjects in the dental outpatient department Public Health Centre Zagreb-Centre had normal pulmonary function and no other systemic diseases. Subjects that underwent a periodontal treatment in the last year and patients with alpha-1 antitrypsin deficiency were excluded from the study. Each subject in case and control group was informed of the aim of the research and voluntarily agreed to the examination with signed informed consent.

Diagnosis of COPD

The disease was diagnosed on the basis of Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria and FEV, FEV1, FVC values[14]. Spirometry is the basic and most frequently performed, fast and simple examination of pulmonary function, at very reasonable cost. It has exceptionally extensive indications, and usually begins with measurement of the subject's basic personal data, such as date of birth, gender, height, weight, profession and smoking. Classification of COPD according to severity is following:

1. Grade FEV1>80%
2. Grade 50% ≤ FEV1<80%
3. Grade 30% ≤ FEV1<50
4. Grade FEV1<30%

In patients with COPD reduced FEV1 and ratio FEV1/FVCx100 known as Tiffeneau index are commonly found. The degree of the spirometric disorder usually correlates with severity, i.e. the stage of COPD.

Periodontal examination

Periodontal examinations were performed by using a dental mirror and standard periodontal probe (PCP 15, Hu-Friedy, Chicago, IL, USA). Periodontal measurements included determination of probing depth (PD) and gingival recession (RE) on all teeth present. Clinical loss of attachment (CAL) for an individual tooth was determined as the distance from the cementoenamel junction to the bottom of the pocket. The aforementioned value was recorded on the nearest milimeter by one calibrated examiner on six places per tooth (mesiobuccally, buccally, distobuccally, mesiolingually, lingually and distolingually). Calibration was performed on ten patients, of which each had at least 10 teeth and probing depth of >6 mm on at least one side of the tooth. The researcher examined the patient on two occasions, with an interval of 48 hours. Calibration was accepted when both measurements conformed in millimetres, at the level of >90%. Plaque index (PI)[21] and papilla bleeding index (PBI)[22] were also recorded on four sites per tooth (buccal, oral, mesial and distal). The study was carried out in accordance with the ethical principles of the Helsinki Declaration and had been approved by the Ethical Committee of the Zagreb University School of Dental Medicine (01-PA-35-1.10.2.2.5./05, on the basis of Article 47 and 50 of the Law on Institutions of Higher Education and Article 150 of the Statute of the School of Dental Medicine).

Statistical analysis

The following methods were used to test the hypothesis on the possibility of correlation between COPB and demographic and periodontal data: χ^2 test, Mann-Whitney test, one-way analysis of variance. It was hypothesized that the COPD group could have an average CAL 4 mm, and the control group could have an average CAL 3 mm, and that the expected variability (standard deviation) could be 1.5 mm with a power of beta effect = 0.80 and significance level alpha = 0.05. Power analysis estimated that the minimal size required in each group was 37 subjects. The subject was the unit of analysis. Logistic regression was done in order to identify risk indicators and to quantify odds ratios (OR) associated with presence of COPD. Analyses of data were carried out by means of SPSS software (Version 10.0, SPSS Inc, Chicago, IL, USA).

Results

The groups of subjects did not differ with regard to age and BMI (Table 1). COPB group had significantly less non-smokers and more former smokers than control group ($p<0.001$). The results in Table 2 show statistically significant difference between the groups with regard to each parameter of periodontal condition, apart from

TABLE 1
BASIC CHARACTERISTICS OF THE STUDY POPULATION

Characteristic	COPD group (n=93)	Control group (n=43)	p
Mean±standard deviation			
Age (years)	65.75±9.65	62.12±11.91	0.084
BMI (kg/m^2)	27.93±5.45	27.90±4.10	0.967
Distribution of subjects (%)			
Gender			
Male	65 (69.9)	18 (41.9)	
Female	28 (30.1)	25 (58.1)	0.002
Education level			
Elementary school	44 (47.3)	9 (20.9)	
High school	40 (43.0)	19 (44.2)	
College or university	9 (9.7)	15 (34.9)	<0.001
Profession-activity	44 (47.3)	9 (20.9)	
active	42 (45.2)	19 (44.2)	
retired	51 (54.8)	24 (55.8)	0.915
Smoking status			
Non-smoker	17 (18.3)	21 (48.8)	
Former smoker	58 (62.4)	13 (30.2)	
Current smoker	18 (19.4)	9 (20.9)	<0.001

p<0.05 is considered statistically significant; t-test and χ^2 test for categorical variables were used; BMI-body mass index.

gingival inflammation. COPD patients had fewer remaining teeth than control patients (p<0.001), higher PI (82.84±22.81 *vs*. 57.15±26.96; p<0.001), higher PD (3.02±0.92 *vs*. 2.57±0.79 mm; p=0.007) higher gingival RE (1.97±1.09 *vs*. 0.91±0.79 mm; p<0.001), and higher mean CAL (4.12±1.74 *vs*. 2.91±1.27 mm; p<0.001).

Smoking status was presented as the smoking period and pack years (number of pack years = number of cigarettes smoked per day x number of years smoked/20). COPD group had longer smoking period than the control group (33.7±13.29 *vs*. 23.7±12.14; p=0.002). Data analysis also showed that male patients were heavier smokers, had poorer oral hygiene and a more advanced stage of COPD comparing to women.

CAL value ≥ 4 mm at at least 60% of the measured sites was applied to define the presence of periodontal disease. Therefore, the continuous CAL variable was dichotomized, where the subjects below those limit values were considered periodontally healthy. Subjects with CAL ≥ 4 mm at <60% of the sites were also classified as healthy. The univariate analysis revealed that the number of extracted teeth, smoking index, male gender and CAL 4:60 differ significantly between the control and COPB group, where CAL 4:60 and a male gender were the most pronounced risk indicators with unadjusted ORs 7.17 and 3.22, respectively (Table 3). Adjusted ORs were produced in multiple logistic regression model that included data on smoking and the severity and extent of periodontal disease. The model was adjusted for age, gender and BMI of subjects. Periodontal disease, defined as CAL ≥ 4 mm at ≥ 60% of the measured sites, was associated with a 3-fold increased OR for COPB. The smoking index was also associated with COPD, but OR was only 1.03 (Table 4).

Discussion

Contemporary findings indicate that smoking is a major risk factor both for COPD and periodontitis. The present study on the increased risk for COPD among periodontitis patients is the first of its kind in Croatia, carried out on a Croatian population. Our findings can help to promote and to support proper oral hygiene and health as well as of prophylaxis of periodontitis. Furthermore it can also be important in creating guidelines for prevention of development and exacerbations of COPD.

According to numerous periodontal indicators, subjects with COPD in this research had statistically significantly poorer periodontal health but also had fewer re-

TABLE 2
UNIVARIATE ANALYSIS OF SMOKING HABITS AND PERIODONTAL HEALTH IN THE COPD AND CONTROL GROUP

Clinical parameter (Mean±standard deviation)	COPD group (n=93)	Control group (n=43)	p
Smoking period (yrs)	33.7±13.29	23.7±12.14	0.002
Pack years (male)	47.1±32.30 (N=65)	22.7±24.22 (N=18)	0.004
Pack years (female)	26.1±23.23 (N=28)	10.8±22.35 (N=25)	0.019
Pack years	40.7±31.28	15.8±23.63	<0.001
Number of teeth	12.2±7.77	19.8±7.90	<0.001
Plaque index (%)	82.84±22.81	57.15±26.96	<0.001
Papilla bleeding index	2.13±0.90	1.8±1.14	0.159
Pocket probing depth (mm/site)	3.02±0.92	2.57±0.79	0.007
Gingival recession (mm/teeth)	1.97±1.09	0.91±0.79	<0.001
Clinical attachment loss (mm/site)	4.12±1.74	2.91±1.27	<0.001

p<0.05 is considered statistically significant; t-test was used

TABLE 3
UNADJUSTED OR FOR RISK INDICATORS

	COPB	Control	Sig.	OR	95% CI
Age (years)	65.73±9.62	62.12±11.86	0.085	1.033	0.998–1.070
Smoking index (pack years)	40.76±31.28	15.78±23.63	<0.001	1.035	1.018–1.052
BMI (kg/m^2)	27.93±5.44	27.89±4.11	0.967	1.002	0.932–1.076
No. of missing teeth	19.84±7.77	12.23±7.90	<0.001	1.125	1.069–1.184
Gender					
female	28 (30.1%)	25 (58.1%)			
male	65 (69.9%)	18 (41.9%)	0.002	3.224	1.522–6.830
CAL 4:60					
No	43 (46.2%)	37 (86%)			
Yes	50 (53.8%)	6 (14%)	<0.001	7.171	2.762–18.616

OR-odds ratio, CI-confidence interval; BMI, body mass index.

TABLE 4
MULTIVARIATE LOGISTIC REGRESSION MODEL

		B	Std. Error	Wald	Sig.	OR	95% CI
COPB	Intercept	–3.222	1.825	3.116	0.078		
	Age (years)	0.007	0.022	0.109	0.742	1.007	0.965–1.051
	Smoking index (pack years)	0.027	0.010	7.373	0.007	1.027	1.007–1.047
	BMI (kg/m^2)	0.043	0.047	0.832	0.362	1.044	0.952–1.144
	No. of missing teeth	0.060	0.034	3.161	0.075	1.062	0.994–1.134
	Male gender	0.587	0.459	1.638	0.201	1.799	0.732–4.423
	CAL 4:60	1.146	0.581	3.889	0.049	3.145	1.007–9.822

OR-odds ratio, CI-confidence interval; BMI, body mass index.

maining teeth than subjects in the control group. All indicators of the periodontal condition such as PI, RE, PD and CAL were statistically significantly greater in the COPD group, apart from the involvement of gingival inflammation, which is in correspondence with previous studies[23]. Nicotine from cigarettes leads to vasoconstriction of the vascular system and consequently reduces gingival bleeding, i.e. decreased PBI index[24,25].

Researchers noted loss of alveolar attachment ≥4mm as a statistically significant factor for the development and progression of COPD, which is present to a great extent in patients with an advanced stage of COPD[25,26]. Subjects with advanced periodontal disease defined as CAL ≥4 mm at ≥60% of the measured sites were associated with higher ORs for COPD (OR 3.1), which means that presence of periodontal disease could increase the chance for COPD. According to the Research, Science and Therapy Committee of American Academy of Periodontology, »CAL assessments give a better overall estimate of the amount of damage to the periodontium than do PD measurements«[27].

The complex association of demographic, health and periodontal variables with COPB is more clearly shown in the results of the multiple logistic regression. According to this study smoking and PD were the main indicator of risk for COPD. Smoking produced very low OR (1.03), but PD (CAL 4:6) produced higher OR (3.2) and was the only significant risk indicator. In present study smoking status in the COPD group was on average 10 years longer than the smoking status of subjects in the control group. According to the mean smoking indices, the heaviest smokers were male subjects from the COPD group.

The effect of smoking on periodontal tissues seems to be more pronounced in men than in women[28–30]. Former smokers also have almost 4 times higher risk of developing COPD[26,31,32]. All the aforementioned authors confirmed the negative effect of smoking on the periodontium (greater probing depth, greater recession and significantly greater loss of alveolar attachment). Hyman and Reid associated smoking as the main, basic factor in the occurrence and complications of both periodontal disease and COPD[2].

El-Solh et al. associated poor oral hygiene with colonisation of dental plaque as a reservoir of bacteria which cause respiratory infection and pneumonia[33]. Microbiological analysis of dental plaque showed that 57% of the subjects had dental plaque colonised with aerobic pathogens *Staphylococcus aureus* (45%), mainly present in isolates, gram negative bacilli (42%) and *Pseudomonas*

aeruginosa (13%)[12,34,35]. Consequently, aerobic respiratory pathogens colonised in the dental plaque can be an important reservoir for the occurrence of hospital pneumonia that can be significantly decreased in hospitalised patients by improved oral hygiene[11]. Oral cavity has long been considered a potential reservoir for respiratory pathogens. The mechanisms of infection could be aspiration into a lung of oral pathogens capable of causing pneumonia, colonization of dental plaque by respiratory pathogens followed by aspiration, or facilitation by periodontal pathogens of colonization of the upper airway by pulmonary pathogens. These bacteria of oral origin, especially *Aggregatibacter actinomycetemcomitans*, may stimulate inflammatory response associated with respiratory epithelial cells, by means of increased cytokines production and inflammatory cells recruitment[1,36].

The currently available data from observational studies are insufficient to accurately estimate the strength of the association. Our study noted findings that are derived from retrospective case–control study. Prospective cohort epidemiologic studies could better estimate possibilities for progression and exacerbation of COPD.

In conclusion, a particularly important aim of the present study was to continually inform current and risk patients and medical workers of the great importance of maintaining correct oral hygiene, frequent dental check-ups, increased awareness and education on the health of the teeth and mouth, thereby reducing the possibility of persistent inflammation and microorganisms in the oral cavity.

Although poor oral hygiene is not solely responsible for COPD, there are many other factors such as environmental pollutants, smoking, allergies, viral infections and genetic factors that are contributing occurrence of COPD. 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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PARODONTNA BOLEST POVEĆAVA RIZIK ZA KRONIČNU OPSTRUKTIVNU BOLEST PLUĆA

S A Ž E T A K

Svrha ovog istraživanja bila je utvrditi može li parodontna bolest biti indikator rizika za kroničnu opstruktivnu bolest pluća (COPB). Ispitivana skupina se sastojala od 93 pacijenata s COPB (prosječna dob 65,8 godina). Kontrolnu skupinu je sačinjavalo 43 sistemski zdravih osoba (prosječna dob 62,1 godina). Kod obje skupine utvrđen je respiratorni i parodontni status. Pacijenti s COPB imali su signifikantno lošiji parodontni status u odnosu na kontrolnu skupinu ($p<0.05$) obzirom na svaki pokazatelj parodontnog zdravlja (osim upale gingive). Kod istih su izmjerene i veće vrijednosti indeksa plaka u odnosu na kontrolnu skupinu (82,84±22,81 *vs*. 57,15±26,96; $p<0.001$), veće dubine sondiranja (3,02±0,92 mm *vs*. 2,57±0,79 mm; $p=0,007$), izraženije recesije gingive (1,97±1,09 mm *vs*. 0,91±0,79 mm; $p<0,001$) te veći prosječni gubitak kliničkog pričvrstka (CAL) (4,12±1,74 mm *vs*. 2,91±1,27 mm; $p<0,001$). Uz kontrolu ostalih čimbenika rizika, multipli logistički regresijski model u ispitivanoj skupini je pokazao da je parodontna bolest, izražena kao CAL ≥4 mm na ≥60% zubnih ploha, povezana s omjerom rizika od 3,2 (95% CI 1,0–9,8). Podaci ukazuju da bi parodontna bolest mogla biti indikator rizika za COPB.