Abstracts of articles published in important Implantology, Prosthodontics and Periodontics journals from around the world

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Effect of the timing of restoration on implant marginal bone loss: a systematic review

Suarez F, Chan HL, Monje A, Galindo-Moreno P, Wang HL

J Periodontol. 2013 Feb;84(2):159-69

Background: The advancement in implant dentistry has allowed shortened treatment time by restoring the implants earlier. Whether the timing of restoration has an impact on implant marginal bone level has not been systematically analyzed. The aim of this study is to compare marginal bone loss (MBL) between implants that were restored with the following protocols: 1) immediate restoration/loading (IR/L); 2) early loading (EL); and 3) conventional loading (CL). **Methods:** An electronic literature search from three databases (until November 2011) and a hand search in implant-related journals were conducted. Clinical human studies in English language that had reported a comparison of MBL between implants with IR/L, EL, or CL with ≥12-month follow-up were included. In addition, the minimal number of implants had to be 10 for each group. Implants with both immediate placement (IP) and delayed placement (DP) were included and analyzed separately. An assessment of the publication bias for the included randomized clinical trials (RCTs) was performed. **Results:** The initial search resulted in 1,640 articles, of which 27 articles in full text were further evaluated for eligibility. Finally, 11 studies (eight RCTs, two controlled clinical trials, and one retrospective study) were qualified and classified into four groups: 1) IR/L + DP versus CL + DP (n = 6 articles); 2) IR + DP versus EL + DP (n = 2 articles); 3) EL + DP versus CL + DP (n = 1 article); and (4) IL + IP versus CL + IP (n = 2 articles). A meta-analysis performed for group 1 showed 0.09 mm (95% confidence interval = -0.27 to 0.09 mm) difference in the mean MBL, favoring the IR/L protocol but without significant difference (P = 0.33). No significant difference in MBL was found for groups 2 through 4 after adjusting for the implant placement level. The eight RCTs were determined to be at moderate-to-high risk of publication bias. **Conclusions:** This meta-analysis does not show an effect of the timing of restorations on implant MBL. The selection of restoration protocols should be based on factors other than MBL.

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Association of periodontitis with the risk of oral leukoplakia

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Oral Oncol. 2012 Sep;48(9):859-63

Background: Oral leukoplakia is an oral lesion suspected to be of premalignant character. Besides smoking and alcohol, the risk factors for the development of this oral lesion are still less identified. The purpose of this study was the search for a possible influence of periodontitis on the risk of leukoplakia. **Methods:** We used data from 4233 subjects (2116 women and 2117 men) who were recruited for the population-based Study of Health in Pomerania (SHIP) and finished a standard medical and dental examination. One hundred two-three cases with oral leukoplakia were 1:2 age and sex-matched with 246 healthy control subjects. Measures of bleeding on probing and clinical attachment loss were related to oral leukoplakia. **Results:** We found increased periodontal measures in subjects with leukoplakia. Adjusting for risk factors and possible confounders revealed a periodontitis-related dose-dependent increase in the probability of having oral leukoplakia. Odds ratios adjusted for socioeconomic factors and smoking computed for the second, third and fourth quartiles of clinical attachment loss were OR=1.7 (0.6-5.0), 3.3 (0.8-13.1) and 5.3 (1.2-22.7), respectively. For bleeding on probing the respective odds ratios were OR=2.0 (0.8-4.90), 2.9 (1.1-7.8) and 3.8 (1.5-9.8), respectively. Measures of systemic inflammation and of lipid metabolism were important cofactors attenuating these figures. While smoking is a risk factor of leukoplakia, oral hygiene is protective. In a follow-up survey, the leukoplakia subjects had lost more teeth than their counterparts (p=0.043). **Conclusion:** Periodontitis increases the risk of oral leukoplakia and, therefore, the risk of mucosal lesions predisposing to oral cancers.

Periodontal disease, Porphyromonas gingivalis serum antibody levels and orodigestive cancer mortality

Ahn J, Segers S, Hayes RB

Carcinogenesis. 2012 May;33(5):1055-8

Periodontitis, the progressive loss of the alveolar bone around the teeth and the major cause of tooth loss in adults, is due to oral microorganisms, including Porphyromonas gingivalis. Periodontitis is associated with a local overly aggressive immune response and a spectrum of systemic effects, but the role of this condition in orodigestive cancers is unclear. We prospectively examined clinically ascertained

periodontitis (N = 12,605) and serum IgG immune response to P.gingivalis (N = 7852) in relation to orodigestive cancer mortality among men and women in the National Health and Nutrition Examination Survey III. A detailed oral health exam was conducted from 1988 to 1994 in survey Phases I and II, whereas serum IgG for P.gingivalis was measured from 1991 to 1994 in Phase II only. One hundred and five orodigestive cancer deaths were ascertained through 31 December 2006. Periodontitis (moderate or severe) was associated with increased orodigestive cancer mortality [relative risks (RR) = 2.28, 95% confidence interval (CI) = 1.17-4.45]; mortality risks also increased with increasing severity of periodontal disease (P trend = 0.01). Periodontitis-associated mortality was in excess for colorectal (RR = 3.58; 95% CI = 1.15-11.16) and possibly for pancreatic cancer (RR = 4.56; 95% CI = 0.93-22.29). Greater serum P.gingivalis IgG tended to be associated overall with increased orodigestive cancer mortality (P trend = 0.06); P.gingivalis-associated excess orodigestive mortality was also found for healthy subjects not exhibiting overt periodontal disease (RR = 2.25; 95% CI = 1.23-4.14). Orodigestive cancer mortality is related to periodontitis and to the periodontal pathogen, P.gingivalis, independent of periodontal disease. Porphyromonas gingivalis is a biomarker for microbe-associated risk of death due to orodigestive cancer.

Periodontitis and diabetes associations with measures of atherosclerosis and CHD

Southerland JH, Moss K, Taylor GW, Beck JD, Pankow J, Gangula PR, Offenbacher S

Atherosclerosis. 2012 May;222(1):196-201

Objective: Diabetes has been linked with more severe periodontal disease and with coronary heart disease (CHD). The purpose of this study was to determine if periodontal infection was a significant modifier in the risk that diabetes poses for increased carotid artery intimal-medial wall thickness (IMT) and more advanced atheroma lesions as reflected in atherosclerotic plaque calcification measured by acoustic shadowing. **Methods and Results:** Comparisons for analyses of cardiovascular outcomes were performed based upon periodontitis and diabetes status. Periodontitis was measured using pocket depth and attachment loss at six sites per tooth. Cross-sectional data on 6048 persons aged 52-74 years were obtained from the Dental Atherosclerosis Risk in Communities Study. Participants without diabetes (n=5257) were compared to those with diabetes (n=791). Dependent variables were thick IMT (>1 mm), presence of acoustic shadowing, and prevalent CHD. All models were adjusted for the following covariates: gender, age, race/center, LDL and HDL

cholesterol, BMI, triglycerides, hypertension, smoking, income and education. For multivariate model building, all non-normally distributed variables were transformed and multivariable logistic regression analyses were performed to evaluate the relationship between periodontal infection, diabetes, and cardiovascular outcomes. Individuals with diabetes and with severe periodontitis were found to be significantly more likely to have IMT>1 mm [OR=2.2, (1.4-3.5)], acoustic shadowing [OR=2.5, (1.3-4.6)], and CHD [OR=2.6, (1.6-4.2)] compared to those without diabetes or periodontal disease. **Conclusion:** Results from this study suggest that among people with diabetes, periodontal disease may increase the likelihood of subclinical atherosclerotic heart disease and CHD.

Periodontal disease and the oral microbiota in new-onset rheumatoid arthritis

Scher JU, Ubeda C, Equinda M, Khanin R, Buischi Y, Viale A, et al

Arthritis Rheum. 2012 Oct;64(10):3083-94

Objective: To profile the abundance and diversity of subgingival oral microbiota in patients with never-treated, new-onset rheumatoid arthritis (RA). **Methods:** Periodontal disease (PD) status, clinical activity, and sociodemographic factors were determined in patients with new-onset RA, patients with chronic RA, and healthy subjects. Multiplexed-454 pyrosequencing was used to compare the composition of subgingival microbiota and establish correlations between the presence/abundance of bacteria and disease phenotypes. Anti-Porphyromonas gingivalis antibody testing was performed to assess prior exposure to the bacterial pathogen P gingivalis. **Results:** The more advanced forms of periodontitis were already present at disease onset in patients with new-onset RA. The subgingival microbiota observed in patients with new-onset RA was distinct from that found in healthy controls. In most cases, however, these microbial differences could be attributed to the severity of PD and were not inherent to RA. The presence and abundance of P gingivalis were also directly associated with the severity of PD and were not unique to RA. The presence of P gingivalis was not correlated with anti-citrullinated protein antibody (ACPA) titers. Overall exposure to P gingivalis was similar between patients with new-onset RA and controls, observed in 78% of patients and 83% of controls. The presence and abundance of Anaeroglobus geminatus correlated with the presence of ACPAs/rheumatoid factor. Prevotella and Leptotrichia species were the only characteristic taxa observed in patients with new-onset RA irrespective of PD status. **Conclusion:** Patients with new-onset RA exhibited a high prevalence of PD at disease onset, despite their young age and paucity of smoking history. The subgingival microbiota profile in patients with new-onset RA was similar to that in patients with chronic RA and healthy subjects whose PD was of comparable severity. Although colonization with P gingivalis correlated with the severity of PD, overall exposure to P gingivalis was similar among the groups. The role of A geminatus and Prevotella/Leptotrichia species in this process merits further study.