PERIODONTAL DISEASE ACTS SYNERGISTICALLY WITH SYSTEMIC INFLAMMATION ON ASYMMETRIC DIMETHYLARGININE IN HYPERTENSIVE PATIENTS: EVIDENCE OF A DOSE-DEPENDENT EFFECT

ACC Poster Contributions
Ernest N. Morial Convention Center, Hall F
Monday, April 04, 2011, 3:30 p.m.-4:45 p.m.

Background: Chronic periodontal disease elicits a low-grade systemic inflammatory response, while it has also been shown to impair endothelial function. It is uncertain whether, in the setting of hypertension a dose-dependent association of periodontal disease indexes (PDIs) with vascular damage beyond systemic inflammation exists.

Methods: We studied 108 - aged 52±9 years - untreated hypertensive subjects (24h systolic/diastolic blood pressure [BP] 131±11/83±9mmHg) with diverse severity of periodontal disease (i.e. mean clinical loss of attachment, maximum probe depth and gingival index). Subjects underwent office and ambulatory BP measurements, echocardiography and periodontal examination; From fasting venous blood samples we assessed metabolic profile, and measured asymmetric dimethylarginine (ADMA) and high sensitivity C reactive protein (hsCRP) levels.

Results: With respect to the median of hsCRP and ADMA (1.79 mg/l and 0.81 μmol/l respectively) the study population was divided into four groups: low-ADMA/low-hsCRP (n=30), low-ADMA/high-hsCRP (n=27), high-ADMA/low-hsCRP (n=21) and high-ADMA/high-hsCRP (n=30). The high-ADMA/high-hsCRP group resulted significantly older compared to both low-ADMA/low-hsCRP and high-ADMA/high-hsCRP groups, while high compared to low-ADMA groups demonstrated increased low-density lipoprotein cholesterol. PDIs were increased in those with high compared with those with low-hsCRP, while the addition of high-ADMA contributed significantly to this finding. After adjustment for confounders, high-ADMA/high-hsCRP was significantly associated - by means of adjusted z-scores - with mean clinical loss of attachment, maximum probe depth and gingival index by 10.33, 8.84 and 2.74 times more often with respect to the low-ADMA/low-hsCRP pattern.

Conclusion: PDIs are associated in a dose-dependent manner with the molecular surrogate of endothelial dysfunction, namely ADMA, in untreated hypertensives and increased systemic inflammation further contributes to that phenomenon.