Oral Infection as a Risk Factor for Preeclampsia

Mona Z Zaghloul

Microbiology Unit, Department of Clinical Pathology, Ain Shams University Hospitals, Cairo, Egypt

*Corresponding author: Zaghloul MZ, Ain Shams University Hospitals, Cairo, Egypt. Tel: 02-24023494; E-mail: monazaki_810@hotmail.com

Received date: December 21, 2015; Accepted date: January 5, 2016; Published date: January 8, 2016

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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 inflammation in connective tissue destruction and bone loss [7,11]. Herrera et al. [12] 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 Enucleatum and Capnocytophaga spigutena 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.

References


