Does periodontitis cause heart disease?

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Several studies investigating the relation between infections—including dental infections—and various clinical manifestations of atherosclerotic vessel disease have been published during the last decade. The topic has proved difficult to study and this is especially true for dental infections, as they share several common etiologic factors with e.g. coronary heart disease (CHD). These include smoking, low social class and unfavourable health care practices of the individual. Two studies published in this issue address the relation between periodontitis and CHD.1,2

Buhlin et al. compared 50 patients with severe periodontitis with 46 non-periodontal individuals. Absence of CHD was verified by medical history and exercise test. A wide array of lipoproteins and inflammatory parameters were determined in the participants. Individuals with periodontitis had significantly higher levels of circulating monocytes and C-reactive protein (CRP) and lower levels of HDL-cholesterol, IgA anti-Hsp60 and anti-Hsp65 antibodies in comparison with the controls. In contrast, no differences between patients and controls were observed for total cholesterol, haptoglobin, elastase, IL-6, TNFα receptor-1 or α-1-antitrypsin levels. In a multivariate analysis adjusting for the effect of sex, age and smoking, body mass index (BMI) and CRP were significantly associated with periodontitis.

Persson et al. compared 80 patients surviving acute myocardial infarction with controls matched for age, sex, socio-economic status and smoking. The study was specifically designed to have as good as possible matching of the controls for confounding factors. The investigators used receiver operator characteristic (ROC) curves to analyse the strength of association between periodontitis and CHD for different levels of alveolar bone loss assessed radiographically. In a multivariate analysis adjusting for the effect of sex, age and smoking, body mass index (BMI) and CRP were significantly associated with periodontitis.

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As these two studies were cross sectional, they are unable to prove a causal relation between periodontitis and CHD. Yet they provide valuable new information about this difficult topic.

The study of Buhlin et al. suggests that periodontitis, once considered a purely local disease, may cause systemic inflammation and lipid changes known to increase the risk of CHD. In their material, the levels of HSP antibodies were significantly lower in patients with periodontitis. The authors hypothesise that inability to produce these antibodies might promote systemic inflammation and thus increase CHD risk in these individuals. However, when BMI and CRP were taken into account, the changes in lipoproteins and inflammatory markers no longer had significant association with periodontitis. Furthermore, earlier studies have shown that coronary atherosclerosis is associated with increases in the levels of these antibodies.3

The study of Persson et al. suggests that radiographically assessed alveolar bone loss represents the 'accumulated effects' of periodontitis and it is the best way to assess the severity of periodontitis in this kind of studies. This is important in large studies, because radiographic assessment is much easier to obtain than thorough clinical examination, the assessment can be done in a blinded fashion and several independent observers can be used.

A causal relation between infection-periodontitis, Chlamydia pneumoniae or other—and CHD has not been firmly established. The existing data, however, certainly warrants further studies in this field. These studies should take into account what we have learned so far. For example, there is data strongly suggesting that infections act in concert with other CHD risk factors like lipids4 and inflammation5 in increasing CHD risk. Differences in this kind of factors between groups of individuals compared in earlier studies may have contributed to the discrepant results obtained. Furthermore, infections seldom appear alone. The 'infectious burden', defined as the number of different infections that can be detected with serological and other methods in an individual, seems to be
a stronger determinant of CHD risk than any single infection. Therefore studies focusing on one type of infection only will probably not find the answer. Taken together — back to work!

References