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RESEARCH ARTICLE

Dental & Craniofacial Research

Epidemiological risk factors for periodontal pockets and clinical attachment loss among Greek adults

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Abstract

Aim: *The aim of this study was to identify variables related to deep periodontal pockets and clinical attachment loss.*

Materials and methods: *The study population consisted of 575 Greek adults, 259 males and 316 females aged 35 to 69 years who referred in a private practice for periodontal treatment. Participants completed a self-administered questionnaire which included several epidemiological variables and underwent an oral clinical examination. The analyses performed by multinomial logistic regression model to estimate the possible associations among the variables examined.*

Results: *31.3% of the participants showed a mean probing pocket depth of >6.00 mm, and 67.1% showed a mean clinical attachment loss of ≥5.0 mm. Male gender, lower socio-economic status, smoking; irregular dental follow-up and a diabetes mellitus history were consistent statistically significant potential risk factors for probing pocket depth of ≥4.00 mm and clinical attachment loss of ≥3.00 mm.*

Conclusion: *These results confirm previous findings regarding the principal role of cigarette smoking and various epidemiological variables in the etiology of deep periodontal pockets and periodontal loss of attachment.*

Keywords: *periodontal disease; epidemiology; periodontal pockets; attachment loss; adults; risk factors*

Introduction

Periodontal Disease (PD) is one of the most common chronic infectious diseases, and its overall prevalence varies from 10 to 90% in adults [1], depending on diagnostic criteria. PD prevalence is particularly high in a general adult population and is the main cause of tooth extraction in adults aged ≥40 years [2]. It has been reported that PD affects not only oral physiological tissues but also systemic diseases such as diabetes mellitus (DM), cardiovascular disease, pre-mature birth, and bacterial respiratory disease [3-5].

During the progression of PD deep periodontal pockets and clinical attachment loss (CAL) observed through the destruction of the periodontal tissue and alveolar bone. Those conditions can lead to gingival recession and deeper periodontal pockets [6]. The degree of pocket depth reflects the inflammation activity, whereas the degree of CAL can be used as an index to assess PD severity [7].

PD, gingivitis and periodontitis mainly is a chronic infectious disease which leads to the formation of a biofilm on tooth and root surfaces and results in destruction of periodontal tissue caused by an uncontrolled host response to pathogenic bacteria and their toxins [8,9]. Severity and progression of PD is associated with microbiological burden, host's susceptibility and modified by behavioral and environmental factors [10]. PD is caused mainly due to bacteria found in periodontal pocket and gingival crevice, however the disease may be affected indirectly by various risk indicators that can lead to systemic immunological responses, severity of inflammatory reactions and the vascular circulation [6,11]. The mentioned risk indicators can affect PD initiation and progression and differ among countries examined [12].

Previous reports have identified smoking [13,14], DM [15-17], genetic predisposition [18,19], increasing age [20,21], male gender [22,23], social and psychological factors [24,25], some ethnicities, low educational level, poor economic status, and poor oral hygiene [26-29], to be associated with periodontitis as risk factors or risk indicators.

Additional potential risk factors recorded in previous reports such as osteoporosis, angina, allergy, frequency of dental visits, and family history of edentulism [30], whereas international surveys have shown that gingivitis and periodontitis are more prevalent in lower socio-economic and educational level populations [31].

The aim of the current research was to identify epidemiological risk factors associated with periodontal pockets and CAL in a sample of Greek adults.

Material and methods

Study population

PA cross-sectional retrospective descriptive study was conducted from November 2017 to September 2018. The reference population for this study was comprised of adult males and females aged 35 to 69 years who lived in the urban area of the 3rd large city in Greece.

The mentioned age group is recommended for epidemiological surveys which examine the oral health of adults and was based on the World Health Organization (WHO) recommendations [32, 33] to assess disease prevalence. This procedure yielded a sample size of 575 individuals. Participants included in the study completed a health medical and dental questionnaire and underwent an oral clinical examination.

Patient's selection criteria

Individuals who had undergone a previous periodontal treatment, conservative or surgical, within the previous six months were excluded from the study sample. None of the participants had received a prescription of anti-inflammatory or systemic antibiotics or other systemic drugs the previous six weeks [34]. Participants was necessary to have a mean of 20 natural teeth, since large numbers of missing teeth could lead to over- or under estimate the dental variables and the possible associations that were under consideration. Those criteria were determined because of the potential effects on the oral tissues.

Oral clinical examination

One well trained and calibrated dentist performed the examinations. The clinical measurements concerned each tooth in all quadrants, except for the 3rd molars and the remaining roots, probing pocket depth (PPD) and clinical attachment loss (CAL) recorded using a William's 12 PCP probe (PCP 10-SE, Hu-Friedy) at six sites (facial, mesio-facial, disto-facial, lingual, disto-lingual and mesio-lingual). In the current research mean PPD and CAL were used as indices for the assessing of PD severity.

Mean PPD was categorized into three groups: 0-3.00 mm (no disease/mild disease), ≥4.0 mm <6.0 mm (moderate disease) and ≥6.0 mm (severe disease) [35], and mean CAL was also categorized into three groups: 1-2.00 mm (slight), 3-4.00 mm (moderate) and ≥ 5.0 mm (severe) [36].

Clinical measurements of PPD and CAL concerned the immediate full millimeter. In cases the tooth cervix was destructed by decay, erosion, abrasion, or another lesion, or the cement-enamel junction (CEJ) was covered by a filling or prosthetic restoration its location was recorded by extrapolating the CEJ location from the adjacent teeth, whereas no record was recorded in case was not visible.

Patient's selection criteria

Participants completed a self-administered questionnaire. The content of the questionnaire based on the University of Minnesota Dental School Medical Questionnaire [37] with some modifications and included the following demographic and socioeconomic indices: age (35-69 years), gender (male, female), smoking status (never smokers and former/current smokers), socio-economic status (≤1,000 and >1,000 €/month), educational level (elementary level, graduated from University/College), presence or absence of DM, frequency of a regular dental follow-up (no examination/year and 2 times/year).

A randomly chosen sample of 115 (20%) individuals was re-examined clinically by the same dentist after 3 weeks in order to establish the intra-examiner variance. After consideration of the double examined individuals no differences were recorded between the 1st and the 2nd clinical assessment *(Cohen's Kappa= 0.94).*

Ethical consideration

In Greece only experimental studies must be reviewed and approved by authorized committees (Dental Schools, Greek Dental Associations, Ministry of Health, etc) and the current study was not an experimental one. However, it was carried out in full accordance with the World Medical Association Declaration of Helsinki. Individuals who accepted the invitation to participate in the study protocol signed an informed consent form.

Statistical analysis

For each individual the mean value of PPD and CAL at the six sites per tooth recorded and coded as dichotomous variables. Never smokers, individuals with lower educational (elementary level) and socio-economic (income/monthly ≤1,000 €) status, females, individuals with an irregular dental follow-up and individuals who do not suffer from DM coded as 0. Age groups distribution was coded as 0, 1, 2 and 3 for ages 35-39, 40-49, 50-59 and 60-69, respectively.

Multinomial logistic regression model was carried out to produce weighted population estimates. Mean PPD and CAL values were the dependent variables. In the multinomial models, reference group for all comparisons were the following groups: 0-3.00 mm mean PPD group and 1-2.00 mm mean CAL group. Adjusted Odds Ratios (AOR's) and 95% CI (Confidence Interval) were also assessed. Statistical analysis performed using the statistical package of SPSS ver.19.0 and a p value less than 5% ($p< 0.05$) was considered to be statistically significant.

Results and discussion

Table 1 represents the epidemiological variables according to the age groups of the study population. A total of 244 participants, or 42.4% of the study sample aged >50 had a mean PPD of 4.0 mm or more compared to those aged <50 who were 189 (32.9%), whereas 268 (46.6%) of those with the same ages showed a mean CAL of 3.00 mm or more compared to those aged <50 who were 177 (30.8%).

Tables 2 and 3 show the distribution of mean PPD and mean CAL according to selected potential risk factors. A total of 253 individuals (44,0%) showed a mean PPD of ≥4 to <6.00 mm and 180 (31.3%) had a mean PPD of >6.00 mm. Similarly, 103 individuals (17.9%) showed a mean CAL of 3-4.0 mm and 386 (67.1%) showed a mean CAL of ≥5.0 mm.

According to the multinomial regression model, male gender, lower socio-economic status, smoking, irregular dental follow-up and a DM history were consistent statistically significant potential risk factors for PPD of ≥4.00 mm. Participants with a mean PPD of 0-3.00 mm were the reference group (Table 4).

The application of the same statistical model showed that all the potential risk factors examined were found to be statistically

	35-39 years old N (%)	40-49 years old N (%)	50-59 years old N (%)	60-69 years old N (%)
Gender				
Females	5252	85 48.6	8641.1	36 39.6
Males	4848	90 51.4	12358.9	55 60.4
Educational level				
Low	4848	92 52.6	11555	50 54.9
High	5252	83 47.4	9445	41 45.1
Social/economic level				
Low	3131	77 44.0	8842.1	32 35.2
High	6969	98 56.0	12157.9	59 64.8
Smoking status				
No	4343	6336	8540.7	37 40.7
Yes	5757	11264	12459.3	54 59.3
DM history				
No	7676	13878.9	14669.9	58 63.7
Yes	2424	3721.1	6330.1	33 36.3
Dental Follow-up				
No/year	4848	6034.3	3918.7	28 30.8
2 times/year	5252	11565.7	17081.3	63 69.2
Mean PPD				
0-3.0mm	4747	39 22.3	42 20.0	14 15.4
≥4.0-<6.0mm	4242	77 44.0	94 45.0	40 44.0
≥6.0mm	1111	59 33.7	73 35.0	37 40.6
Mean CAL				
1-2.0mm	5454	44 25.1	2110	11 12.1
3-4.0mm	3232	67 38.3	7837.3	32 35.2
≥5.0mm	1414	64 36.6	11052.7	48 52.7

Table 1. *Epidemiological variables according to the age groups*

	Mean PPD 0-3.0 mm N (%)	Mean PPD ≥4-<6.00 mm N (%)	Mean PPD ≥6.0 mm N (%)
Gender			
Females	80 56.3	95 37.5	8446.7
Males	62 43.7	158 62.5	9653.3
Educational level			
Low	63 44.4	112 44.3	13072.2
High	79 55.6	141 55.7	5027.8
Social/economic level			
Low	46 32.4	9838.7	10860.0
High	96 67.6	15561.3	7240.0
Smoking status			
No	73 51.4	10139.9	5419.3
Yes	69 48.6	15260.1	12680.7
DM history			
No	95 66.9	20179.4	12267.8
Yes	47 33.1	5220.6	5832.2
Dental Follow-up			
No/year	26 18.3	17970.8	17970.8
2 times/year	116 81.7	7429.2	7429.2

Table 2. *Distribution of mean PPD according to selected potential risk factors*

	Mean CAL 1 - 2.0 mm N (%)	Mean CAL 3-4.0 mm N (%)	Mean CAL ≥5.0 mm N (%)
Gender			
Females	31 36.0	5149.5	177 45.9
Males	55 64.0	5250.5	209 54.1
Educational level			
Low	13 15.1	4947.5	223 57.8
High	73 84.9	5452.5	163 42.2
Social/economic level			
Low	26 30.2	4745.6	155 40.2
High	60 69.8	5654.4	231 59.8
Smoking status			
No	66 76.7	4644.7	126 32.6
Yes	20 23.3	5755.3	260 67.4
DM history			
No	65 75.6	7068.0	28373.3
Yes	21 24.4	3332.0	10326.7
Dental Follow-up			
No/year	20 23.3	6159.2	21455.4
2 times/year	66 76.7	4240.8	17244.6

Table 3. *Distribution of mean CAL according to selected potential risk factors*

	Mean PPD ≥4 - <6.00 mm	Mean PPD ≥6.0 mm
Gender		
Females	1.00	1.00
Males	1.47* (0.94-2.31)	1.38* (0.73-1.83)
Educational level		
Low	1.00	1.00
High	1.11 (0.62-2.00)	1.19 (0.69-1.63)
Social/economic level		
Low	1.00	1.00
High	1.49* (0.56-1.94)	1.76* (0.82-2.10)
Smoking status		
No	1.00	1.00
Yes	1.80** (0.76-2.17)	1.77** (0.49-2.30)
DM history		
No	1.00	1.00
Yes	1.18 (0.61-1.57)	1.54* (0.68-2.31)
Dental Follow-up		
No/year	1.00	1.00
2 times/year	1.14 (0.47-1.98)	1.37* (0.61-2.05)

* p≤ 0.05; ** p≤ 0.01; Individuals with a mean PPD of 0.00-3.00 mm are the reference group

Table 4. *Adjusted odds ratios (OR) and 95 % Confidence Intervals (95% CI) of PPD and selected potential risk factors among the study population*

	Mean CAL 3-4.0 mm	Mean CAL ≥5.0 mm
Gender		
Females	1.00	1.00
Males	1.32* (0.71-2.46)	1.25* (0.58-1.65)
Educational level		
Low	1.00	1.00
High	1.22 (0.28-1.91)	1.93* (0.56-2.59)
Social/economic level		
Low	1.00	1.00
High	1.45* (0.70-1.83)	1.77** (0.62-1.88)
Smoking status		
No	1.00	1.00
Yes	1.58** (0.43-2.07)	1.94** (0.71-2.63)
DM history		
No	1.00	1.00
Yes	1.12 (0.51-1.87)	1.87* (0.67-2.11)
Dental Follow-up		
No/year	1.00	1.00
2 times/year	1.21 (0.47-1.98)	1.82** (0.49-2.16)

*p≤ 0.05; ** p≤ 0.01; Individuals with a mean CAL of 1.00-2.00 mm are the reference group.

Table 5. *Adjusted odds ratios (OR) and 95 % Confidence Intervals (95% CI) of CAL and selected potential risk factors among the study population*

significant with a mean CAL of ≥ 3.00 mm (Table 5). In that case the reference group included individuals who showed a mean CAL of 1-2.00 mm.

The current retrospective study investigated the associations between various epidemiological variables and PD indices in terms of PPD and CAL using the cross-sectional data of 575 individuals referred to a dental clinic for periodontal treatment. The disadvantages of retrospective studies are that characterized by limitations as do not show high reliability as the prospective ones because of the effect of systemic biases during the samples election, recall biases and known and unknown confounders that can lead to biased correlations. Another limitation was that the study population was not randomly selected from a representative population but consisted of PD patients' that referred for periodontal treatment; however its size was estimated using the equation proposed by Lwanga and Lemeshow [33].

In the current study it was found that male gender was significantly associated with the worst hbn values of PPD and CAL, ≥4.00 mm and >3.00 mm, respectively. A large amount of previous reports have recorded more serious periodontal destruction among males compared to the female population [22,38-43]. There as on for those gender differences are not clear, and could be explained by the facts that males have poorer oral hygiene practices than females [44], access to dental care is different between both genders [45] and also could be attributed to the ignorance of oral hygiene, which is usually observed among males [43,46]. However, the association recorded between gender and the disease is not clearly visible and is not considered as strong and consistent, as gender is a demographic variable, which may interfere with the effects of other factors and it must be controlled for searching the disease.

The results also showed that low level of education was significantly associated with a mean of CAL ≥ 5.00 mm, whereas OR for PPD ≥6.0 mm was 1.19. That means a higher prevalence of PD was assessed among individuals with lower educational level than among the ones with higher educational level. Similar findings have been confirmed by previous studies [40,47-50].

PD patients exhibited lower educational levels and had difficulties with their affiliation to social health services. The same report recorded worse PD indices in individuals lacking education, or with basic primary education [51]. Jiang et al. [52] observed that risks of oral diseases increase in patients with lower educational or academic training, or lack health insurance affiliation. Similarly, Ababneh et al. [49] found that this link could be associated to difficulties to access health services and other help for the maintenance of suitable oral health, whereas Borrell et al. [48] observed that individuals with lower school educational levels were 3 times more susceptible to suffer from PD that those with higher educational level.

Significant associations observed between socio-economic status (SES) and the worse mean PPD and CAL. Previous studies have recorded significant difference in PD severity among individuals of different SES [7,12,47-49,53-57]. Similar association has been established with other socio-economic parameters such as level of education and income and could be attributed to the close link between educational level income and occupation [58,59]. Other possible explanations are that those individuals are seeking for complex periodontal treatments which are not covered by benefit plans of the General System for Health, and probably the difficulty in providing dental treatment and oral hygiene aids.

However, some studies have shown a weak association between SES and periodontitis after adjustment for oral hygiene and smoking [22,50]. Individuals with higher SES wish to have better periodontal health and this is in accordance with the general belief that those individuals have healthier behaviors than individuals with lower SES [60]. This has been adduced to the better oral health awareness caused by educational level of the individuals. Several diseases and disorders are hypothesized to be associated with SES. Individuals who have higher educational level, greater purchasing power and live under more favorable conditions, show better health conditions than the ones who have lower educational levels and live under less favorable conditions [61]. On the other hand, analysis of the association between social and economic factors and PD could lead to the development of public policies to improve the populations' health [62].

Recently, there has been an increasing emphasis on the importance of social, environmental and economic factors in an effort to understand oral diseases, and public health investigation has focused on the social determinants of health and illness with the recognition of the limitations of the traditional preventive access in improving health and reducing social disparity [63].

In developing countries populations with low SES show a higher prevalence of diseases compared with populations with

higher socioeconomic levels [64] because the social statuses of a population are a determinant of health status [65]. This association must also apply to oral health which is a crucial contributor to general health [66]. In addition, oral health is now considered an important and integral part of general health because poor oral health causes pain, discomfort, affects speech and can affect daily functioning and the general perception of health [67]. Gingival condition is clearly related to lower SES, but the relationship between SES and periodontitis is less direct. It can be certain that gingival health is better among individuals with higher education and more secure income. SES is a modifiable factor and it can be examined in multivariate models for the disease.

Strong associations also observed between smoking status and a mean PPD of ≥4.00 mm and CAL ≥3.00 mm in the current study. The relationship between smoking and periodontal health has been investigated and a large amount of epidemiological, clinical and in vitro studies have provided strong evidence that smoking negatively affects periodontal health and suggest mechanisms by which this may occur [7,12,38,49,51,68-72]. Cigarette smoking is an environmental risk factor most associated with the progression of PD [73]. Smokers are 2 to 7 times more likely to suffer from periodontitis, whereas heavy smokers are twice as likely to present CAL and alveolar boneloss (ABL) than light smokers [40,74,75].

It has been shown that smokers were 2.7 times more likely to have moderate to advanced PD, compared with non-smokers [76]. Significant associations between smoking and both CAL and ABL have also been found [40].

Cigarette smoking affects the inflammatory and immune responses, as well as the microvasculature [77], causesan essential destructive effect on the periodontal tissues and contributes to PD progression [75]. It seems that cigarette smoking modify the host response to the bacteria in dental plaque [79,80]. Smokers with PDs eem to haveless clinical inflammation signs and gingival bleeding compared to non-smokers [81-83], however in fact smokers may show less gingival bleeding than non-smokers with lower plaque indices [84], observation that shows an alteration of the blood vessels diameter which drench the gingival tissues [85]. It has also been suggested that reduced bleeding is associated with an underlying disruption of the immune responseand that this condition may lead to the increased CAL and ABL [86,87]. That finding could be attributed to the fact that nicotine causes a local vascular constriction and edema which can reduce blood circulation, and clinical signs of inflammation [88]. It has been revealed that in the development of nicotine related periodontitis the nicotine acetylcholine receptor plays a crucial role [89].

Cross-sectional and longitudinal surveys have shown that the risk of developing PD as measured by CAL and ABL increases in heavy smokers. Those studies recorded that former smokers, clinically defined as two or more years since quitting smoking, showedless CAL than current smokers but more than never-smokers [90]. Ors for developing PD as a result of smoking range from 2.5 [84], 3.97 for current smokers and 1.68 for former smokers [90] and 3.25 for light smokers to 7.28 for heavy smokers [40]. In vitro studies have shown altered gingival crevicular fluid inflammatory cytokine profiles, immune cell function [91,92] and altered proteolylic regulation in smokers. Nonetheless, the results of those studies are not consistent and to date no clear mechanism has emerged to explain how smoking may affect PD. A new area of study will examine the relationship between smoking and genetic polymorphisms. However, the results of investigations carried out to date cannot be considered definitive. DM is a modifiable risk factor for PD as it can be controlled but not be treated. The current study showed that DM patients were found to be significantly associated with a mean PPD of ≥6.00 mm and CAL of ≥5.00 mm.

Previous reports have recorded associations between PD and the presence of DM and this association is considered to be bidirectional, e.g. diabetes is a risk factor for periodontitis, and periodontitis is a possible factor for DM [93-97].

An important oral clinical sign of DM is gingivitis and periodontitis. Undiagnosed or poorly controlled DM type 1 or 2 can lead to higher risk for PD, whereas poor control of diabetes in the presence of calculus is associated with higher frequency PPD of ≥4.00 mm [74,75]. Previous reports have found a relationship between DM and an increased susceptibility to oral infections including PD [98-102]. In addition, periodontitis also progresses more rapidly in cases of poor DM control [103], and early age of its appearance is considered as a risk factor for more severe diseases [104].

Surveys which have investigated the association between both diseases show heterogeneity in aim and design and both positive and negative observations have been drawn regarding the association examined. Periodontal indices examined have included CAL, gingivitis, and ABL. In general, no difference has been observed between type 1and type 2 DM regarding its action in periodontal tissues [90].

Another cross sectional study showed an OR of 2.8 to 3.4 for developing PD in type 2 diabetics compared to non-diabetics patients [105]. Similarly, longitudinal studies have shown an increased risk of periodontal destruction in DM patients as compared to non-DM with an OR of 4.2 [106,107].

Lack of a regular dental follow-up was significantly associated with a mean PPD of ≥6.00 mm and CAL of ≥5.00 mm, finding that was in agreement with the findings of previous studies [49,108,109]. Only one study recorded different findings [110].

As has already mentioned individuals with a SES are generally wish to have better periodontal health and this is in agreement with the general belief that people in higher SES have healthier oral behaviors and lifestyles than do people in lower SES [60].

Conclusions

In conclusion, the results support previous findings regarding cigarette smoking, especially its role in the etiology of severe PPD and CAL. It was also found evidence of an association between male gender, lower socio-economic level, presence of Diabetes Mellitus and irregular dental follow-up and the periodontal indices examined.

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