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Adverse Pregnancy Outcomes Associated with Periodontal Disease: A Review and Update

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ABSTRACT

Background: Periodontal diseases are considered a risk factor for adverse pregnancy outcomes, including preterm birth, fetal growth restriction, low birth weight, pre-eclampsia and gestational diabetes. Periodontal bacteria originating in the gingival biofilm directly affect the feto-placental unit subsequent to bacteremia and inflammatory mediators secreted by the subgingival inflammatory site are carried to the feto-placental unit, where they then cause an inflammatory response are two possible mechanisms involve. The role of dentists is important to promote good oral hygiene in expecting mothers during routine prenatal visits to reduce chances of adverse pregnancy outcomes.

Keywords: low birth weight, periodontitis, preterm birth, pregnancy.

INTRODUCTION

Adverse pregnancy outcomes (APOs) are serious events that every year cause the death or disability of many newly born infants worldwide. [1,2] The most common adverse pregnancy outcomes are represented by low birth weight (LBW), preterm birth (PTB), and pre-eclampsia (PE). Adverse pregnancy outcomes (APOs) have been defined as (a) pre-term birth, when there is a delivery before 37 completed weeks (<259 days); (b) pre-eclampsia, which is a multisystem disorder of pregnancy characterized by maternal hypertension and proteinuria after the 20th gestational week; (c) low and very low birth weight, depending on whether the weight of the baby is less of 2500 g or <1500 g and (d) the spontaneous death of the fetus with <20 weeks (miscarriage) or between 20 and 36 weeks (stillbirth). PD during pregnancy starts by dental plaque and is increased by the action of pregnancy hormones. Periodontal diseases are considered a risk factor for APO, including preterm birth, fetal growth restriction, low birthweight, pre-eclampsia and gestational diabetes. However, the efficacy of periodontal treatment during pregnancy is controversial. [3]

Mechanism of spread: Two pathogenic mechanisms might explain the potential effect of periodontal diseases on pregnancy outcomes. First, periodontal bacteria originating in the gingival biofilm directly affect the feto-placental unit subsequent to bacteremia. Second, inflammatory mediators secreted by the subgingival inflammatory site are carried to the feto-placental unit, where they then cause an inflammatory response. [4]

Mechanism 1: Periodontal bacteria translocation into the maternal fetal unit as a direct mechanism of association oral microbiota

A direct relationship between worsening of PD and pregnancy has been demonstrated in many studies. [5] During pregnancy, the classical manifestations of PD (bleeding on probing, increase of pockets depth) are exacerbated. These clinical signs are reduced after childbirth. [6] PD is an inflammatory response of the host to the presence of dental plaque, leading to the loss of teeth, if untreated.[7] Pregnancy-associated PD is similar, but estrogen and progesterone can exacerbate gingival edema and vasculature. Recent studies established changes in putative pathogens of PD during pregnancy.[8] Periodontal pockets are a reservoir of oral microbiota. Modifications in oral microbiota may be considered as a potential mechanism for developing PD during pregnancy. A recent study reported that the worsening in PD was associated with the increase of "red complex" bacteria like Porphyromonas gingivalis and Prevotella.[9] However, the proportions of the "red complex" bacteria did not differ during pregnancy, although significant differences were found for all the pathogens after childbirth. A recent study reported that bacteria loading of Porphyromonas gingivalis and Tannerella forsythia at the 3rd month of pregnancy was associated with worsening in PD measured by bleeding on probing. [10]

Host response: PD is surely caused by bacteria, but the progression and worsening are due to a host immune response. The inflammation caused by PD is not limited to the oral cavity. It is hypothesized that episodes of bacteraemia and dissemination of endotoxins from periodontal pockets can induce the activation of the systemic immune response. Bacteria or bacterial endotoxins in the systemic circulation may induce pro-inflammatory cytokine production. These cytokines, then further activate the inflammatory response, which results in a chronic low-grade systemic up-regulation of the inflammatory molecules involving IL-6 and C-reactive protein .[11] The inflammatory response also activates inflammatory and endothelial cells and may result in endothelial dysfunction. In pregnancy, the immune response plays a pivotal role in maintaining a healthy equilibrium between the mother and fetus. During a normal pregnancy, the specific immune response is shifted towards a Th2-type immune response, and the inflammatory response is also activated. [12] The increased expression of activation markers on monocytes and granulocytes, differences in monocyte cytokine production, and increased circulating levels of pro-inflammatory cytokines and inflammatory markers, such as C-reactive protein, characterizes this activation of the inflammatory response during pregnancy.[13]

Mechanism 2: Release of Pro-Inflammatory Molecules from Periodontal Inflamed Tissues and Systemic Inflammation as an Indirect Mechanism of Association

Periodontitis represents a constant systemic challenge for immuno competent cells, with the activation of pro-inflammatory cascades that contribute to the systemic inflammatory status of the subject.[14] This is represented by an increase in acute phase response molecules, such as C-reactive protein (CRP) and pro-inflammatory cytokines (TNF-α and IL-6). However, the biological mechanisms and the molecules involved in the relationship between periodontitis and GDM are mostly unknown. [15]

CONCLUSION

Periodontal diseases in the pregnant mother significantly increase the risk of subsequent preterm birth or low birth weight. While it remains important to promote good oral hygiene during routine prenatal visits, there is no convincing evidence, on the basis of existing case control and prospective studies, that treatment of periodontal disease will reduce the risk of preterm birth.

Consequently, large randomized, placebo-controlled, masked clinical trials are required.

Conflict of Interest: None

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