



Maternal Periodontal Disease and Pregnancy Outcomes



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Submission: 📅 March 05, 2018; **Published:** 📅 March 29, 2018

Editorial

Preeclampsia is a disorder of uncertain etiology of human pregnancy. It defines by high blood pressure and high protein in urine. If it left without treatment, it will change to eclampsia which leads to high rates of morbidity and mortality [1]. Its prevalence is beyond 10% and is the main maternal death. If preeclampsia occurs before 32 weeks of gestation, it is considered as early-onset and is more likely of high morbidity rate. Preeclampsia is associated with multiple maternal and fetal adverse effects [2]. Periodontal disease (PD) is a chronic infection of gingival with dental supportive structures caused by periopathogen bacteria. PD is identified by extensive damage to dental supportive structures as alveolar bone accompanied by forming pocket and gingival recession. This involves of direct tissue damage as a result of bacterial plaque and of indirect by bacterial effects on the immune system [3]. Inflammation and infection play a role in the pathogenesis of preterm birth through various path physiological mechanisms. It occurs in 15% of women and is more in pregnancy which specified by clinical attachment loss. PD is either gingivitis or periodontitis. Interestingly, published data have indicated that preeclampsia cases have significantly higher attachment loss and gingival recession than control cases [4,5].

Periodontitis was assumed as a risk factor for preeclampsia, since infection is involved in etiology and pathogenesis of preeclampsia and as a factor for adverse pregnancy outcomes [5]. Maternal periodontitis is associated with increased threat of induced preterm birth and low birth weight [6]. Thus, PD is a public health problem since it is independent risk factor for adverse pregnancy outcomes; however, it is preventable and curable. The association between PD and preeclampsia has recently been in debate [7]. Periodontitis induced by gram-negative anaerobic bacteria that causes elevations of pro inflammatory mediators. The fetal-placental unit in woman with progressive periodontitis is exposed to inflammatory mediators that participate preterm labor and delivery [4,6,8]. For example, aggressive periodontitis is correlated with elevated levels of interleukin-6 in saliva. When elevated in the serum or amniotic fluid, interleukin-6 predicts preterm labor and delivery. The association between progressive

periodontitis and risk for preterm before 37 weeks has significantly been stated in preeclampsia women. Transmission of bacteria from mouth to placenta and induction of inflammation may harm the endothelium of placental vessels, thus, producing preeclampsia. Periodontal infection causes transient and low- grade bacteremia and end toxemia which promotes inflammatory and immune responses as well as elevated circulating C-reactive protein in PD inducing adverse pregnancy outcomes. Other inflammatory mediators from inflamed periodontal tissue as PGE2 and cytokines inducing inflammation that causes injury to placenta as well as preeclampsia. In general, similarities between preeclampsia and atherosclerosis are stated [9]. Thus, atherosclerosis is associated with endothelial dysfunction and seems it is associated with periodontitis. Other epidemiologic factors as obesity, hypertension and black race are also contributed. One of the reasons for abnormalities in endothelial function is the presence of severe inflammatory responses. The possibility of long-term exposure to inflammatory mediators and/or bacteria is associated with disease progression and necessary to adversely affect birth outcomes. It should also be mentioned that repeated periodontal therapy may activate immune responses, which in turn affect pregnancy outcomes. This may occur through increased TLR-4 expression on placental trophoblasts that is associated with preeclampsia but not preterm labor. The reasons may be of an abnormal thromboplastic invasion of uterine vessels, immunologic mismatched between mother and child, lack of mother's proper compatibility with cardiovascular and inflammatory changes of pregnancy, diet and genetics. However, treatment of PD during pregnancy is safe and is not contraindicated in pregnancy. Thus, control of oral diseases improves the woman's quality of life and is a potential aspect to reduce the transmission of oral bacteria from mothers to children.

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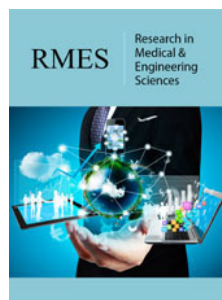
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