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Dental plaque as a possible risk factor for general disease – results from longitudinal studies

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Hippocrates held a belief that the body must be treated as a whole, and in his millennium lecture Professor Harald Löe asked the question, 'Half a century of plaque removal, what's next?' There is growing evidence implicating chronic inflammation and infection, as we see in gingivitis and periodontitis, to be risk factors for coronary heart disease, other systemic diseases and premature death. Inflammation is a key feature in many chronic diseases. Periodontal disease is characterized by chronic infection and inflammation in the periodontal tissue(1). Periodontal disease is initiated by biofilm or bacteria on the teeth, which trigger an immune inflammatory response in the adjacent host tissue (2). In some individuals, the reaction to bacteria may lead to an excessive host response resulting in general inflammation response (3). In this presentation the possible involvement of dental plaque, gingival inflammation, periodontal disease in the development of early carotid lesions, systemic diseases as well as premature death will be discussed. Also the role of oral bacteria in the etiology of systemic diseases will be discussed; the periodontal pocket bacteria frequently penetrate the gingival epithelial barrier. The systemic diseases discussed today include coronary heart disease, diabetes, aspiration pneumonia, pregnancy complications, premature death and stress related depression (4–8).

In recent years the hypothesis has regained considerable attention that periodontal disease representing a chronic Gram-negative infection may have consequences, beyond the periodontal tissues themselves. Evidence has been brought forward suggesting significant associations between periodontal diseases and other non-oral conditions, including cardiovascular disease and pregnancy complications. The potential impact of this association is proven to be causative and could be significant from a public health standpoint, especially as periodontal disease

affects 10–20% of the population, and it is both preventable and treatable (2).

Periodontal diseases have long been recognized as a public health problem. In the past decade there has been a conceptual shift from periodontal diseases as an oral problem to periodontitis having an impact on systemic health. Recent evidence suggests a strong relationship between periodontal inflammatory disease and systemic diseases (9). The gingival wound surface area in severe periodontitis may be as much as 20 square centimetres. Periodontal pathogenic micro-organisms have the potential to translocate from this periodontal lesion into the systemic circulation to other organ systems. This rather big inflammatory process in the gingival connective tissue may lead to an excessive host response, manifesting as a local and general inflammatory response, which can result in chronic inflammation (10, 11). Chronic infection and inflammation are now being increasingly considered as new risk factors for development of systemic diseases, and periodontal disease may increase systemic levels of inflammatory mediators and thus potentially contribute to the inflammation-associated process (9, 12).

This presentation includes several longitudinal studies. Firstly, early carotid atherosclerosis in subjects with periodontal disease, the second is early carotid atherosclerosis in subjects with low and high levels of dental plaque and gingival inflammation. The third is periodontitis and premature deaths – results from a 16-year prospective study of a randomized control sample of young adults in Sweden. The fourth is myocardial infarctions in Sweden in subjects with periodontal diseases in a prospective study and influence of psychosocial stress on dental plaque, gingival inflammation and periodontal disease.

The material which was used in these studies was collected from the registry file in Sweden. In Sweden all people born on the 20th of any month are listed in registry files, and this file contains 105 998 subjects in the Stockholm area aged between 30 and 40 years. We randomized 3273 subjects from this big cohort, and then 1676 subjects were clinically examined, and they will constitute the subjects in the longitudinal studies I'm going to present.

The first study is on early carotid atherosclerosis in subjects with periodontal diseases. In this study the involvement of periodontal disease in the development of early carotid atherosclerosis vascular lesions has been evaluated. The aim was to assess the role of periodontitis in the development of atherosclerosis by evaluating in a prospective way the relationship between periodontal disease and subclinical signs of carotid atherosclerosis in patients with documented periodontitis of at least 16 years duration. Eighty-two patients with periodontal disease and 31 periodontally healthy individuals were chosen at random and subjected to a clinical oral examination in 1985. Atherosclerotic risk factors analysis and carotid ultrasonography was performed during re-examination 16 years later. Common carotid artery intima-media thickness and lumen diameter were measured and the intima-media area was calculated, both on the left and right side.

The results from this study showed that significantly more patients with periodontal disease had lower education, were smokers and had hereditary risk factors for atherosclerotic disease. These patients also had higher plasma cholesterol levels. The measured intima-media thickness and lumen diameter as well as calculated intima-media area were significantly greater in patients than in controls. In a multiple logistic regression analysis, periodontal disease appeared to be a principle independent predictor associated 4.6 times higher risk for increased carotid wall thickness; after adjustments for potential confounding factors. Periodontal disease also appeared to be a main independent predictor associated with increased intima-media area (relative risk 5.2) (13). The present results indicate that periodontal disease is associated with the development of early atherosclerotic carotid lesions in subjects without any symptoms of overt atherosclerotic disease. All these patients did not have any known cardiovascular diseases before we started the study.

As a dental hygienist I was interested in looking at dental plaque and gingival inflammation, so in another study early carotid atherosclerosis in subjects with low and high levels of dental plaque and gingival inflammation was investigated. One hundred and thirty-nine subjects – 67 females and 67 males – participated in this study. These subjects underwent a clinical oral examination including plaque index and gingival index, and the common carotid artery intima-media thickness was measured and the intima-media area was calculated. The results of this study were that a significant difference for intima-media thickness was found for the left carotid artery between subjects with high and low levels of plaque. The results for high and low levels of gingival inflammation also showed a significant difference for intima-media thickness on the left side. This was also true for the calculated intima-media area, where significant differences between high and low levels of gingival inflammation were found for the left side. So, the results indicated that dental plaque and gingival inflammation were in some way associated with the development of early atherosclerotic carotid lesions.

In a third study we investigated the relation between periodontitis and premature death in a 16 year prospective study of a randomized control sample of young adults in Sweden. Premature death was evaluated from a file with all causes of death and compared with an oral examination from 1985 for this group. The objective of the study was to evaluate the role of periodontitis in premature death. The mean age for premature death was 47.3 years. A significant difference was found for all clinical parameters between the deceased and survivors. The plaque index was measured in 1985 for deceased people and for people who were alive in 2001. For gingival inflammation and any missing molars there was also a significant difference. The result of the logistic regression analysis (death as dependent variable), showed that subjects with periodontitis and missing molars had 3.6 times the odds for being dead. We concluded that young individuals with high levels of dental plaque, gingival inflammation and periodontitis with missing molars, seemed to be at increased risk for premature death.

In a fourth study we investigated prospectively the incidence of myocardial infarctions in subjects with periodontal diseases. The aim of this study was to study myocardial infarctions in subjects with periodontal disease in a 16-year longitudinal study. From the initially screened subjects, 11 subjects were diagnosed as having had a myocardial infarction. The subjects with myocardial infarction were diagnosed at the hospital according to the international classification of diseases. The subjects with acute myocardial infarction had significantly higher GI scores and number of teeth with pockets 5 mm and deeper than subjects without infarction. The mean age for males was 45.6 years having had a myocardial infarction and for females 50.3 years. The multiple regression analysis with myocardial infarction as the dependent variable showed periodontal disease with missing molars as independent predictor associated with myocardial infarction after adjustment for several other independent variables. So subjects with periodontal disease seem to be at risk for myocardial infarctions.

From here I would like to turn over to another situation in our lives, and that is stress. In our study cohort we have also been looking at stress. Stress, a 21st century problem and we looked at the influence of psycho-social stress on dental plaque, gingival inflammation and periodontitis. In this study, the involvement of psychosocial stress in the development of gingival inflammation was evaluated. The psycho-social stress was measured as being a widow/widower; being divorced; and/or unemployed; anxiety; exhaustion and stress-related depression. Depressed patients exhibit alterations in both the cellular and humoral immune responses that may impair host defence mechanisms and promote accumulation maybe of periodontal pathogens and increase the risk for periodontal disease.

To answer these questions the subjects were asked to fill in a questionnaire concerning marital status and socioeconomic data, and we found that a widow/widower had a significantly higher plaque index compared with other groups, and this was also true for gingival index. The widow/widower group also showed significant differences in the number of teeth with pockets 5 mm and deeper compared with the other groups. The present results indicate that psychosocial stress could be involved in the development of periodontal disease.

I started my presentation with the question from Professor Harald Löe 'Half a century of plaque removal, what's next?' Basically, the answer is the same as it would have been half a century ago; mechanical removal of plaque through tooth brushing and other mechanical cleansing procedures continues to be the most reliable means of controlling plaque, gingivitis and periodontitis, provided the cleaning performed is sufficient and thoroughly and performed at appropriate intervals. But the impact of oral hygiene goes beyond gingivitis and periodontitis; it may be healthy for the body as a whole. Thus the studies I reviewed here, create a basis for prophylactic measurements for oral health, which in the view of prevalence of the studied diseases and the cost incurred by society as a result, appears to be efforts well spent.

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