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Review Article

An overview of periodontal disease in pregnancy

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ABSTRACT

Periodontal disease (PD), an inflammatory and infectious condition affecting the tissues supporting teeth, is prevalent among pregnant women due to hormonal changes. Studies indicate a link between PD and adverse pregnancy outcomes, including preterm labor and low birthweight newborns, likely due to increased inflammatory mediators. Oral pathogens like *Tannerella forsythia* and *Porphyromonas gingivalis* have been associated with these complications. The most prevalent oral conditions affecting pregnant women that may influence the course of their pregnancy are periodontitis, gingivitis, and dental caries. From supragingival plaques to subgingival infections and PD, these conditions are linked to one another. Elevated maternal serum antibodies to these pathogens and inflammatory markers correlate with poor pregnancy outcomes. Animal models further support the connection between PD and fetal health issues. While research suggests that periodontal treatment may reduce the risk, the exact mechanisms remain complex and multifactorial. Integrating dental care into prenatal care is vital for prevention and management.

Keywords: Maternal PD, Pregnancy, Neonatal complications, Preterm delivery, Low birth weight

INTRODUCTION

Periodontal disease (PD) is an inflammatory and/or infectious disease of the tissues surrounding and supporting the teeth.[1,2] It is estimated that in the United States, PD afflicts over 23% of women between the ages of 30 and 54 years.[3] The most prevalent oral conditions affecting pregnant women that may influence the course of their pregnancy are periodontitis, gingivitis, and dental caries. From supragingival plaques to subgingival infections and PD, these conditions are linked to one another. When PD is at its highest severity, inflammation of all the tissues supporting the teeth, such as the gingiva, cementum, alveolar bone, and periodontal ligaments, can occur. Pregnant women are more likely to acquire PD and dental caries due to the complicated physiological changes that occur during pregnancy. The prevalence of PD in pregnant women varies considerably across different populations. A range of factors, including socioeconomic status, oral hygiene practices, and genetic predisposition, contribute to these variations. Several studies have reported a higher prevalence of PD among pregnant women compared to the general population, with estimates ranging from 30% to 75%.[4] In Saudi Arabia, 70-90% of expectant mothers face one or more dental issues.[5,6] The most common dental issue found was PD (30-60%), which was followed by dental caries and odontalgia (30-40%).[5-7] Research suggests that PD in expectant mothers is linked to adverse pregnancy and neonatal outcomes, such as preterm labor and low birth weight (LBW) newborns.[8-10] This relationship demonstrates biological plausibility because PD raises levels of inflammatory mediators, which are generated in response to periodontal bacteria and their toxins. Prostaglandins (PGE2), which appear to impact placental function and likely have a bearing on initiation of labor

and following fetal health outcomes, are increased by the likelihood of chronic inflammation with raised circulating cytokines, such as tumor necrosis factor-α, interleukin-8 (IL-8), and IL-1β.[11] According to recommendations based on evidence created by a number of professional associations, the initial prenatal visit should involve oral screening to detect signs of PD early on. This review aims to provide an overview of the various aspects of PD in pregnancy and its association with adverse pregnancy outcomes.

LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on November 5, 2023, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed PD in pregnancy. There were no restrictions on date, language, participant age, or type of publication.

DISCUSSION

PD primarily involves inflammation and destruction of the periodontium, comprising the gingiva, periodontal ligament, cementum, and alveolar bone. In pregnancy, hormonal changes, particularly increased levels of estrogen and progesterone, can exacerbate the inflammatory response to dental plaque, leading to gingivitis and potentially advancing to periodontitis.[12] In addition, PD has been linked to several health conditions, including cardiovascular disease, diabetes, respiratory infections, and Alzheimer's disease in recent observational studies.[13-16] While the exact mechanisms are not fully understood, it is believed to involve the direct effects of periodontal bacteria and immune responses that can damage blood vessels.[17] Periodontal infections can also affect glycemic control. Researchers have also explored the connection between periodontal health and adverse pregnancy outcomes, particularly those related to inflammation. Infections and inflammation are associated with preterm delivery and LBW. This has led to investigations into the potential link between placental infection and adverse pregnancy outcomes.[18-20] Additionally, inflammation plays a role in preeclampsia.[21] It is hypothesized that PD may contribute to adverse pregnancy outcomes through systemic infectious and inflammatory processes, and clinical trials have examined the impact of treating periodontal infections on these outcomes.

When oral hygiene is lacking, periodontal bacteria tend to accumulate within the gingival crevice of the teeth and organize themselves into a structured entity known as a bacterial biofilm.[22] The presence of these periodontal pathogens in plaque and saliva samples has been significantly linked to adverse pregnancy outcomes. Notably, *Tannerella forsythia*, *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans* (formerly known as *Actinobacillus actinomycetemcomitans*), *Treponema denticola*, *Fusobacterium nucleatum*, and *Campylobacter rectus* have shown such associations.[23] However, it is important to note that other studies investigating these same pathogens did not find a similar connection.[24,25] It is believed that in developed biofilms, these bacteria possess a range of virulence factors, which include lipopolysaccharide (LPS).[22] These factors have the potential to directly harm the periodontal tissues or prompt the host to initiate a localized inflammatory response. Inflammatory mediators like PGE2 and IL-1β released in response to the presence of these oral pathogens make their way into the gingival crevicular fluid. This response, while aimed at eliminating the infection, can inadvertently result in further damage to the periodontal structures. Additionally, the bacteria themselves or the virulence factors they release may enter the bloodstream, spreading throughout the body and initiating systemic inflammatory responses or infections in distant areas. Researchers have found that elevated levels of inflammatory mediators are positively correlated with an increased risk of adverse pregnancy outcomes.[26,27]

Furthermore, evidence suggests that higher maternal serum immunoglobulin (Ig) G antibodies against periodontal pathogens and elevated cytokine levels are associated with LBW and preterm delivery (PTD), respectively.[11,28] Additionally, higher levels of these inflammatory mediators in amniotic fluid have been shown to increase the risk of preterm delivery.[29]

Lastly, elevated levels of fetal IgM, indicating fetal exposure to periodontal pathogens, have been detected in fetal cord blood samples.[24,30] Taken together, the evidence suggests that the translocation of oral pathogens to the utero-placental unit, along with PD and systemic inflammation, elevates the risk of adverse pregnancy outcomes, including preterm delivery. The ability of periodontal pathogens to trigger local and systemic inflammatory responses has led to the hypothesis that PD may have consequences beyond the mouth. This concept, originally proposed by Miller in 1891 as "focal infection," lacked scientific evidence for many years.[31] However, in the early 1990s, Collins et al demonstrated in animal studies that periodontal infections could lead to pregnancy complications, providing initial evidence of a potential link.[32] Researchers have since explored this association in humans using various research methods.

The mechanisms underlying these associations are complex and multifactorial, involving systemic inflammation, bacterial dissemination, and placental dysfunction. This is why the relationship between PD and adverse pregnancy outcomes has been extensively studied. Epidemiological studies on PD and pregnancy outcomes can be broadly classified into two categories:

case-control and cohort studies. In case-control studies, researchers identify mothers who experienced adverse pregnancy outcomes and compare their history of PD with that of healthy control subjects. A number of case-control studies have reported a significant association between PD and pregnancy complications.[25,33-36] These studies examined various aspects, including the connection between PD and LBW, preterm births, preterm LBW, or pre-eclampsia. Those showing a positive association indicated that pregnant women with PD were up to 7.5 times more likely to experience pregnancy complications. However, it is crucial to interpret these findings cautiously, as case-control studies can sometimes overestimate the strength of associations. In cohort studies, researchers track women over time to assess whether those with PD have a higher incidence of adverse pregnancy outcomes compared to those without the disease. Several published cohort studies have also reported an association between PD and pregnancy complications, and one suggested a possible link.[29,37-41] Similar to case-control studies, cohort studies varied in factors such as sample size, population diversity, definitions of PD, and pregnancy outcomes. Notably, one recent cohort study examined the impact of active PD during pregnancy and found that women with progressing disease were more likely to experience very preterm deliveries compared to those without disease progression.[37]

In recent years, researchers have conducted studies to investigate whether PD is a potentially reversible cause of adverse pregnancy outcomes. These studies randomly divided women with PD into two groups, with one group receiving periodontal treatment during pregnancy while the other did not. The goal was to determine whether periodontal therapy could reduce the incidence of pregnancy complications and establish PD as an independent risk factor for obstetric complications. Few randomized intervention studies have been published where the intervention involved scaling and root planing of all teeth, with or without the use of a chlorhexidine mouth rinse or metronidazole.[42-44] One of the studies reported a 28 percent reduction in preterm LBW births in the group receiving periodontal treatment, but this difference was not statistically significant.[44] Another study suggested that PD is an independent risk factor for preterm LBW, while the third study concluded that scaling and root planing might reduce preterm deliveries.[42,43] Overall, these three studies collectively indicate a positive trend, where periodontal treatment appears to lead to a significant reduction in the rate of preterm delivery and an increase in birth weight. However, the results were not always statistically significant, possibly due to small sample sizes. Additionally, most participants in these studies were black and/or of low socioeconomic status, which are significant risk factors for both PD and preterm birth, making the generalizability of the findings somewhat limited but promising for high-risk populations.

Another line of evidence comes from microbiological studies, which shed light on the potential association between PD and adverse pregnancy outcomes. PD is primarily an infectious disease caused by anaerobic gram-negative bacteria. These bacteria are categorized into different clusters based on their characteristics and colonization patterns in the periodontal sulcus. Several studies have examined the prevalence of these periodontal bacteria and maternal-fetal antibody responses to them in pregnant women.[45] These studies have suggested that mothers without a protective immunoglobulin (Ig) G response against certain bacteria, especially those in the "red" cluster, have a higher rate of preterm deliveries.[24] Moreover, fetuses with a robust IgM response to periodontal pathogens, particularly when accompanied by an inflammatory response, are at a significantly higher risk of preterm birth. These findings indicate that maternal periodontal infection, in the absence of protective maternal antibodies, can lead to the systemic dissemination of oral organisms to the fetus, resulting in preterm deliveries. This also raises the possibility of maternal immunization as a potential protective measure during pregnancy. Furthermore, neonates with elevated IgM antibodies to specific oral pathogens, such as *P. gingivalis* and *C. rectus*, are more likely to be admitted to the neonatal intensive care unit (NICU) and have extended NICU stays.[46] This suggests that these oral pathogens may serve as primary fetal infectious agents, contributing to pregnancy complications.

Additionally, experimental animal models have been employed to explore the mechanistic aspects of the potential association between PD and pregnancy complications. In these models, periodontal bacteria are injected into small chambers implanted subcutaneously in pregnant animals.[32,47,48] The aim is to create a site of infection distant from the fetal-placental unit, mimicking a simplified model of periodontal infection. Results from these studies have shown that maternal infection with periodontal pathogens, such as *P. gingivalis* or *C. rectus*, can negatively impact fetal growth and viability. These bacteria can disseminate from the subcutaneous chamber to maternal organs, the placenta, and fetal tissues, leading to inflammation and tissue damage. This damage can result in reduced fetal size, an increased risk of perinatal death, and potentially long-term neurological consequences for surviving offspring.

Given the potential impact of PD on maternal and fetal health, preventive strategies and therapeutic interventions are crucial. Routine dental care and oral hygiene education should be integrated into prenatal care to identify and manage PD promptly. Periodontal treatment options, including scaling and root planing, should be considered safe during pregnancy and may help reduce the risk of adverse pregnancy outcomes.[49] Moreover, lifestyle modifications, such as smoking cessation and glycemic control for pregnant women with diabetes, can contribute to improved periodontal health.

CONCLUSION

PD in pregnancy is a multifaceted issue with significant implications for both maternal and fetal health. This comprehensive review has provided insights into the epidemiology, pathophysiology, and associations with adverse pregnancy outcomes. A multidisciplinary approach involving obstetricians, dentists, and other healthcare providers is essential to promoting early detection, prevention, and management of PD in pregnant women. By addressing this complex relationship, healthcare professionals can contribute to better maternal and fetal outcomes and ultimately improve public health.

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