FOCAL INFECTION THEORY: A FOCUS ON CURRENT ASPECTS

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ABSTRACT
Systemic health is often closely linked to the state of the oral health. Systemic diseases and conditions have oral manifestations and oral cavity reflects the signs of systemic diseases at its early stage. The mouth–body connection or focal infection is proved through numerous published researches. This article reviews pertinent literature detailing both the history of the focal infection theory and current focus on the possible association between oral and systemic diseases.

KEYWORDS: Focal infection, Systemic diseases, Periodontitis, Periodontal medicine.

INTRODUCTION
The relationship of our teeth and oral cavity to overall health is undisputable. The mouth body connection or focal sepsis theory is proved through numerous researches. A focal infection is a localized or generalized infection caused by the dissemination of microorganisms or toxic products from a focus of infection. Billings gave the first definition of focal infection: A focus of infection may be described as a circumscribed area of tissue infected with pathogenic microorganisms. [1]
HISTORY
The first report of focal infection was reported by Hippocrates who attributed the cure of a case of arthritis to a tooth extraction. In 1800s Benjamin Rush, an American physician also related arthritis cure to tooth extraction.[1]

In 1890, the dentist and physician WD Miller published his treatise, The Microorganisms of the Human Mouth. The local and general diseases which are caused by them. A year later in Dental Cosmos first used the term “Focal infection”. [4] Miller did not advise extraction of teeth as a focus of infection and also suggested ‘treating and filling root canals’. In 1900, the English physician, William Hunter, reported in the British Medical Journal on ‘Sepsis as a Cause of Disease’ listing poor oral health and the expanding use of ‘conservative dentistry’ as a cause of diseases attributed to focal infection. Miller promoted importance on sterilization of instruments would prevent spread of infection.[5]

The era of focal infection in medicine began in 1912 when the physician, Frank Billings,[2,6] introduced the concept of focal infection to American physicians through case reports that tonsillectomies and dental extractions claim to remove various foci of infections at distant organs.

In the 1920s, the theory of focal infection was the main cause taught as the cause of a wide range of illnesses with infected teeth[7] All pulpless teeth were focus of infection and the extraction of healthy teeth was justified to prevent focal infection.

In 1935, Cecil and Angevine[8] published an analysis of 200 cases of rheumatoid arthritis that documented no benefit from tonsillectomy or dental extractions, but rather occasional exacerbations of the arthritis and concluded that: ‘focal infection is a splendid example of a plausible medical theory which is in danger of being converted by its enthusiastic supporters into the status of an accepted fact,’ and that ‘the time has arrived for a complete reevaluation of the focal infection theory.’

In 1939, Vaizey and Clark Kennedy[9] demonstrated that patients who were edentulous developed subsequent arthritis and dyspepsia and that edentulism actually caused indigestion rather than cured it.
In 1940, Reimann and Havens published the most influential criticism of focal infection theory and observed that: the theory of focal infection had not been proved due to the following observations:

(1) Its infectious agents was not known
(2) Large groups of people whose tonsils are present are no worse than those whose tonsils have been removed,
(3) Patients who had tonsillectomy continue to suffer from the disease, for which they were removed,
(4) Any beneficial effects can seldom be ascribed to surgical procedures alone,
(5) Beneficial effects that occasionally occur after surgical measures are often lead by harmful effects or no effects.
(6) Foci of infection would be cured after recovery from systemic disease or when general health is improved with oral hygiene and nutritious diet. Only areas superficially accessible to surgery were listed as foci of infection. While deeper structures were conspicuously absent leaving one of its harshest critics to comment that a focus of infection was: ‘anything readily accessible to surgery.’\[10]\]

During 1950s came an end of focal theory of infection era when authorities who felt that focal infection was an important factor in systemic disease have become skeptical and recommend less radical procedure in treatment of diseases.\[4,11]\ There was a support for the theory till late 1950s\[12]\ and later focal infection vanished as the primary cause of chronic, systemic diseases. In 1990s' emergence of epidemiological associations between dental infections and systemic diseases, researches have been cautious, seeking association between dental infections like periodontitis and systemic diseases which marked the beginning of Periodontal medicine.\[13]\

**MECHANISM OF SPREAD FROM ORAL INFECTION**\[14]\n
Three mechanisms oral infections to secondary systemic effects have been proposed. These are: (a) metastatic spread of infection from the oral cavity due to transient bacteremia, (b) metastatic injury from the effects of circulating oral microbial toxins, and (c) metastatic inflammation caused by immunological injury by oral microflora.

**Metastatic infection**

Oral infections and dental procedures can cause transient bacteremia. The microorganisms that enter systemic circulation are eliminated by the reticuloendothelial system within
minutes and does not lead to clinical symptoms or it will lead to increase in body temperature. If the microorganisms have favorable conditions, they multiply.

**Metastatic injury**
Bacteria have the ability to produce diffusible proteins, or exotoxins, which include cytolytic enzymes and dimeric toxins. The exotoxins have pharmacological actions and are powerful and lethal poisons.

**Metastatic inflammation**
Antigens enter the systemic circulation and react with specific antibody to cause immunocomplexes that give rise to acute and chronic inflammatory lesions and conditions.

**Periodontitis and Systemic diseases**
To discuss all the systemic diseases associated with periodontitis is beyond the scope of this review. However well documented possible associations between periodontitis and systemic diseases are discussed below.

**Periodontitis and Pre term low birth infants**[^15]
The etiology of pre term low birth weight is multifactorial. There is ongoing debate regarding relationships between periodontitis during pregnancy and adverse pregnancy outcomes. Periodontal inflammation produce significant amounts of proinflammatory cytokines, like interleukin 1-beta (IL-1β), IL-6, prostaglandin E2, and tumor necrosis factor-alpha (TNF-α), which may have systemic effects. Low birth weight, defined as birth weight less than 2,500 g, which is a significant public health issue in both developed and developing countries. Research suggests that the bacteria bloodstream from the inflamed periodontal tissues and target the fetus, leading to premature labor and low-birth-weight (PLBW) babies. One mechanism begins with deleterious effects of endotoxins released from Gram-negative bacteria responsible for periodontal disease. periodontal disease appears to be an independent risk factor for PLBW and there is a need to expand preventive measures for pregnant women in harmonization with the gynecological and dental professions. Systemic review[^16] in 2013 suggested that, maternal periodontitis is modestly but significantly associated with LBW (low birth weight) and preterm birth. Data from prospective studies followed a similar pattern, but associations were generally weaker. It was found that Maternal periodontitis was significantly associated with pre-eclampsia. Maternal periodontitis is modestly but independently associated with adverse pregnancy outcomes.
**Periodontitis and Diabetes mellitus**

Epidemiological data confirm that diabetes is a major risk factor for periodontitis; there is a three fold increase in susceptibility to periodontitis in diabetics. There is a clear relationship between degree of hyperglycemia and severity of periodontitis. The mechanisms that underpin the links between these two conditions are not completely understood, but involve immune functioning, neutrophil activity, and cytokine biology. There is emerging evidence to support the existence of a two-way relationship between diabetes and periodontitis, with diabetes increasing the risk for periodontitis, and periodontal inflammation negatively affecting glycaemic control.\[^{17}\] Several meta-analyses have confirmed that effective periodontal therapy can result in reduced Glycated haemoglobin (Hb A1c)\[^{18,19}\]

**Periodontitis and cardiovascular diseases**

Evidence continues to support the association between periodontal infections, atherosclerosis and vascular disease. Recommending periodontal treatment for the prevention of atherosclerotic cardio vascular disease (CVD) is not warranted based on scientific evidence. Periodontal treatment must be recommended on the basis of the value of its benefits for the oral health of patients, recognizing that patients are not healthy without good oral hygiene. However, the emergence of periodontal infections as a potential risk factor for CVD is leading to a convergence in oral and medical care that can only benefit the patients and public health.\[^{20}\] A systematic review concluded in 2008 Periodontal disease is a risk factor or marker for CHD (Coronary heart diseases) that is independent of traditional CHD risk factors, including socioeconomic status. Further research in this important area of public health is warranted.\[^{21}\]

**CONCLUSION**

In this era of Periodontal medicine, researches have been cautiously exploring to establish definitive conclusions on the nature of associations between periodontitis and systemic diseases. Oral health has a direct and or indirect impact on the overall general health. In susceptible individuals, however periodontal infection may act as risk factor or may be involved in pathogenic mechanisms. Dentists must improve their knowledge and clinical exposure of relevant systemic conditions in order to interact and relate meaningfully with their medical colleagues. Regular dental checkup is strongly advocated in the light of current knowledge.
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