The Interrelationship between Perio

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Cardiovascular diseases such as atherosclerosis and myocardial infarction occur as a result of a complex set of genetic and environmental factors. Analysis of all the common risk factors for stroke and heart attack, including age, smoking, high levels of serum lipids, diabetes and socioeconomic status can only account for one-half to two-thirds of the variation in the incidence of cardiovascular disease.

Numerous studies show that oral infection, especially periodontitis, may affect the course and pathogenesis of a number of systemic diseases, such as cardiovascular disease, bacterial pneumonia, diabetes mellitus and low birth weight. Evidence in support of an association between periodontal and cardiac conditions has been demonstrated and several theories have been proposed to explain the link.

In the early 20th century, Sir William Osler fully appreciated what was killing his patients with endocarditis. The theory of focal infection was firmly believed in and promulgated during that period. A focal infection is a localized infection that can disseminate microorganisms or toxic products to contiguous or distant tissues. The teeth and jaws, tonsils, sinuses, fingers and toes, bronchi and the gastrointestinal tract were the obvious sources blamed for such diseases as arthritis, nephritis and endocarditis. As a result of this theory, many physicians of the era often recommended preventive full mouth extractions whenever a 'focal infection' became suspected. These ideas were anecdotal and in time the theory fell on the wayside because these drastic treatments never quite reversed the course of the disease. Furthermore, many patients with the same diseases had no evident focus of infection and foci of infection are as common in apparently healthy persons as those with disease. With the recent progress in classification and identification of oral microorganisms and the realization that certain microorganisms are normally found only in the oral cavity this idea of bacterial dissemination via the blood stream through discontinuities of the oral tissues, has resurged.

The teeth are the only non-shedding surfaces in the body and bacterial levels can reach more than 10^{11} microorganisms per mg of dental plaque. Periodontal diseases are bacterial infections that destroy the attachment fibers and supporting bone (periodontium) that hold teeth. The consequence of such events can be measured as the 'gums' separate from the teeth, forming pockets. These are shallow at first but can eventually deepen, at a variable rate, depending on bacterial and host factors that set the stage. Increasing pocket depths are a sign of the disease progressing as more periodontal tissues

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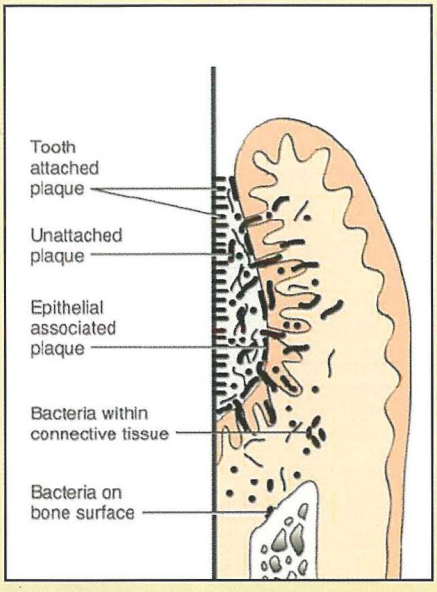

Figure 1: Plaque–bacteria associated with the crown / root surface and periodontal tissues

are destroyed and the teeth eventually become loose.

Human periodontal infections are associated with complex microfloras in which approximately 500 species have been encountered. These infections are predominantly anaerobic, with gram-negative rods being the most common isolates. Their endless presence burden and challenge the host continuously. In the depths of periodontal pockets, the anatomic closeness of these microfloras to the bloodstream facilitates bacteremia and systemic spread of bacterial products, components and immunocomplexes.

Microorganisms may gain entrance to the blood and circulate throughout the body but are usually eliminated by the reticuloendothelial system within minutes (transient bacteremia) and as a rule lead to no other clinical symptoms. However, if the disseminated microorganisms find favorable conditions, they may settle at a given site and, after a certain time lag, start to multiply (infective endocarditis, brain abscess, cavernous sinus thrombosis, sinusitis, lung abscess/infection, prosthetic joint infection).

Some gram-positive and gram-negative plaque bacteria have the ability to produce diffusible proteins, or exotoxins which are lethal poisons which cause local injury. Conversely, endotoxins are part of the outer bacterial membranes released after cell death. When introduced into the host, these lipopolysaccharides give rise to a large number of pathological manifestations not just locally but even at a distance from the site (cerebral infarction, acute myocardial infarction, abnormal pregnancy outcome, persistent pyrexia, idiopathic trigeminal neuralgia, toxic shock syndrome, systemic granulocytic cell defects, chronic meningitis).

Inflammation in tissues distant from the infection may also develop through the effects of soluble antigen entering the bloodstream and reacting with circulating specific antibody. These immunocomplexes may give rise to a variety of acute and chronic inflammatory reactions at the sites of deposition (Behçet's syndrome, chronic urticaria, uveitis, inflammatory bowel disease, Crohn's disease).

Most researchers believe that the increased risk to the progression of coronary heart disease is caused by an exaggerated inflammatory response to periodontal pathogens such as *Porphyromonas gingivalis* in a susceptible host.

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