

Cairo F., Gaeta C., Dorigo, W. et al. Periodontal pathogens in atheromatous plaques. A controlled clinical and laboratory trial. J Periodont Res 2004; 39:442-6. (25 Refs)

Purpose: 1) to ascertain the presence of periodontal bacteria DNA [A. a, P. g, F. nucleatum, P. intermedia and Tannerella forsythensis (formerly B. forsythus)] in carotid atheromatous plaques in dentate and edentulous cardiovascular patients and 2) to assess the concomitant presence of the same bacterial DNA, if any, in periodontal pockets and in carotid atheroma in the same dentate patient.

Materials and Methods: 19 dentate patients (14 males and 5 females, with a mean age of 71.37+6.14 years) as test group, and 21 edentulous (> 2 years) patients (15 males and 6 females, with a mean age of 73.33+6.11 years) as control group are studied. All patients were identified as candidates for carotid endarterectomy. After complete periodontal examination, subgingival plaque was collected from the deepest pocket, and after supragingival scaling, samples were also collected with a sterile paper tip point inserting in the pocket. Atheromatous plaques were harvested from all patients during surgery. Thereafter, DNA extraction was carried out from both subgingival and atheromatous plaque samples. Extracted DNA was utilized for PCR method to detect bacterial species.

Findings and Conclusions: In clinical examination, 11 patients were smokers. The mean tooth loss was 13.0+6.25, mean clinical attachment level was 4.69+1.58, mean PD was 2.87+0.82, mean plaque score was 75.95%+26.81, and mean BOP was 58.85+27.15. The detected DNA from subgingival plaque were, T. forsythensis; 79% of patients, F. nucleatum in 63%, P. intermedia in 53%, P. g. in 37% and A. a.in 5% of samples. No statistically significant association between the periodontal pathogens was found. The carotid specimens of all 40 patients revealed evidence of severe atherosclerosis, but no DNA of periodontal bacteria was detected by PCR in any of the carotid samples in either patient group. The result tends to exclude a direct correlation between the detection of periodontal bacteria DNA in oral lesions and its concomitant presence in carotid atheroma, and do not support the previous findings that reported a frequent presence of periodontal pathogens in carotid atheroma lesions.

Saygun I, Kubar A, Ozdemir A, Slots J. Periodontitis lesions are a source of salivary cytomegalovirus and Epstein-Barr virus. J Periodont Res 2005; 40: 187-91. (40 Refs)

Purpose: The purpose of the study was to test the hypothesis that inflamed periodontium is a source of salivary herpes viruses, using Human cytomegalovirus (HCMV) and Epstein-Barr virus (EBV) periodontal and salivary counts, and to determine the effect of periodontal therapy on salivary herpes virus counts.

Materials and Methods: 20 periodontitis patients, 21 to 56 years old, participated in the study. All study patients were systemically healthy. 9 individuals younger than 35 years old were assigned the diagnosis of aggressive periodontitis and 11 individuals 35-56 years old were diagnosed chronic periodontitis. Periodontal therapy consisted of OHI, S&RP, and modified Widman flap surgery of all deep periodontal sites. Virological samples were obtained prior to definitive periodontal treatment. Immediately prior to surgery, unstimulated whole saliva was collected in a glass cylinder, followed by a periodontal pocket sample from the single deepest probing site in the dentition (6-10mm). After removing supragingival plaque, a sterile periodontal curette was inserted to the bottom of the test pocket and subgingival material was removed by a single stroke. A gingival tissue specimen was harvested in conjunction with the surgery. After raising buccal and lingual flaps, a total of 36-50 mg of pocket epithelium, underlying connective tissue, and granulation tissue was removed by a periodontal curette from the periodontal lesion. At 3 months posttreatment, 2 aggressive and 5 chronic periodontitis patients were available for virological sampling of the subgingival sites and saliva. A 5'-nuclease (TaqMan) real-time PCR assay was used to identify and quantify genomic copies of HCMV and EBV. The relationship between subgingival, gingival tissue, and salivary herpesvirus counts was evaluated using the non-parametric Spearman's and Kendall's tau rank correlation coefficient.

Findings and Conclusions: At baseline, most of the 20 subjects revealed HCMV DNA and EBV DNA in periodontal samples and in saliva. Significant positive correlations were identified between gingival tissue and salivary levels of HCMV and EBC. Periodontal pocket depth at sample sites correlated with EBV DNA counts in saliva. In the aggressive periodontitis subgroup, HCMV DNA counts in gingival tissue were positively correlated with HCMV DNA counts in saliva. The chronic periodontitis patients showed positive correlation between periodontal pocket depth at sample sites and EBV DNA counts in saliva. Periodontal therapy caused a reduction in full-mouth average periodontal pocket depth, in plaque index, and in gingival index. Following treatment, HCMV DNA counts decreased 37.5x in subgingival sites and 64.6x in saliva. EBV DNA counts decreased by 5.7x in subgingival sites and 12.9x in saliva.

Periodontitis lesions are sites for HCMV and EBV oral persistence and constitute an important source of infective herpesvirus in saliva. Reducing the viral load in saliva may diminish the risk of viral transmission among individuals in close contact.

Li-Jane Ling, Chuan-Chen Ho, Ching -Yi Wu et al. Association between human herpes viruses and the severity of periodontitis. J Periodontol 2004;75:1479-85. (45 Refs)

Purpose: The aim of this study was to evaluate the relationship between herpes viruses and the severity of periodontitis.

Materials and Methods: The study population consisted of 20 patients who were randomly selected from patients attending the Dental department of the Taipei Veterans General Hospital. The patients ranged in age from 20 to 64 years and were systemically healthy, did not have any periodontal treatment including scaling and root planning within the 6 months prior to the study and female patients were not pregnant or breast feeding. The six Ramjford teeth (3,9,12, 19, 25, and 28) were selected to evaluate the periodontal status of the participants. Parameters measured included; the Plaque Index, Gingival Index, Probing depth and the position of the gingival margin relative to the CEJ. Following probing the presence of bleeding on probing of the sites within 30 seconds was also recorded. After periodontal examination one sample of subgingival plaque was collected from each examined tooth by inserting a size 30 paper point into the gingival sulcus or periodontal pocket for 20 seconds. The paper points were placed in microfuge tubes containing TE buffer and stored at -80 degree C until required for analysis. The thawed subgingival periodontal samples were used to recover viral nucleic acids by preferential binding to silica particles in the presence of high concentration of guanidium thiocyanate as described by Parra and Slots. Primers and the experimental conditions for the viral nested-Polymerase Chain Reaction were performed as described by Parra and Slots and Aitken et al.

Findings and Conclusions: When all 120 sites were examined data revealed that the plaque index was significantly associated with probing depth. In addition, the gingival index was positively correlated with bleeding on probing, probing depth and attachment loss. Bleeding on probing was also associated with clinical attachment loss. The presence of HSV, HCMV or EBV -1 was equally distributed between both sexes and further showed no age predilection. When the total sites were examined, the prevalence of HSV was significantly higher in the subgroups that had a lower plaque index. The prevalence of HCMV was also significantly higher in the subgroup that had a lower plaque index. The prevalence of HSV was highly associated in the subgroups that had higher gingival index, positive bleeding on probing, deeper probing depth or higher clinical attachment loss. No significant association was observed between the prevalence of HCMV and gingival index, bleeding on probing, probing depth, or clinical attachment loss. The prevalence of EBV-1 was significantly higher in the subgroup with higher probing depth. There was no significant association between the prevalence of EBV-1 and the other clinical parameters. This study thus demonstrates that HSV is related to the severity of periodontal disease in terms of clinical attachment loss.