

Evaluation of Risk Indicators for Clinical Attachment Loss in a Greek Adult Population

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Abstract:

The aim of the current study was to assess the prevalence and extent of Clinical Attachment Loss (CAL) and to find out variables associated with CAL in a Greek adult population older than 40 years of age. The study sample consisted of 1,240 adults, 580 males and 660 females who were patients of three private practices. The participants were interviewed and underwent an oral clinical examination. CAL assessment was carried out on six sites for each tooth. Prevalence and severity of CAL and the association with variables such as age, gender, socio-economic status (SES), educational level, smoking status, frequency of tooth brushing and dental follow-up, glucose, and blood pressure levels, probing pocket depth (PPD) and bleeding on probing (BOP) were assessed, using univariate and multivariate regression models. The prevalence of CAL \geq 3.0 mm was 22.1% for both genders, whereas the regression model showed that lower SES ($p=0.001$), smoking ($p<0.001$), irregular daily tooth brushing ($p=0.013$), BOP ($p<0.001$) and deeper periodontal pockets ($p=0.001$) were statistically significantly associated with CAL severity. Poor oral hygiene as expressed by PPD, BOP and irregular daily tooth brushing, lower SES and smoking, were significantly associated with CAL severity.

Keywords: Periodontitis, Clinical attachment loss, Epidemiology, Risk factor

Introduction

Periodontal Disease (PD) is one of the most common chronic infectious diseases, its overall prevalence varies from 10 to 90% and is the leading cause for tooth extraction among adults aged \geq 40 years [1,2]. PD and especially periodontitis is a teeth supporting tissues chronic inflammatory condition which is caused by dental plaque accumulation. Subgingival biofilms in periodontal pockets extend apically and lead to host -immune response to the bacterial plaque species which is responsible for the extent and severity of periodontitis [3,4]. The severity of PD varies over time, it depends on the biofilm's quantity and quality and factors modifying the presence of dental plaque [5]. Moreover, its severity and progression is associated with oral pathogens burden, host's susceptibility and modified by behavioral and environmental factors [6]. The host -immune reaction to the plaque pathogens results in the production of cytokines, chemokines and other inflammatory mediators which cause tissue damage and leads to destruction of the alveolar bone, attachment loss and periodontal pockets formation [4]. The immuno -inflammatory process associated with periodontitis results in an apical migration of the epithelial attachment and loss of periodontal soft and hard tissues [7]. If the patient does not receive a periodontal treatment inflammatory reaction can lead to gingival recession deeper periodontal pockets, more attachment loss, and eventually tooth loss [8].

Clinical diagnosis of periodontitis can be made by the measuring of periodontal pocket depth (PPD), clinical attachment loss (CAL), alveolar bone loss (ABL), or a combination of those indices [9].

CAL is a clinical indicator of the degree of remaining tooth support, and is an index for assessing PD severity, whereas (PPD) is an index of inflammatory activity [8,10]. Oral microorganisms play a crucial role in chronic periodontitis etiology [11], however, various risk indicators are involved in PD initiation and progression and differ among countries [3]. Such indicators are genetic predisposition [12], male gender [10,13], increasing age [2], smoking [14-16], ethnicities, educational level, socio-economic status, oral hygiene [14,15,17], social and psychological factors [18], plaque and bleeding indices [14,15] number of missing teeth, dental plaque accumulation, [17, 19], and diabetes mellitus (DM) [14,15]. Those indicators can lead to systemic inflammatory reactions and are involved in the severity of those reactions [8].

Aging is associated with PD, although this association could be attributed to the cumulative periodontal breakdown over time than to an age-related, intrinsic deficiency that contributes to susceptibility to PD [13]. Epidemiological studies have shown more severe PD based on the assessment of CAL and bone loss, among older age groups compared to younger groups [2,19-21]. Adult males present a higher risk for CAL [2,16], whereas smoking is also an important risk indicator, which contributes to a higher prevalence and severity of periodontitis in adults [22], as significant associations between cigarette smoking and both CAL and alveolar bone loss have been recorded [16,19-21]. Only one study has documented the distribution of chronic periodontitis its determinants, prevalence, severity, extension, and risk factors in Greece [23].

However, there is a considerable lack of methodological consistency in periodontal epidemiology, as significant variations have been proposed to the definition of periodontitis [24,25].

Materials and Methods

Study design

A cross-sectional, epidemiological study was carried out between 2019 and 2020. The study size was estimated considering the CAL prevalence determined by Hyman et al. [16], with 95% confidence interval and 1.5% range of error, whereas the age group was based on the World Health Organization (WHO) recommendations [26,27] for assessing disease prevalence. This procedure led to a study sample of 1240 individuals [16]. The participants filled in a health medical and dental questionnaire and underwent an oral clinical examination by a trained and calibrated dentist.

From the study protocol we excluded individuals who had less than 20 natural teeth, since large numbers of missing teeth could lead to over or underestimate the indices and associations examined, those who had undergone a previous periodontal treatment, conservative or surgical, within the previous six months and those who had received a prescription of anti-inflammatory or systemic antibiotics or other systemic drugs the previous six weeks [28]. Those conditions could have potential effects on the oral tissues and could lead to biased secondary associations.

Medical and Dental Questionnaire

Participants completed a modified Minnesota Dental School Medical Questionnaire [29]. The questionnaire contained information regarding age, gender, smoking and alcohol consumption, present illness, past medical history and family history, dental history and other demographic and socioeconomic parameters. Measurements of blood glucose and blood pressure were based on the current medical files of the participants.

According to age the participants were categorized as 40-49, 50-59, 60+, educational level as elementary level, graduated from University/College; socio-economic status as $\leq 1,000$ and $>1,000$ €/month; smoking status as never smokers and former/current smokers; presence or absence of DM; presence or absence of hypertension; frequency of a regular dental follow-up as ≤ 2 examinations/year and >2 times/year; frequency of tooth brushing as ≤ 2 times/daily and >2 times/daily. PD indices were categorized as 0-3.00 mm (no disease/mild disease) and ≥ 4.0 mm (moderate and severe disease) for mean PPD [30], ≤ 3.00 mm (slight) and ≥ 3.00 mm (moderate and severe) for mean CAL [31], and BOP as present or not within 30 seconds following probing with gentle pressure at six sites per tooth [32].

Periodontal tissue examination

Periodontal clinical indices were assessed at six sites (mesio-buccal, midbuccal, disto-buccal, disto-lingual, mid-lingual and mesio-lingual) in all teeth, excluding third molars, using a manual periodontal probe (UNC-15; Hu Friedy Mfg. Co. Inc., Chicago, IL USA). PPD and CAL clinical measurements concerned the immediate full millimeter.

A randomly chosen sample of 250 (20%) individuals was reexamined clinically by the same dentist after 3 weeks in order to establish the intra-examiner variance. No differences were recorded after the second clinical examination (Cohen's Kappa=0.92).

Statistical analysis

The mean value of PPD and CAL at six sites per tooth was recorded and coded as dichotomous variables for each participant.

Females, individuals with lower educational (elementary level) and socio-economic (income/monthly $\leq 1,000$ €) [33] status, never smokers, individuals with an irregular dental follow-up and daily tooth brushing, individuals who did not suffer from DM or hypertension were coded as 0. Individuals with absence or mild periodontal pockets, those with slight attachment loss and those without BOP were coded as 0. The associations between CAL and demographic/clinical parameters or risk factors were analyzed using the chi-square test (univariate analysis).

Multivariate regression analysis was carried out to investigate the associations between the dependent variable, CAL, and independent ones that were determined by the enter method. Finally, the independent variables were included to stepwise method in order to estimate gradually the variables that showed significant associations with the dependent one. Unadjusted (UOR's) and Adjusted Odds Ratios (AOR's) and 95% CI (Confidence Interval) were also assessed. All statistical analyses were performed using SPSS statistical package (SPSS PC19.0, SPSS, Inc., Chicago, IL, USA). A p-value of < 0.05 was considered statistically significant.

Ethical consideration

The current cross-sectional study was not an experimental one. In Greece only experimental studies must be reviewed and approved by authorized committees (Dental Schools, Greek Dental Associations, Ministry of Health, etc.). 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.

Results and Discussion

Table 1 presents the epidemiological variables according to the univariate analysis and shows that lower SES ($p=0.001$), smoking ($p=0.000$), irregular tooth brushing ($p=0.013$), deep periodontal pockets ($p=0.001$) and BOP ($p=0.000$) were statistically significantly associated with moderate and severe CAL (≥ 3.00 mm), which means that those factors were significantly associated with an increased risk for CAL aggravation. The distribution of mean CAL showed that 967 individuals (77.9%) had a mean CAL < 3.0 mm (slight condition) and 273 (22.1%) had a mean CAL ≥ 3.00 mm, (moderate and severe condition).

Similarly, 151 individuals (12.2%) with deep periodontal pockets ≥ 4.0 mm showed moderate and severe CAL (≥ 3.00 mm), and 163 individuals (13.1%) with BOP also showed moderate and severe CAL. The application of the multivariate regression analysis model confirmed the previous associations. Regarding the indices examined, after controlling for possible confounders such as age, gender, smoking status, educational level and SES (Table 2).

The aims in the present cross-sectional study were to assess the prevalence of CAL and to determine some of the various risk factors of CAL in a Greek adult population. The results indicated that 77.9% of the individuals showed mild CAL (< 3.0 mm) and 22.1% of them showed moderate and severe CAL (≥ 3.00 mm). Similar previous studies recorded different prevalence for mild CAL, which ranged from 55.2 % [34] to 82.4 % [35] of the study sample, for moderate and severe CAL, which ranged from 17.6% [35], 41.9% [36], 44.8% [34] to 89.7% [37], and for severe CAL, which ranged from 3.6% [38], 19.7% [2], 46.7% [39], 67.1% [23], to 79% [17] of the study individuals. In some of the mentioned studies the differences could be attributed to the age composition of the sample investigated as their samples consisted of younger individuals (≥ 20 years old) [37,39]. Other studies recorded that CAL prevalence 3-4.0 mm occurred in 17.9% [24], ≥ 4.00 mm occurred in 51% [40] and in 13.2% [36] of the individuals of the study, and < 5.00 mm occurred in 96.4% [38] of the individuals of the study. The mentioned variations of CAL prevalence in different population samples worldwide could be attributed to significant differences in age composition, cultural factors, SES and living habits of the populations investigated [38]. No association between increasing age and CAL severity was also recorded, finding that was in line with another recent study in Greece [23]. However, previous reports did not confirm such a finding [2,20,34,35,38,41,42].

Male gender did not show more severe CAL compared to females, finding that was not in agreement with those from previous reports [2,10,13,17,23,34,35,41-43], except for few studies [38]. These differences may be explained by the facts that females usually are more aesthetically conscious, thus would be more worried about visiting the dentist whereas, males have poorer oral hygiene practices than female's ignorance of oral hygiene and less dental visit behaviors [44]. CAL severity was not significantly higher between lower and higher educational level of the individuals investigated, according to the multivariate analysis, finding that was in line with a previous study [41]. However, similar studies have revealed that low educational level was significantly associated with CAL severity [23,41,42,45-47]. Worse PD indices have been detected in individuals who lack education or having a basic primary education. Moreover, those individuals had difficulties with their access to social health services [48]. Risks of oral disease have shown an increased rate in individuals with lower educational or academic training or lack health insurance access [47].

A statistically significant association between lower SES, which is a modifiable environmental factor, and the worse mean CAL was recorded in the current study, finding that has been confirmed by previous reports which have revealed significant difference in PD severity among individuals of different SES [3,17,23,37,45-47]. Few studies have not recorded such an association [42], or a weak association between SES and PD [13].

	Mean CAL < 3.0 mm	Mean CAL ≥3.0 mm	p-value	Odds Ratio and 95% Confidence Interval
Gender				
Males	425 (44.0)	125 (45.8)	0.589	0.928 (0.709-1.216)
Females	542 (56.0)	148 (54.2)		
Age				
40-49	387 (40.0)	80 (29.3)	0.051	————
50-59	342 (35.4)	129 (47.3)		
60+	238 (24.6)	64 (23.4)		
Educational level				
Low	439 (45.4)	132 (48.4)	0.387	0.888 (0.679-1.162)
High	528 (54.6)	141 (51.6)		
S/economic level				
Low	341 (35.3)	166 (60.8)	0.000*	0.351 (0.266-0.463)
High	626 (64.7)	107 (39.2)		
Smoking status				
No	375 (38.8)	159 (58.2)	0.000*	0.454 (0.346-0.597)
Yes	592 (61.2)	144 (41.8)		
Glucose level				
Normal level	857 (88.6)	238 (87.2)	0.512	1.146 (0.763-1.720)
Abnormal level	110 (11.4)	35 (12.8)		
Blood pressure				
Normal level	805 (83.2)	221 (81.0)	0.376	1.169 (0.827-1.653)
Abnormal level	162 (16.8)	52 (19.0)		
Dental Follow-up				
≤ 2 times/year	407 (42.1)	120 (44.0)	0.582	0.927 (0.707-1.215)
>2 times/year	560 (57.9)	153 (56.0)		
Tooth brush frequency				
≤ 2 times/daily	389 (40.2)	171 (62.6)	0.000*	0.401 (0.304-0.529)
>2 times/daily	578 (59.8)	102 (37.4)		
Probing pocket depth				
0-3.00 mm	532 (55.0)	122 (44.7)	0.003*	1.514 (1.155-1.983)
≥ 4.0 mm	435 (45.0)	151 (55.3)		
Bleeding on probing				
Absence	544 (56.3)	110 (40.3)	0.000*	1.906 (1.450-2.504)
Presence	423 (43.7)	163 (59.7)		

Table 1. Univariate analysis of the study sample regarding each independent variable examined

Variables in the Equation									
		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
Step 1 ^a	gender	,083	,142	,344	1	,558	,920	,696	1,216
	educ.lev	,043	,127	,115	1	,735	1,044	,814	1,338
	socioec.stat	-,258	,141	10,601	1	,031*	1,581	1,200	2,083
	smok.stat	,863	,143	36,532	1	,000*	2,370	1,791	3,135
	dent.f.up	-,312	,137	5,190	1	,023*	1,367	1,045	1,788
	tooth.brush	-,442	,143	,010	1	,022*	1,714	,967	2,041
	glucose.lev	,147	,162	15,885	1	,082	1,199	1,019	1,824
	blood.press	,205	,141	18,418	1	,077	1,131	0,813	1,714
	Bop	,751	,150	24,935	1	,006*	2,120	1,578	2,847
	Ppd	,505	,146	11,932	1	,001*	2,157	1,244	2,208
	Constant	1,578	,211	55,960	1	,000	,206		
Step 4 ^a	socioec.stat	,455	,131	12,087	1	,001*	1,576	1,220	2,037
	smok.stat	,841	,138	37,037	1	,000*	2,318	1,768	3,040
	tooth.brush	-,331	,133	6,238	1	,013*	1,393	1,074	1,806
	Bop	,755	,147	26,249	1	,000*	2,128	1,594	2,841
	Ppd	,496	,145	11,754	1	,001*	2,042	1,642	2,280
	Constant	1,550	,158	96,685	1	,000	,212		

a. Variable(s) entered on step 1: gender, educ.lev,, socioec.stat, smok.stat, dent.f.up, tooth.brush, glucose.lev, blood.press, Bop, Ppd. (* p-value: statistically significant)

Table 2 Presentation of associations between independent variables and CAL severity according to Enter (first step) and Wald (final step) method of multivariate logistic regression analysis model.

Many epidemiologic studies have revealed that smoking is a crucial risk factor for PD [10, 37,49,50] and various mechanisms have been proposed which are implicated in its pathogenesis and contributes to PD progression [35]. A significant association observed between smoking status and a mean CAL ≥ 3.00 mm in the current study. Similar reports have confirmed the mentioned association [16,17,34,35,38,42,43,51], and have shown that smoking is a significantly high risk factor for CAL severity.

This might be related to the crucial role of smoking in development and progression of periodontitis by affecting host immune responses [49]. However, to date it remains unclear the mechanism which explain how smoking may affect PD. It is possible that genetic susceptibility and genetic polymorphisms may explain the mentioned influence [13]. Cigarette smoking is a modifiable environmental risk factor most associated with the progression of PD [20], as smokers are 2 to 7 times more likely to suffer from periodontitis, whereas heavy smokers are twice as likely to present CAL and ABL than light smokers [43]. Cross-sectional and longitudinal surveys have recorded that the risk of developing PD as measured by CAL and ABL increases in heavy smokers [52]. CAL severity was found significantly higher among individuals who brushed their teeth irregularly (≤ 2 times/daily) than those who brushed their teeth in a regular way (>2 times/daily) in the present report. This observation was in agreement with previous studies indicating that a higher rate of individuals who did not brush their teeth regularly had poor oral hygiene and had a significant association with pocket formation and CAL severity [34,38], whereas only in one study was found that dental care was not significantly associated with CAL progression [42].

Tooth brushing is essential for periodontal health maintenance as reduces accumulation of dental plaque and in turn prevents gingivitis and periodontitis [52]. The current study showed that individuals who do not follow a regular dental check-up (>2 times/year) had more severe CAL compared with those who visit their dentist regularly, finding that was in agreement with the findings of previous studies [17,47], except for one study which did not confirm such a finding [54]. DM is another modifiable environmental risk factor for PD indices as it can be controlled but not be treated. The current study showed that DM patients were found to be insignificantly associated with CAL severity, observation that was in agreement with those from previous reports [40, 42]. However, similar studies revealed that individuals with a DM history were statistically significant associated with CAL severity [20,23,35,55].

DM can cause CAL and the treatment of PD may improve clinical symptoms and signs of DM, suggesting that a cross-susceptibility exists between PD and DM [56]. DM, also increases the susceptibility to inflammatory tissue destruction and leads to CAL by reducing neutrophil functions or by accelerating the synthesis of advanced glycosylation and products [57]. Previous reports suggest that this association is considered to be bidirectional, e.g. DM is a risk factor for periodontitis, and periodontitis is a possible factor for DM [58]. The current study recorded no association between individuals with hypertension and the healthy ones regarding CAL severity, finding that was in line with previous reports [23,59,60]. In contrary, other studies recorded a significant association between high blood pressure and CAL severity [35,61-64]. Clinical hypertension and deteriorated arterial blood pressure regulation are systemic conditions that are associated with immune function and inflammation and may worsen chronic periodontitis [65].

The current study has some limitations which should be taken into account when interpreting the outcomes. Some independent epidemiological variables were collected through questionnaires which, even though commonly used in research, can introduce biases, such as self-reported tooth brushing frequency or SES, which was difficult to reconcile with clinical findings.

Conclusions

Within the limitations of this study, it can be concluded that, a low prevalence of severe CAL among adults. Greek population was recorded. All risk factors mentioned, low SES, smoking, irregular daily tooth brushing, BOP and deeper pocket depth, were strongly associated with the prevalence, extent, and severity of CAL. It is obvious that further studies with larger sample including adolescents and adult Greek population in different geographic locations are recommended.

References

1. Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ (2012) CDC Periodontal Disease Surveillance workgroup: Prevalence of periodontitis in adults in the United States: 2009 and 2010. *J Dent Res.* 91:914-920.
2. Bouchard P, Boutouyrie P, Mattout C, Bourgeois D (2006) Risk assessment for severe clinical attachment loss in an adult population. *J Periodontol.* 77:479-489.
3. Haffajee AD, Socransky SS (1994) Microbial etiological agents of destructive periodontal diseases. *Periodontol* 2000. 5: 78-111.
4. Brown LJ, L e H (1993) Prevalence, extent, severity and progression of periodontal disease. *Periodontol* 2000 2: 57-71.
5. Kornman KS (1987) Nature of periodontal diseases: Assessment and diagnosis. *J Periodontal Res.* 22:192-204.
6. Schaefer AS, Richter GM, Nothnagel M, Laine ML, Ruhling A, et al. (2010) A 3' UTR transition within DEFB1 is associated with chronic and aggressive periodontitis. *Genes Immun.* 11: 45-54.

7. Kinane DF, Podmore M, Murray MC, Hodge PJ, Ebersole J (2001) Etiopathogenesis of periodontitis in children and adolescents. *Periodontol* 2000. 26:54-91.
8. Kinane DF (2001) Causation and pathogenesis of periodontal disease. *Periodontol* 2000. 25:8-20.
9. Shaju Jacob P (2011) Measuring periodontitis in population studies: a literature review. *Rev Odonto Cienc.* 26: 346-354.
10. Albandar JM (2002) Global risk factors and risk indicators for periodontal diseases. *Periodontol* 2000. 29:177-206.
11. Tanner ACR, Maiden MF, Macuch PJ, Murray LL, Kent RL Jr (1998) Microbiota of health, gingivitis, and initial periodontitis. *J Clin Periodontol.* 25:85-98.
12. Schaefer AS, Richter GM, Nothnagel M, Manke T, Dommisch H, et al. (2010) A genome wide association study identifies GL T6D1 as a susceptibility locus for periodontitis. *Hum Mol Genet.* 19:553-562.
13. Genco RJ (1996) Current view of risk factors for periodontal diseases. *J Periodontol.* 67:1041 -1049.
14. Borrell LN, Crawford ND (2012) Socioeconomic position indicators and periodontitis: examining the evidence. *Periodontology* 2000. 58: 69-83.
15. Genco RJ, Borgnakke WS (2013) Risk factors for periodontal disease. *Periodontology* 2000. 62: 59-94.
16. Hyman JJ, Reid BC (2003) Epidemiologic risk factors for periodontal attachment loss among adults in the United States. *J Clin Periodontol.* 30:230-237.
17. Susin C, Dalla Vecchia CF, Oppermann RV, Haugejorden O, Albandar JM (2004) Periodontal attachment loss in an urban population of Brazilian adults: effect of demographic, behavioral, and environmental risk indicators. *J Periodontol.* 75:1033-1041.
18. Akhter R, Hannan MA, Okhubo R, Morita M (2005) Relationship between stress factor and periodontal disease in a rural area population in Japan. *Eur J Med Res.* 10:352-357.
19. Tanner AC, Kent R Jr, Van Dyke T, Sonis ST, Murray LA (2005) Clinical and other risk indicators for early periodontitis in adults. *J Periodontol.* 76:573-581.
20. Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, et al. (1994) Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol.* 65(3):260-267.
21. Grossi SG, Genco RJ, Machtei EE, Ho AW, Koch G, et al. (1995) Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *J Periodontol.* 66(1):23-29.
22. Albandar JM, Streckfus CF, Adesanya MR, Winn DM (2000) Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *J Periodontol.* 71(12):1874-1881.
23. Chrysanthakopoulos NA (2018) Epidemiological risk factors for periodontal pockets and clinical attachment loss among Greek adults. *Dent Craniofac Res.* 1(3):114.
24. Holtfreter B, Schutzhld S, Kocher T (2014) Is periodontitis prevalence declining? A review of the current literature. *Cur Oral Health Reports.* 1:251-261.
25. Savage A, Eaton KA, Moles DR, Needleman I (2009) A systematic review of definitions of periodontitis and methods that have been used to identify this disease. *J Clin Periodontol.* 36: 458-467.
26. World Health Organization: Oral health surveys: basic methods. (4thEdn). Geneva: WHO. 1997:47p.
27. Lwanga SK, Lemeshow S. Sample size determination in health studies. A practical manual. Geneva: WHO; 1991.
28. Machuca G, Segura-Egea JJ, Jimenez-Beato G, Lacalle JR, Bullón P (2012) Clinical indicators of periodontal disease in patients with coronary heart disease: A10 years longitudinal. *Med Oral Patol Oral Cir Bucal.* 17: e569-574.
29. Molloy J, Wolff LF, Lopez-Guzman A, Hodges JS (2004) The association of periodontal disease parameters with systemic medical conditions and tobacco use. *J Clin Periodontol.*31: 625-632.
30. Cutress TW, Ainamo J, Sardo-Infrii J (1987) The community periodontal index of treatment needs (CPITN) procedure for population groups and individuals. *Int Dent J.* 37:222-233.
31. Armitage GC (1999) Development of a classification system for periodontal diseases and conditions. *Ann Periodontol.*4:1-6.
32. Machtei EE, Christersson LA, Grossi SG, Dunford R, Zambon JJ, et al (1992) Clinical criteria for the definition of "established periodontitis". *J Periodontol.* 63:206-214.
33. Vonneilich N, Jöckel KH, Erbel R, Klein J, Dragano N, et al (2011) Does socioeconomic status affect the association of social relationships and health? A moderator analysis. *Int J Equity Health* 10:43.

34. Amran AG, Alhadj MN, Amran AN (2016) Prevalence and Risk Factors for Clinical Attachment Loss in Adult Yemenis: A Community-Based Study in the City of Dhamar. *Am J Health Res.* 4(3): 56-61.
35. Rhee GB, Ji S, Ryu JJ, Lee JB, Shin C, et al. (2011) Risk assessment for clinical attachment loss of periodontal tissue in Korean adults. *J Adv Prosthodont.* 3:25-32.
36. Dye BA, Tan S, Smith V, Lewis BG, Barker LK, et al. (2007) Trends in oral health status: United States, 1988-1994 and 1999-2004. *Vital Health Stat* 11. :1-92.
37. Holtfreter B, Schwahn C, Biffar R, Kocher T (2009) Epidemiology of periodontal diseases in the Study of Health in Pomerania. *J Clin Periodontol.* 36: 114-123.
38. Rao SR, Thanikachalam S, Sathiyasekaran BWCS, Vamsi L, Balaji TM, et al (2014). Prevalence and Risk Indicators for Attachment Loss in an Urban Population of South India: *Oral Health Dent Manag.* 13: 60-64.
39. Bourgeois D, Bouchard P, Mattout C (2007) Epidemiology of periodontal status in dentate adults in France, 2002-2003. *J Periodontal Res.* 42:219-227
40. Morris AJ, Steele J, White DA (1998) The oral cleanliness and periodontal health of UK adults in 1998. *Br Dent J.* 191:186-192.
41. Gamonal J, Mendoza C, Espinoza I, Muñoz A, Urzúa I, et al. (2010) Clinical attachment loss in Chilean adult population: First Chilean National Dental Examination Survey. *J Periodontol* 81(10):1403-1410.
42. Haas AN, Wagner MC, Oppermann RV, Rösing CK, Albandar JM, et al. (2014) Risk factors for the progression of periodontal attachment loss: a 5-year population-based study in South Brazil. *J Clin Periodontol.* 41(3):215-223.
43. Grossi SG, Genco RJ, Machtei EE, Ho AW, Koch G, et al. (1995) Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *J Periodontol.* 66:23-29.
44. Christensen LB, Petersen PE, Krstrup U, Kjølner M (2003) Self-reported oral hygiene practices among adults in Denmark. *Community Dent Health.* 20:229-235.
45. Torrungruang K, Tamsailom S, Rojanasomsith K, Sutdhibhisal S, Nisapakultorn K, et al. (2005) Risk indicators of periodontal disease in older Thai adults. *J Periodontol.* 76:558-565.
46. Borrell LN, Burt BA, Warren RC, Neighbors HW (2006) The role of individual and neighborhood social factors on periodontitis: the third National Health and Nutrition Examination Survey. *J Periodontol.* 77:444-453.
47. Ababneh KT, Abu Hwaj ZMF, Khader YS. (2012) Prevalence and risk indicators of gingivitis and periodontitis in a Multi-Centre study in North Jordan: across sectional study. *BMC Oral Health.* 12:1.
48. Ramírez Maya JC, Lopera NS, López AP, Agudelo-Suárez AA, Botero JE (2017) Periodontal disease and its relationship with clinical and sociodemographic variables in adult patients treated in a service/ teaching institution. *Rev Odontol Mexicana.* 21: e160-e167.
49. Palmer RM, Wilson RF, Hasan AS, Scott DA (2005) Mechanisms of action of environmental factors-tobacco smoking. *J Clin Periodontol.* 32:180-195.
50. Vouros ID, Kalpidis CDR, Chadjipantelis T, Konstantinidis AB (2009) Cigarette smoking associated with advanced periodontal destruction in a Greek sample population of patients with periodontal disease. *Intern Acad Periodontol.* 11: 250-257.
51. Haffajee AD, Socransky SS (2001) Relationship of cigarette smoking to attachment level profiles. *J Clin Periodontol.* 28: 283- 295.
52. Tomar SL, Asma S (2000) Smoking-attributable periodontitis in the United States; findings from NHANES III. *National Health and Nutrition Examination Survey.* *J Periodontol.* 71:743-751.
53. Joshipura KJ, Kent RL, De Paola PF (1994) Gingival recession: intraoral distribution and associated factors. *J Periodontol.* 65: 864-871.
54. Ogawa H, Yoshihara A, Hiroto T, Ando Y, Miyazaki H (2002) Risk factors for periodontal disease progression among elderly people. *J Clin Periodontol.* 29:592-597.
55. Meng H (2007) Association between periodontitis and diabetes mellitus. *Beij Da Xue Xue Bao.* 39:18-20.
56. Preshaw PM, Foster N, Taylor JJ (2007) Cross-susceptibility between periodontal disease and type 2 diabetes mellitus: an immunobiological perspective. *Periodontol* 2000.45:138-157
57. Nishimura F, Iwamoto Y, Soga Y (2007) The periodontal host response with diabetes. *Periodontol* 2000. 43:245-253.
58. Hong M, Kim HY, Seok H, Yeo CD, Kim YS, et al. (2016) Prevalence and risk factors of periodontitis among adults with or without diabetes mellitus. *Korean J Intern Med.* 31:910-919.

59. Rodríguez-Iturbe B, Pons H, Quiroz Y, Johnson RJ (2014) The Immunological Basis of Hypertension. *Am J Hypertens.* 27:1327-1337.
60. Nesse W, Dijkstra PU, Abbas F, Spijkervet FKL, Stijger A, et al.(2010) Increased prevalence of cardiovascular and autoimmune diseases in periodontitis patients: a cross-sectional study. *J Periodontol.* 81:1622-1628.
61. Tsakos G, Sabbah W, Hingorani AD, Netuveli G, Donos N, et al. (2010) Is periodontal inflammation associated with raised blood pressure? Evidence from a National US survey. *J Hypertens.* 28:2386-2393.
62. Holmlund A, Holm G, Lind L (2006) Severity of periodontal disease and number of remaining teeth are related to the prevalence of myocardial infarction and hypertension in a study based on 4,254 subjects. *J Periodontol.* 77:1173-1178.
63. Iwashima Y, Kokubo Y, Ono T, Yoshimuta Y, Kida M, et al (2014) Additive interaction of oral health disorders on risk of hypertension in a Japanese urban population: the Suita Study. *Am J Hypertens.* 27:710-719.
64. Martin-Cabezas R, Seelam N, Petit C, Agossa K, Gaertner S, et al. (2016) Association between periodontitis and arterial hypertension: A systematic review and meta-analysis. *Am. Heart J.* 180:98-112.
65. Rivas-Tumanyan S, Spiegelman D, Curhan GC, Forman JP, Joshipura KJ (2012) Periodontal disease and incidence of hypertension in the health professionals follow-up study. *Am J Hypertens.* 25:770-776.

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