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Periodontal Medicine: Poor periodontal health as a risk for systemic health

Dental Science

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ABSTRACT

The molecular aspect of pathogenesis of periodontal diseases has already proved its immuno inflammatory nature and various systemic and environmental factors also influence the disease process. Recent evidences indicate that we need to change how we think about the pathogenesis of periodontal disease as it is not limited to the periodontal tissues. Chronic nature of periodontal infection and inflammatory markers associated with the disease process may also affect health of other organ systems. This review highlights about proposed mechanisms of periodontal infection as a risk factor for systemic health or as a factor involved in the deterioration of existing systemic condition.

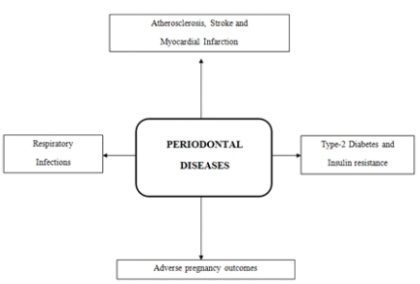
Introduction:

Periodontal medicine is a rapidly emerging branch of periodontology which deal with two way relationship between periodontal disease & systemic diseases. Periodontal medicine offers new insight into the concept of oral cavity as one system interconnected with the whole body.[1] The field of periodontal medicine addresses two important questions: 1. Can the inflammatory response to bacterial infection of the periodontium have an effect remote from the oral cavity? 2. Is periodontal infection a risk factor for systemic diseases or conditions that affect human health?[1] This scientific paper discusses briefly about the role of periodontal infection as a risk factor for systemic health and conditions.

Focal infection theory revisited.

W.D Miller in 1891 proposed a theory[2] that local infection affecting a small part of the body can lead to subsequent infection or symptoms in other part of the body due to spread of infecting agent itself or toxins produced by it. William hunter[3] identified carious tooth, gingivitis and periodontitis as foci of infection and considered oral cavity as a potential focus of infection. He advocated extraction of teeth to prevent the source of sepsis. However, in the era of 1940 & 1950, widespread extraction of the entire dentition failed to reduce the systemic condition of the patient. Hunter's concept of "oral cavity as a focus of infection" was discarded due to inadequate scientific support, but it encouraged extensive research in the immunology and microbiology of periodontitis and its impact on the systemic health. Systemic impact of the periodontitis depends on the severity of the inflammation/infection and the individual's degree of resistance to the infection.

Fig: 1- Organ systems possibly influenced by Periodontitis



1. Effect of Periodontitis on Cardiovascular diseases (CVD)

Various authors have reported a hypothetical link between periodontitis and increased risk of CVD. 1989, Matilla et at.[4] in a case control study found association between poor oral health and acute myocardial infarction. Patients with periodontal disease share common risk factor as patients with CVD such as age, gender(male), lower socio-economic status, stress and smoking. Systematic review by Scanapieco and colleagues (2003)[5] periodontal disease may be modestly associated with atherosclerosis, myocardial information and other cardiovascular events. Janket et al. (2003)[6] – meta analysis - 19 % increased risk of cardiovascular diseases in periodontitis patients and various clinical studies suggesting the role of biomarkers and mediators of inflammation in periodontitis as a link to inflammation in cardiovascular diseases. Myocardial infarction is the most common CVD that results due to either atherosclerosis or thromboembolism. Indirect role of periodontitis and its disease markers in the pathogenesis of atherosclerosis and thrombosis have been investigated and several models linking both the disease processes have also been suggested.

Figure 2: Proposed model and mechanism in role of periodontal infection in thrombogenesis

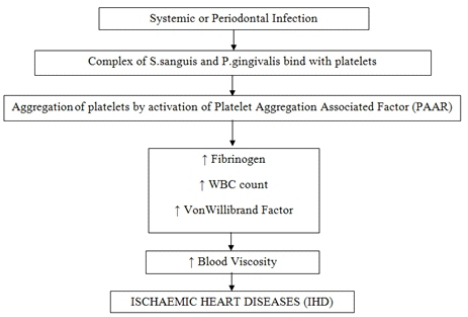
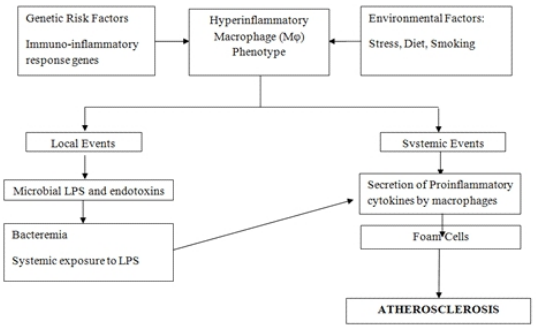


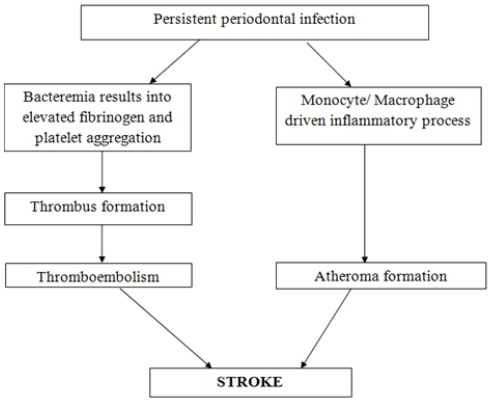
Figure 3: Proposed link between role of periodontal infection in atherosclerosis



2. Periodontal disease and Stroke

In a case control study by Syrjenan et al.[7] found that poor dental health is a significant risk factor for cerebral ischemia or stroke. 25% of all strokes patients had severe periodontitis compared to controls. There are two types of stroke: 1. Hemorrhagic that results due to aneurysm and 2. Non-hemorrhagic that results due to cerebral atherosclerosis and thromboembolism. Periodontal infections can play role into the pathogenesis of non-hemorrhagic stroke by providing a persistent bacterial challenge to arterial endothelium.

Figure 4: Proposed model for role of periodontal infection in Stroke



3. Periodontal disease and Diabetes mellitus (DM)

DM is a multi-factorial, heterogeneous group of metabolic disorder characterized by chronic hyperglycemia, absolute or relative deficiency of insulin or resistance to the action of insulin. Principal etiological factors for diabetes are environmental exposure (sedentary lifestyle), genetic predisposition, autoimmunity, viral infection and insulin resistance. Chronic hyperglycemia results into enzymatic irreversible glycation of body proteins and formation of advanced glycated end products (AGEs). excessive AGEs bind with macrophage and monocyte receptors and stimulated macrophages proliferate, upregulate proinflammatory cytokines and production of oxygen free radicals. Release of proinflammatory cytokines lead to recruitment of other immune cells (T & B cells), vascular injury, altered wound healing and altered tissue turnover rates. All these factors collectively result into impairment in function of end organ and subsequent damages. Severe complications of DM that are also considered as end organ damages are: 1.Neuropathy, 2.Nephropathy, 3.Retinopathy, 4. Altered wound healing and 5. Macrovascular disease. Periodontitis is very common and severity increases clinically with patients of "poor metabolic control" in diabetes, therefore periodontitis was added as a sixth complication of DM by Loe et al. in 1993. Numerous studies have identified prevalence of periodontitis in poorly controlled diabetes. Grossi et al.[8] reported diabetics are twice as likely to exhibit attachment loss and mobility as non-diabetics. Emrich et al.[9] found out the risk of developing destructive periodontitis increases three fold in diabetic patients. Nelson et al. found that diabetics were 2.6 times more likely to presents with alveolar bone loss compared to non-diabetics. Very few studies but they have examined the impact of periodontitis and its treatment on the glycemic control at the diabetes patients. Tylor et al.[10] reported periodontitis is a risk factor for worsening of glycemic control six times over a 2 year period in diabetics. Collin et al. reported patients with severe periodontitis have higher HbA1c then the diabetics with mild periodontitis. Saremi et al. reported death rate from ischemic heart disease was 2.3 time high and death rate from diabetes nephropathy was 8.5 times high in severe periodontitis patients with diabetes. Milles et al. showed that treatment of periodontitis with scaling rootplaning and systemic doxycycline therapy for 2 weeks improved glycemic control in type-2 diabetic patients. Teeuw WJ et al. periodontal treatment leads to improvement in glycemic control in type -2 diabetic patients for at least 3 month.

Figure 5: Effect of infection of glycemic control (FFA- free fatty acid, TRG- triglycerides)

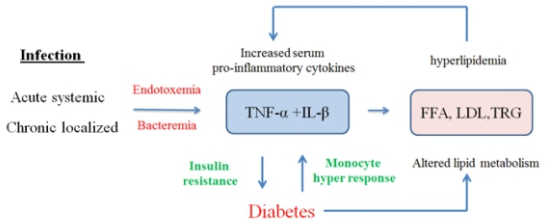
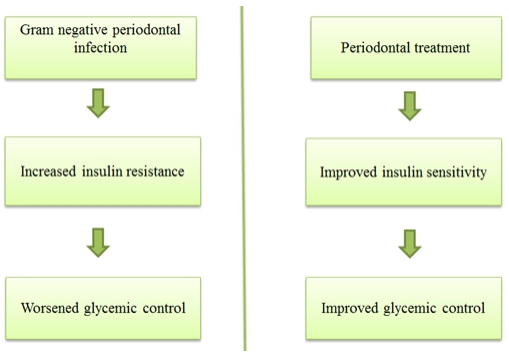


Figure 6: Potential effect of periodontal infection and periodontal therapy on glycemic control of diabetics



4. Periodontal disease and adverse pregnancy outcome (APO)

Types of adverse pregnancy outcome (APO) are premature rupture of membranes (PROM), preterm Birth (PT), low Birth Weight (LBW), pre-eclampsia, miscarriage, perinatal morbidity and mortality. 1996, a landmark report by Offenbacher et al.[11] created a much interest and research into whether periodontitis may be a possible risk factor for adverse pregnancy outcomes. Offenbacher conducted a case control study on 124 pregnant or postpartum women, Preterm birth was defined as gestation period < 37 weeks and low birth weight - < 2500 grams. He found that women having PTLBW has significantly greater attachment loss then women having normal birth weight infants. Factors responsible for preterm & low birth weight are smoking, alcohol, drug use during pregnancy, low material age, genitourinary tract infection, low socio economic status but these risk factors are not present in about one-fourth of preterm & low birth weight events leading to a search for another causes.

Bacterial vaginosis and PTLBW

Bacterial vaginosis is caused by changes in the vaginal microflora in which normal microflora facultative lactobacilli are replaced by gardenerella vaginalis, peptostreptococcus, prevotella bivia, porphyromonas , bacterioids urolyticus etc. The incidence of pre-term increases three times in women with bacterial vaginosis. Mechanism of pre-term delivery by bacterial vaginosis may be a direct infection of the placenta and amnion-chorine membrane or an

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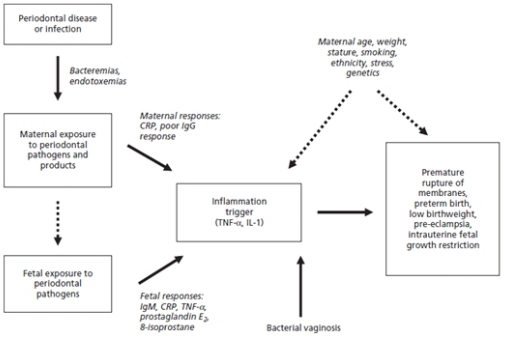
indirect mechanism (possible with chronic infections like periodontitis) by which increased prostaglandins and other pro-inflammatory cytokines may lead to premature rupture of membranes. Amniotic fluid of preterm and low birth weight infant women demonstrate culture positive for F. nucleatum & Capnocytophaga species. Although frequency of detection of oral species such as F. nucleatum and Capnocytophaga is occasional in bacterial vaginosis but authors like Hill et al. suggested a hematogenous spread of infection from the oral cavity. Bacterial invasion of membranes and placenta stimulates production of host derived cytokines including IL-1β, IL-6, TNF-α and increased labor including levels of prostaglandins like PGE2 and PGF2result in membrane rupture or uterus contraction leading to "pre-term birth". Collin's landmark studies on pregnant hamster demonstrated that chronic exposure to periodontal pathogens like P.gingivalis in a chamber model enhances the fetal placental toxicity during pregnancy and it significantly increases production of TNF-α and PGE2 from the placenta. Human studies such as 1996, a case control study on 124 pregnant woman by Offenbacher et al. suggested that periodontal attachment loss more than 3mm in at least 60% of sites had 7.5 times increased risk for LBW. Medianes et al.[12] found high levels of fetal IgM level seropositive to oral Bactria. Jeffcoat et al[13]. in a prospective study found that risk of PTLBW increased 5 times more in periodontitis case. A systematic review by Xiong X et al on 25 studies reported that 18 studies showed significant association between periodontitis and increased risk of adverse pregnancy outcome.

Figure 7: Possible mechanism of periodontitis affecting adverse pregnancy outcomes



Periodontitis and Preeclampsia

Preeclampsia clinically manifest in the second half of the pregnancy by the appearance of hypertension and proteinuria without previous history of hypertension. It affects 5-10% of pregnancies and major cause of perinatal maternal morbidity and mortality. Atherosclerotic like changes in placental tissues produced by inflammatory and oxidative events are responsible for development of preeclampsia. Boggess and co-workers[14] hypothesized that maternal exposure to periodontal pathogens lead to atherosclerotic changes of placental tissues and it increases 2 to 2.5 fold increase in the events of preeclampsia. Contreas et al. reported that 64% of pre-eclamptic women had chronic periodontitis with attachment loss ≥4mm.

Effect of periodontal treatment and adverse pregnancy outcomes

Jeffcoat et al. found reduce rate of LBW in women who received mechanical periodontal therapy during gestation. Conversely, Mickalowicz et al. demonstrated that periodontal treatment improved the periodontal health of the patient and had minimal effect on pregnancy outcome. Chambrone et al. in his systematic review failed to show that the material periodontal treatment can decrease the risk of adverse pregnancy outcomes. Despite of any supporting evidence, pregnant women with periodontal disease should be instructed about importance of periodontal health and should undergo proper treatment.

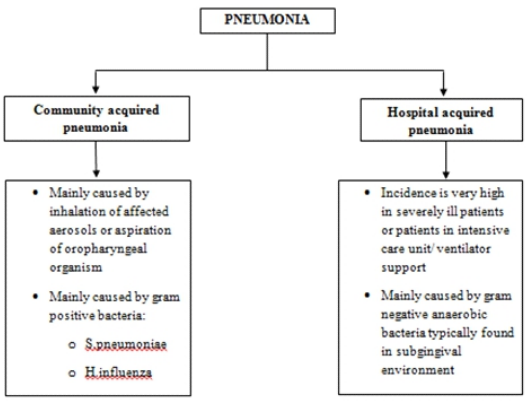
5. Periodontitis and chronic obstructive pulmonary diseases [COPD]

Chronic obstructive pulmonary disease is characterized by airflow obstruction resulting from chronic bronchitis or emphysema. COPD has similar pathologic mechanism with periodontal disease. In both the diseases host inflammatory response is mounted in response to chronic bacterial challenge in periodontitis and factor such as cigarette smoking in COPD. Various Evidence support the role of periodontitis in COPD such as Hayes et al. in a longitudinal study on 1100 men, alveolar bone loss was associated with the risk of COPD. Scanapieco et al.[15] found individuals with poor oral hygiene found to be associated with increased risk of chronic bronchitis and emphysema. Conversely, Hyman et al. conclude no significant association between periodontitis and COPD. Nonsmokers and former smokers with severe periodontitis did not increase the risk of COPD, only current smokers with severe periodontitis showed increased risk of COPD. So, smoking may act as a major "effect modifier" in relation between periodontitis and COPD and that modifier's presence is required to generate the effect. A recent systematic review by Williams et al. stated that insufficient evidence exists on potential association between periodontitis and COPD.

6. Periodontitis and Acute respiratory infection

Pneumonia is an infection of the lungs caused by bacteria, viruses, fungi or mycoplasmas.

Figure 8: Types of pneumonia and hospital acquired or nosocomial pneumonia is frequently caused by periodontopathic bacteria



Oropharyngeal colonization with potential respiratory pathogens (PRP) generally increases during hospitalization subsequent aspiration of PRP may result into hospital acquired pneumonia. El Solah et al. reported that dental plaque has been shown to serve as a reservoir of PRP and it result in persistent source of potential aspiration. Scanapieco et al. reported that poor oral hygiene is very common in severely ill hospitalized patients and PRPs are often isolated from supragingival plaque and buccal mucosa of the intensive care unit patients. In a systematic review by Scanapieco et[15] al. exhibited improved oral hygiene using mechanical tooth brushing and chemical plaque control has potential to decrease the risk of hospital acquired pneumonia.

Periodontal medicine in clinical practice

Following are some important points[1] which need to be encouraged in routine clinical practice to create awareness about importance of oral health among the general population

1. Dental professional should recognize the oral cavity as one of many interrelated organ systems and infection of the periodontium should be given as similar important as infection of other body part.

2. Patient education about biologic plausible role of periodontal infection in systemic disease and emphasis should be given on periodic check up of patents with ischemic heart disease, diabetes and pregnant women.

3. Enhanced community awareness may be derived from newspaper, magazine and audio visual media. However the most reliable origin of information should be the dental and medical profession through daily contact with patient.

4. Dentists need to communicate with physicians so that susceptible patients are evaluated for periodontal disease and receive appropriate treatment.

5. After successful complication of periodontal therapy. Long term periodontal maintenance is essential to reduce the risk of systemic disease.

Summary and conclusion:

Periodontal disease may increase the risk for many systemic diseases such as coronary artery heart diseases, diabetes, pregnancy outcomes stroke and COPD. Biologic plausible mechanisms support the role of periodontal infection in above mentioned conditions but it should not be presented as a causative factor, it should be presented as a risk factor for initiation and aggravation of systemic conditions. It is a modifiable risk factor, unlike the age, gender and genetic influence. So, treatment of the periodontal disease may improve the underlying systemic health and reduces the risk of complications. Longitudinal studies and controlled trials are necessary to establish relationship between periodontal disease and systemic conditions and treatment strategies need to be defined to control the periodontal as well as systemic diseases.

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