

Oral Infection as a Risk Factor for Preeclampsia

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Editorial

Preeclampsia is a common obstetric syndrome affecting approximately 5-10% of pregnant women leading to significant maternal mortality and morbidity. Preeclampsia accounts for 25% of preterm deliveries resulting in adverse neonatal outcome. It is also the third most common direct cause of maternal death in the United Kingdom [1,2]. Preeclampsia is characterized by new onset hypertension exceeding 140/90 mmHg and proteinuria exceeding 300 mg in a 24 hour urine sample, after 20 weeks of gestation in a previously normotensive woman. It is characterized by abnormal vascular response to placentation, reduced organ perfusion, vasospasm, activation of the coagulation system, inflammatory like response, oxidative stress and some perturbation in volume and blood pressure control, affecting the placenta, kidney liver and brain [3-5].

Periodontitis is regarded as a chronic inflammatory oral infection that affects the tooth supporting structures and bone, in which bacteria of dental plaque and calculus and their byproducts are the principal etiologic agents. Teeth, gingival margins and periodontal pockets are places that could harbor bacterial colonization, and that one cubic millimeter of dental plaque contains about (100 million) bacteria [6-8]. Unhygienic oral conditions that results in inflammatory effects as in periodontitis could negatively affect the general health of individuals. A cause and effect relationship between the health condition of the oral cavity and some systemic diseases is attributed to the presence of dental plaque, periodontal and pericoronal infections [9].

There is a positive clinical association between chronic oral infection and various systemic diseases, rheumatoid arthritis, cardiovascular-disease, diabetes mellitus, chronic respiratory diseases and adverse pregnancy outcomes including pre-term low-birth weight and pre-eclampsia [10-13]. Since periodontal pathogens play a role in systemic diseases either through a direct pro inflammatory effect or through indirect host mediated effects triggered by oral infection [14-16]. Both periodontal and pericoronal infections are chronic Gram-negative infections proposed to feature a chronic endotoxins burden that may result in transient translocation of the organisms to the placenta, thereby triggering placental inflammation [11-13]. Host tissues in periodontal infections could mount an immune-inflammatory response to bacteria and their byproducts by activating host-derived cytokines such as interleukins 1 and 6 as well as tumor necrosis factor- alpha (TNF- α) and prostaglandin E2 (PGE2) resulting in connective tissue destruction and bone loss [7,11]. Herrera et al. [17] mentioned that the early identification of risk factors and the treatment of a symptomatic chronic infections lowered the preceding incidences of preeclampsia, they hypothesized that chronic infections may cause increased maternal cytokine levels sufficient to affect vascular endothelial function, thereby making pregnant women prime individuals for the subsequent development of preeclampsia.

Previous studies in human showed that oral microorganism, including *F.nucleatum* and *Capnocytophaga sputigena* were detected in the amniotic fluid of women with intact membranes and in those with preterm labor [18-20]. Moreover, Madianos et al. [21] have assessed the umbilical cord serum for the presence of fetal immunoglobulin M (IgM) to oral pathogen *Porphyromonas gingivalis*, documenting a fetal humoral response to organisms distant from the intrauterine environment and suggesting that translocation of oral pathogens to the uteroplacental unit may occur. These studies are supporting the possibility that oral bacteria or bacterial products can spread through the blood stream to the placenta.

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