

A Methodical Approach On Periodontal Health And Preterm Low Birth Weight

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ABSTRACT

Periodontal disease is a complex disease that is responsible for a chronic inflammatory challenge in various parts of the body. Periodontal infections serve as a reservoir of inflammatory mediators, that act as a threat to the fetal-placental unit and lead to adverse pregnancy outcomes (APO). The microbes in the dental biofilm releases substances that can activate the host immunoinflammatory responses. This challenge could trigger inflammatory mechanisms associated with preterm birth outcomes.

Keywords:*Periodontitis, Preterm low birth weight, Periodontal medicine*

I. Introduction

Periodontal diseases are multifactorial disease which affects the tooth supporting structures, resulting in inflammation and destruction of the periodontium. The progression of bacterial load and their destructive process leads to chronic and systemic challenge by the action of host-derived inflammatory mediators that are capable of initiating and promoting systemic diseases. Periodontal Medicine deals with a two-way relationship in which periodontal disease in an individual may be a powerful influence on an individual's systemic health/disease or the role of systemic disease on influencing an individual's periodontal health or disease.¹ The prevalence of periodontitis is high in pregnant mothers (40%), and they are seven times at higher risk of having preterm or low birth weight baby. The hormonal changes during pregnancy, promotes inflammatory response that facilitates the occurrence of periodontal disease. Periodontal disease may be one of the possible risk factors for preterm low birth weight (PTLBW) infant.²

The concept of oral sepsis was first put forward by William Hunter in 1900. Later an era of focal infection (1915-1950) evolved where in all teeth that were endodontically or periodontally involved were extracted to avoid a possible focus of infection. In 1952 an editorial in the Journal of the American Medical Association stated that focal infection theory has fallen out of favour because many patients with diseases presumably caused by foci of infection have not been relieved of their symptoms by removal of the foci.³

II. Periodontitis and adverse pregnancy outcomes

Primary outcomes

Maternal mortality, Preterm delivery and Perinatal mortality are primary outcomes.

Secondary outcomes

Miscarriage and fetal death before 20 weeks gestational age, Preterm labour rupture of membranes (PPROM), leakage of amniotic fluid in the absence of uterine activity before 37 weeks gestation, Pregnancy-induced hypertension, onset of hypertension (blood pressure $\geq 140/90$ mmHg) in the second half of pregnancy in the absence of proteinuria or other markers of preeclampsia like hypertension and proteinuria, Clinical chorioamnionitis, clinical evidence of intra-amniotic infection with or without laboratory signs of infection, Histological chorioamnionitis, Stillbirth, Very preterm delivery Low birth weight, birth weight of <2500 grams, Birth weight <10th centile for gestational age, Early onset neonatal sepsis, clinical evidence of sepsis with laboratory signs of infection within 72 h after delivery, Neonatal death which includes death of a baby occurring within the first 28 days of life.⁴

III. Periodontitis and Preterm low birth weight

Over the past 25 years, there have been significant advances in perinatal medicine and in understanding of reproductive physiology. However, despite these advances, the prevalence of preterm low-birth weight infants has not changed.

IV. World Health Organization

Delivery at less than 37 weeks of gestation is termed as preterm birth and Delivery at less than 32 weeks is termed very preterm. Delivery at less than 28 weeks, as extremely preterm. Most preterm births are also associated with low birth weight. The international definition of low birth weight adopted by the Twenty-ninth World Health Assembly in 1976 is a birth weight of “less than 2500 gm”.⁵

V. Periodontitis: A significant potential risk factor for preterm birth

Offenbacher et al (1996) provided the evidence regarding the interaction of periodontal pathogens in preterm birth. A case control study of 124 pregnant or postpartum mothers were conducted in which mothers with preterm or low birth-weight babies had significantly worse periodontal disease than those giving birth to normal weight babies. This study has suggested a significant role of cytokines in the mechanism for preterm low birth weight babies and reported that periodontal disease is a significant risk factor with an odds ratio of 7.9 for all preterm low birth weight babies.⁶ Later, Offenbacher et al (2001) hypothesized that common pathways may lead to preterm birth independent of the particular risk factors. Periodontopathic bacteria, mainly gram-negative anaerobes, serve as a source for endotoxin and lipopolysaccharides; inflammatory

mediators including PGE₂, and cytokines are locally increased. It has been reported that systemic increases of inflammatory mediators may lead to preterm birth.⁷

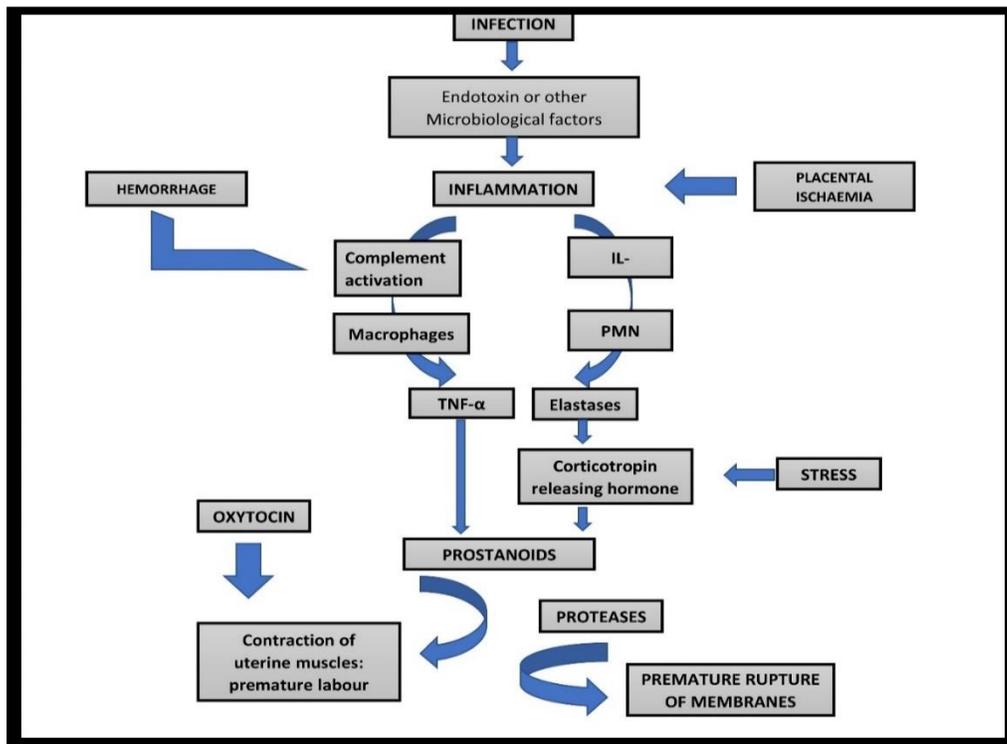


Figure 1: Putative mechanisms involved in preterm labors.[Courtesy: Bey A et al 2011]⁵

VI.Risk indicators for preterm low birth weight babies⁴

Primary indicators

Black race, young mother, domestic violence, low socio-economic status, stress or depression, cigarette smoking, cocaine or heroin use, previous preterm birth or second trimester pregnancy loss/ abortion, family history/inflammatory gene polymorphisms, chronic lung disease, chronic hypertension, diabetes, renal disease.

Secondary indicators

No or inadequate prenatal care, in vitro fertilization, low maternal weight gain late in pregnancy, iron-deficiency anemia, pre-eclampsia, elevated fetal fibronectin, alpha-fetoprotein, alkaline phosphatase, or granulocyte colony-stimulating factor, early contractions, bacterial vaginosis, especially early in pregnancy, chorioamnionitis, placental abruption, placenta previa, hydramnios, pre-eclampsia, multiple fetuses.

VII. Most likely mechanisms that may mediate an association between periodontal infections and adverse pregnancy outcomes⁵

Direct pathway

Micro-organisms can gain access to the amniotic cavity by ascending from the vagina and the cervix or by hematogenous dissemination through the placenta or by accidental introduction at the time of invasive procedures (amniocentesis) or by retrograde spread through the fallopian tubes.

Indirect pathways

Inflammatory mediators locally produced in periodontal tissues, for example, PGE₂, TNF α , circulate and impact the fetal-placental unit and Inflammatory mediators and/or microbial components circulate to the liver, enhancing cytokine production (e.g., IL-6) and acute phase protein responses (e.g., CRP), which then impact the fetal-placental unit.

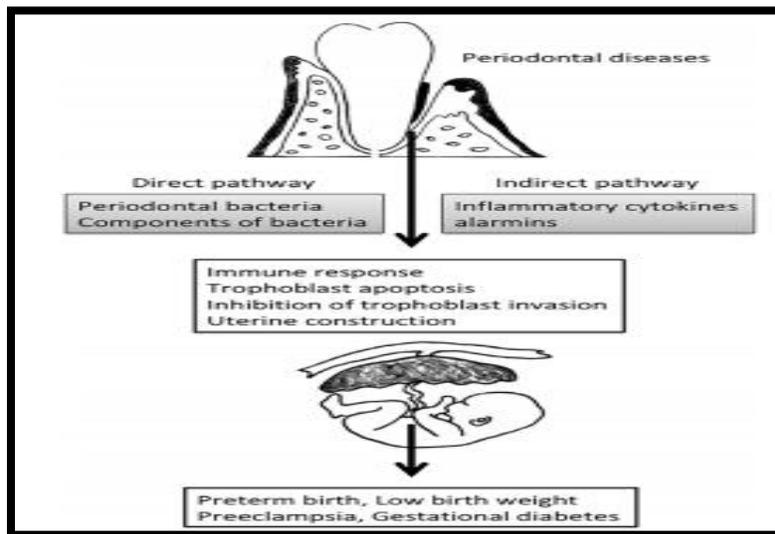


Figure 2: Potential biological mechanisms of periodontal disease inducing adverse pregnancy outcome.

[Courtesy: S K Aizawa et al 2016]⁸

VIII. Role of Periodontitis in preterm LBW

Hill in 1993 reported that bacterial vaginosis is caused by changes in the vaginal microflora in which predominant facultative lactobacilli are replaced by *Gardnerella vaginalis*, including

Prevotella, Bacteroides, Peptostreptococcus, Porphyromonas, and Mobiluncus.⁹ Species most often isolated is *F.nucleatum* which is a common oral species highly prevalent in periodontitis patients and could reach amniotic fluid by hematogenous spread from oral cavity. Collins JG in 1994 demonstrated that *P.gingivalis* implanted in subcutaneous chambers during gestation caused significant increase in PGE2 and TNF alpha levels. This localised subcutaneous infection resulted in a significant increase in foetal death and a decrease in foetal birth weight for those that remained viable compared with control animals that were not inoculated. This suggested that a remote non-disseminated infection with *P.gingivalis* may result in abnormal pregnancy outcomes.¹⁰ In a prospective study by Jeffcoat et al in 2001 were more than 1300 pregnant women subjects with generalized periodontitis subjects examined and concluded that there was five-fold increased risk of preterm birth before 35 weeks of gestation and seven-fold increased risk before 32 weeks compared with women without periodontitis.¹¹ Inadequate pre-natal care is also often cited as a risk factor for poor pregnancy outcomes in low socio-economic status and poorly educated women as reported by Sokol et al in 1980.¹² To the contrary, Quick et al in 1981 stated that adequate utilization of pre-natal care can result in improvement in birth weights as well as lower risk of PTB.¹³ Tellapragada C et al in 2016 evaluated the prevalence of clinical periodontitis among South Indian pregnant women and reported that the prevalence was found to be 10%.¹⁴

IX. Significance of periodontal treatment in reduction of adverse pregnancy outcomes

In a study by Lopez et al he found women who received scaling and root planing before 28 weeks gestation followed by prophylaxis every 2 weeks until parturition, had a low LBW rate of 1.28%.¹⁵ Conversely, Jeffcoat et al 2003 found reduced preterm birth rate in women who received mechanical periodontal therapy during gestation.¹⁶ Michalowicz et al in his study stated that providing periodontal treatment during pregnancy improved periodontal health but has minimal impact on pregnancy outcome.¹⁷ Transient bacteremia as well as hematogenous transmission may occur as a result of periodontal treatment or when there is regression in periodontal conditions after the treatment stops. Hence, it may be necessary to perform multiple treatments to keep the periodontal inflammation under control.¹⁸

X. Conclusion

Maternal periodontitis is modestly, independently, and significantly associated with LBW and preterm birth, but the use of a categorical or a continuous exposure definition of periodontitis appears to impact the findings. The evidence strongly suggests that periodontal infection may have significant negative impact on pregnancy outcome in some women. However regular dental care and periodontal health maintenance can reduce adverse effects in pregnant women.

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