ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 09 | September - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

AN OVERVIEW OF MATERNAL PERIODONTITIS AND ADVERSE PREGNANCY OUTCOMES

Dentistry

Dr. Purobi Choudhury Ph.D Scholar (Periodontics) Nirwan University
Dr. H L Gupta Senior Professor and Principal and Controller, Rajasthan Dental College
Dr. Kopal Sharma* Assistant Professor, Pharmacology, SMS Medical College*Corresponding Author

ABSTRACT

Pregnant women are special population group with many physiological changes that they are no exemption from the relationship between the oral disease and systemic condition. Furthermore, some conditions exaggerates or manifest in a different form during pregnancy. A link between adverse pregnancy outcomes and maternal periodontal disease has been studied in many previous study. This review summaries the correlation between periodontitis and pregnancy outcomes in the form of preterm birth.

KEYWORDS

maternal periodontitis, adverse pregnancy outcomes, preterm birth

INTRODUCTION

Pregnancy represents a typical state marked by profound hormonal, vascular, and immunologic changes that affect multiple systems, including the oral cavity.[1] Among these, periodontal diseases—chronic infections of the tooth-supporting structures caused primarily by gram-negative anaerobic bacteria—are common in women of reproductive age. During pregnancy the prevalence of gingivitis and periodontitis increases due to elevated levels of estrogen and progesterone that modify the host response and vascular permeability of gingival tiss[2]Emerging evidence suggests that maternal periodontal disease (PD) may act as an independent risk factor for adverse pregnancy outcomes (APOs) such as preterm birth (PTB), low birth weight (LBW), preeclampsia (PE), and gestational diabetes mellitus (GDM). Understanding this association has significant clinical implications, as PD is a modifiable and treatable condition.

Epidemiology

Globally, periodontal disease in pregnancy is common, generally affecting about 40–70 % or more of pregnant women depending on the diagnostic threshold. Bleeding on probing (BOP) and gingival changes tend to peak in second to third trimesters due to hormonal modulation of the gingival inflammatory response.[3] In India often the prevalence rate exceeds 50%, with variation across states and urban/rural settings.[4]

Pathophysiology

In the last two decades, more research findings concerning the relationship between periodontal diseases (PD) and adverse pregnancy outcomes (APOs) have emerged.[5-7] Understanding the relationship sufficiently, undermines that the underlying biological mechanisms must be established to better comprehend the chances of a causative relation. Periodontal diseases are defined as a chronic bacterial infection that is progressive in nature with tissue destruction in the supportive environment of the teeth.[7] It is a complex interaction of virulent pathogens and the host's immune response and environmental influences in the presence of hormonal and nutritional influences.[8] Pregnancy is an immune-modulated state with increased inflammatory reactivity, increasing the susceptibility of the oral cavity to the progression of diseases and their systemic manifestations.[9]

A)Hormonal Modulation during Pregnancy

In pregnancy, for instance, hormonal changes characterized by high levels of estrogen and progesterone affect gingival blood vessels, connective tissue metabolism, and the immune system.[10] This promotes more fluid loss, blood vessel leakage, which are vital for the migration of inflammatory cells and facilitate the growth of subgingival flora like Prevotella intermedia and Porphyromonas gingivalis.[11] In addition, progesterone influences the synthesis of prostaglandins, which increases the synthesis of prostaglandin E2 (PGE2), a key mediator in uterine contractions and periodontal tissues.[12] Therefore, numerous pregnant women experience "pregnancy gingivitis" in the absence of any considerable amounts of plaque formation; therefore, the condition progresses to periodontitis.[11,12]

B)Systemic Inflammatory Pathway

The most widely accepted mechanisms linking PD and APOs is the systemic inflammatory hypothesis.[13,14] Periodontopathic bacteria, especially gram-negative anaerobic bacteria, release endotoxins, e.g., lipopolysaccharides (LPS). These stimulate in situ the local production of pro-inflammatory cytokines, e.g., IL-1β, IL-6, TNF-α, and PGE2.[14] These cytokines release into the general circulation, causing an inflammatory cascade, which will affect other organs, e.g., the placenta.[15] Elevated levels in maternal blood, e.g., IL-6, TNF-α, and C-reactive protein, have been shown to be associated with adverse pregnancy outcomes, preterm labor, preeclampsia, and intrauterine growth retardation.[16]

C)Microbial Dissemination and Direct Infection Pathway

Another pathway proposed for the mechanism of infection is the hematogenous dissemination of periodontal pathogens or their virulence factors to the fetoplacental Unit.[17] Infections of the period-ontium lead to several incidents of transient bacteremia with the initiation of tooth brushing, chewing, and periodontal probing.[17] Bacteria then disseminate to the placenta, amniotic fluids, or tissues. Oral pathogens, for example, Fusobacterium nucleatum, Porphyromonas gingivalis, and Campylobacter rectus, in placental tissues and umbilical cord blood from women with preterm births are a strong evidence of this pathway of infection.[18,19] These pathogens are capable of inducing local inflammation in the placenta, leading to villitis and impaired trophoblast invasion and resulting in endothelial disorder.[19] Experimental studies carried out on animals have shown us that infection by F. nucleatum leads to miscarriage and premature birth of the infant, giving us experimental evidence of this occurring.[19] LPS, the bacterial endotoxin, works by inducing TLR2 and TLR4 in the placenta, culminating in the production of inflammatory cytokines, oxidative stress, and apoptosis, leading to compromise of the trophoblast barrier.[20]

D)Host Immune Response and Oxidative Stress

The state of pregnancy is associated with a shift from a Th1 cell-mediated immune response to a T-helper cell 2 (Th2)-predominant response, necessary for tolerance to the fetus. However, with PD, a constant inflammatory response mediated by Th1/Th17 is maintained, causing widespread immune dysregulation[8] This immune dysregulation could result in endothelial activation and oxidative stress, which play a critical role in the development of preeclampsia.[21] A higher level of ROS generation and lipid peroxidation byproducts has been seen in patients with both PD and preeclampsia, showing that their etiology shares a common pathway.[22] Inflammatory responses associated with PD can also increase insulin levels, thereby linking its role to gestational diabetes and hypertension.[8]

E) Genetic and Epigenetic Modulation

New evidence underscores the significance of genetic variations and epigenetic modifications in influencing vulnerability to both PD and APOs.[6] Gene variants that encode cytokines like IL-1β, IL-6, and TNF-α are linked to elevated inflammatory responses and a higher risk of PTB in women with PD.[6] Epigenetic modifications, such as DNA methylation of inflammatory genes caused by periodontal pathogens, could additionally affect placental immune function.[20] These molecular findings indicate

that maternal genetic factors might influence the relationship between periodontal inflammation and obstetric risk.

DISCUSSION

Assessment of maternal periodontitis and preterm birth

Vidhale P et al, 2020[23] evaluated the association between the maternal periodontal health and preterm birth of infants in 90 patients. The mean clinical attachment loss (CAL) was significantly greater in the test group (45 patients having preterm delivery and low birth weight infants) as compared to control group (45 patients having full term delivery with normal birth weight infants).

Rani Balaji VC, et al 2021[24] conducted a study to identify the relationship of gingival health in the first trimester of pregnancy and on birth weight of newborn. In the study group with 121 pregnant women, those with poor periodontal health results in 7.5 fold increased risk of low birth weight (LBW).

Sinha A et al, 2022[25] conducted a retrospective observational case control study to identify if the prevalence of a periodontal infection was connected to Preterm Low Birth Wight after controlling for other known obstetric risk factors and confounders. The study included Group I (control) that is the infants born at 37 weeks or more with a birth weight of more than 2500 g, Group II (case) was the larger group and included the following subsets: Subgroup A (preterm normal weight), Subgroup B (term low birth weight) and Subgroup C (preterm low birth weight). The study found that the prevalence of periodontal disease was higher in the cases (mean score: 2.3693, SD: 0.8772). Thus, a statistically significant link was established between a high periodontal index score and a higher risk of adverse birth outcomes using the Mann-Whitney U test ($p < 0.05$).

In an narrative literature review [26] 2024 that aimed to study the relationship between periodontal disease in pregnant women and the birth of premature babies, it was found that periodontal disease in pregnant mothers is related to the birth of premature babies, with low weight or with both problems, having scientific evidence and sufficient basis to reach this conclusion.

V Anu et al, 2023[27] conducted a case control study in Chennai with 30 rural pregnant women to evaluate the association between preterm labour or low birth weight (LBW) and maternal periodontitis. The study found that the pregnant women with periodontal disease are 56 times risk of preterm labour and low birth weight babies and Chi-square value = 16.425 ($p = 0.000$).

Another Mexican study[28] 2024, determined if periodontitis correlates with the development of PB and LBW. The study was undertaken with 30 participants and it was found through an ANOVA test, that the clinical parameters that defined the presence and severity of periodontitis (BOP, BIOFILM %, PD, CAL), with birth conditions (normal, PB, LBW, and PLBW) were analyzed, no differences in the means were observed within each group, i.e., no significant differences were no in the clinical parameters between patients with normal birth, PB, LBW or PLBW.

Temur I et al, 2025[29] through their research aimed to assess maternal oral health's effects on inflammatory blood markers and determine a possible relationship with preterm low birth weight (PLBW). A statistically significant difference in oral health was recorded, with women delivering low birth weight infants exhibiting poorer outcomes than control groups. Analysis of secondary outcomes indicated that oral health is not an independent predictor of preterm birth; instead, its contribution may be indirect and through systemic inflammation.

Previous studies have also shown the correlation between maternal age and preterm birth with increased maternal age being correlated with increased risk of preterm birth.[30-32] Previously Muwazi et al, also failed to observe a relationship between maternal age and preterm birth.[33] A meta-analysis study by Porto et al, 2021 found that periodontitis among pregnant women was linked with a low birth weight and might be greater than twice as probable.[34] In an earlier research, which established that those women who had been subjected to violence during pregnancy had a 5.3 times higher risk of premature births compared to other women with no history of violence during pregnancy. [32]

CONCLUSIONS

A link between pregnancy complications and maternal periodontal disease of the mother has been demonstrated in various studies. Cohort studies conducted at the time of birth can be useful to make inferences regarding the cause of the disease in the early life, and such studies on oral health can provide information on the correlation between oral health of expecting mothers and associated outcomes of pregnancy.

REFERENCES:

[1] Sachelarie L, Iman A E H, Romina M V, Huniadi A, & Hurjui L L. Impact of hormones and lifestyle on oral health during pregnancy: A prospective observational regression-based study. Medicina. 2024; 60(11):1773
[2] Wu M, Chen SW, Jiang SY. Relationship between gingival inflammation and pregnancy. Mediators Inflamm. 2015; 2015:623427.
[3] Chen P, Hong F, Yu X. Prevalence of periodontal disease in pregnancy: A systematic review and meta-analysis. J Dent. 2022;125:104253.
[4] Shaju JP, Zade RM, Das M. Prevalence of periodontitis in the Indian population: A literature review. J Indian Soc Periodontol. 2011;15(1):29-34.
[5] López-Valverde N, Quispe-López N, & Blanco Rueda J A. Inflammation and immune response in the development of periodontal disease: A narrative review. Frontiers in Cellular and Infection Microbiology. 2024; 14:1493818.
[6] Fine N, Chadwick J W, Sun C, Parbhakar K K, Khoury N, Barbour A et al. (2021). Periodontal inflammation primes the systemic innate immune response. Journal of Dental Research. 2021; 100(3), 318–25.
[7] Hajishengallis, G. Periodontitis: From microbial immune subversion to systemic inflammation. Nature Reviews Immunology. 2015; 15(1), 30–44.
[8] Tattar R, da Costa B D C, & Neves V C M. The interrelationship between periodontal disease and systemic health. British Dental Journal. 2025; 239(2): 103–08.
[9] Cetin I, Pileri P, Villa A , Calabrese S, Ottolenghi L, & Abati, S. Pathogenic mechanisms linking periodontal diseases with adverse pregnancy outcomes. Reproductive Sciences. 2012; 19(6), 633–641.
[10] Shamsi M, Hidarnia A, Niknami S, Rafiee M, & Karimi M. Oral health during pregnancy: A study from women with pregnancy. Dental Research Journal. 2013; 10(3): 409–410.
[11] Arbildo Vega H I, Padilla-Cáceres T, Caballero-Apaza L, Cruzado-Oliva, F H Mamani-Cori V, Cervantes-Alagón S, et al. Effect of treating periodontal disease in pregnant women to reduce the risk of preterm birth and low birth weight: An umbrella review. Medicina.2024; 60(6): 943.
[12] Martínez-García M, & Hernández-Lemus E. Periodontal inflammation and systemic diseases: An overview. Frontiers in Physiology. 2021; 12: 709438.
[13] Offenbacher S, Katz V, Fertik G, et al. Periodontal infection as a possible risk factor for preterm low birth weight. Journal of Periodontology. 1998; 69(10): 1103–1113.
[14] Scannapieco F A , & Cantos A. Oral inflammation and infection, and chronic medical diseases: Implications for the elderly. Periodontology. 2016; 72(1): 153–175.
[15] Madianos P N, Bobetsis Y A, & Offenbacher S. Adverse pregnancy outcomes and periodontal disease: Pathogenic mechanisms. Journal of Clinical Periodontology; 2013; 40(Suppl 14): S170–S180.
[16] Canzoneri BJ, Lewis DF, Groome L, Wang Y. Increased neutrophil numbers account for leukocytosis in women with preeclampsia. Am J Perinatol. 2009; 26(10):729-32.
[17] Han YW, Wang X. Mobile microbiome: oral bacteria in extra-oral infections and inflammation. J Dent Res. 2013; 92(6):485-91.
[18] Han YW, Fardini Y, Chen C, Iacampo KG, Peraino VA, Shamonki JM, et al. Term stillbirth caused by oral Fusobacterium nucleatum. Obstet Gynecol. 2010 Feb;115(2 Pt 2):442-45.
[19] Katz J, et al. Presence of periodontal pathogens in placental tissues. Placenta. 2021; 104: 1–7.
[20] Bobetsis YA, Graziani F, Gürsoy M, Madianos PN. Periodontal disease and adverse pregnancy outcomes. Periodontol 2000. 2020; 83(1):154-174.
[21] Brown MA, Magee LA, Kenny LC, Karumanchi SA, McCarthy FP, Saito S, et al. International Society for the Study of Hypertension in Pregnancy (ISSHP). Hypertensive Disorders of Pregnancy: ISSHP Classification, Diagnosis, and Management Recommendations for International Practice. Hypertension. 2018;72(1):24-43.
[22] Rahajoe P S, et al. Oxidative stress markers in periodontal disease and preeclampsia. Journal of Clinical Medicine. 2021; 10(4): 1234.
[23] Vidhale P, Puri S, & Bhongade M L. Maternal periodontal disease and preterm low birth weight. Clinical Epidemiology and Global Health. 2020; 8(4), 1152–54.
[24] Balaji V C, Saraswathi K, & Manikandan S. Periodontal health in first trimester and birth outcomes. Indian Journal of Dental Research.2021; 32(2), 181–86.
[25] Sinha A, Singh N, Gupta A, Bhargava T, Mahesh P C, & Kumar P. Periodontal status and preterm/low birth weight. Cureus. 2022; 14(11): e31735.
[26] Santos P G, Ribeiro A C, Travassos R M, da Paz Júnior F, da Paz E S, & Guaraná C F. Periodontal disease and premature birth. Lumen et Virtus. 2024; 15(39): 3493–506.
[27] Anu V, Sowndarya V, Rajkumari G S, Sravya G, Suganya K, & Subha S. Maternal periodontitis and adverse outcomes. International Journal of Scientific Research Archive.2023; 9(1), 277–280.
[28] Zárate-Rodríguez H E, Espinosa-Arreola M, Rocha-Rocha V M, et al. Periodontitis during pregnancy. Oral. 2024;25(77): 2278–2284.
[29] Temur I, Temur K T, Dönertas S N, Dönertas A D, & Kacmaz M. Maternal oral health and neonatal outcomes. BMC Pregnancy and Childbirth. 2025; 25(1): 231.
[30] Londero A P, Rossetti E, Pittini C, et al. Maternal age and outcomes. BMC Pregnancy and Childbirth. 2019; 19(7): 261.
[31] Soltani M, Tabatabaee HR, Saeidinejat S, Eslahi M, Yaghoobi H, Mazloumi E, et al. Assessing the risk factors before pregnancy of preterm births in Iran: a population-based case-control study. BMC Pregnancy Childbirth. 2019;19(1):57.
[32] Uwambaye P, Munyanshongore C, Rulisa S, Shiau H, Nuhu A, Kerr MS. Assessing the association between periodontitis and premature birth: a case-control study. BMC Pregnancy Childbirth. 2021;21(1):204.