Chapter

Periodontal Disease and Pregnancy Outcome

Girish Suragimath

Abstract

Periodontal diseases are silent infections that often go undiagnosed until irreparable damage occurs to the teeth and oral structures. These chronic oral infections are characterized by the presence of a biofilm matrix that adheres to the periodontal structures and serves as a reservoir for bacteria (plaque). Response of the body toward the bacterial challenge of dental plaque can lead to bone loss and the migration of the junctional epithelium, resulting in periodontal pocketing and periodontal disease. This bacterial insult can result in destruction of the periodontal tissues that precipitates a systemic inflammatory and immune response leading to the release of several cytokines and immunomodulatory agents, which may affect systemic conditions and diseases. The influence of periodontal infection on systemic disease and conditions documented include coronary heart disease (CHD) and CHD-related events such as angina, infarction, atherosclerosis, and other vascular conditions; stroke; diabetes mellitus; preterm labor, low birth weight delivery, and preeclampsia; and respiratory conditions such as chronic obstructive pulmonary disease. Adverse pregnancy outcomes, including preeclampsia, preterm delivery, and intrauterine growth restriction, and fetal demise affect a significant number of pregnancies and are a major source of both maternal and neonatal morbidity and mortality. This chapter highlights the two-way relationship between periodontitis and adverse pregnancy outcome.

Keywords: adverse pregnancy outcome, preterm birth, low birth weight baby, periodontal disease, periodontal therapy

1. Introduction

Periodontitis has prevailed in human history from the dawn of civilization and still is a major cause of tooth loss in adult population. The etiology of periodontal disease (PD) is complex in nature, and it is a multifactorial disease, which is largely influenced by genetic, environmental, and microbial factors [1]. The periodontal disease begins at the gingiva and progress downwards and affects the tooth-supporting structures, i.e., periodontal ligament, cementum of the root, and alveolar bone. The clinical features of periodontitis are bleeding from the gums, pus discharge, dull gnawing pain, bad breath, mobility of teeth, pathological tooth migration, gingival recession, and exfoliation of teeth in severe cases.

Periodontal disease results from a complex interplay between the subgingival biofilm and the host immune inflammatory event, which develop in the gingival and periodontal tissues in response to the challenge presented by the bacteria [2]. The bacteria may initiate the disease, but the progression is host immune-mediated,

and several inflammatory cells and enzymes are released which have a detrimental effect on other cells, tissues, and organ systems. There is a shift from healthy nonpathogenic flora to a huge virulent and infectious anaerobic flora in the periodontal disease. These bacteria and their toxins and various pro-inflammatory mediators penetrate into systemic circulation. The penetration of bacterial toxins and host-mediated immunomodulatory mediators into systemic circulation can have a toxic effect on the cells and organs elsewhere in the body. Environmental, physical, social, and host stresses may affect and modify disease expression through a multitude of pathways.

Systemic diseases tend to increase the periodontitis progression and can complicate the treatment of periodontal diseases. Periodontal infection may significantly enhance the risk for certain systemic diseases or alter the natural course of systemic conditions. There is a two-way relationship between periodontal disease and systemic disease or condition in an individual. The influence of periodontal infection on systemic disease and condition documented includes coronary heart disease (CHD) and CHD-related events such as angina, infarction, atherosclerosis, and other vascular conditions; stroke; diabetes mellitus; preterm labor, low birth weight delivery, and preeclampsia; and respiratory conditions such as chronic obstructive pulmonary disease [3].

Adverse pregnancy outcomes have been attributed to infections and inflammatory conditions in the vagina and elsewhere in the body. The potential role of chronic bacterial infections elsewhere in the body remote from the fetal-placental unit, which may influence the health and growth of babies in the placenta, has been studied immensely. This realization that infection in any part of the body can affect the placenta has led to the idea that periodontal disease can be a possibility in adverse pregnancy outcome. Local elevation of pro-inflammatory prostaglandins and cytokines due to "chronic gram-negative infection" in the periodontal diseases can be a risk factor [4]. Periodontal diseases have shown to increase the systemic levels of some of these inflammatory mediators [5]. Periodontal disease has a possibility to influence pregnancy outcome through an indirect mechanism, involving inflammatory mediators or a direct bacterial assault on the amnion and causing preterm low birth weight babies (PLBW). This chapter highlights the bi-directional relationship between pregnancy, pregnancy outcome, and periodontal disease.

1.1 Focal infection theory revisited

William Hunter, a British physician in 1900, first developed the idea that oral microorganisms were responsible for a wide range of systemic conditions that were not easily recognized as being infectious in nature. Hunter also identified gingivitis and periodontitis as foci of infection and advocated the extraction of teeth with these conditions to eliminate source of sepsis. He also thought that oral organisms had specific actions on different tissues and that these organisms were acted by producing toxins, thereby resulting in low-grade superinfection that produce systemic effect over prolonged periods. The Hunter theory became widely accepted, thereby leading to wholesale extraction of teeth. The focal infection theory fell into disrepute during the 1940s and 1950s when widespread extraction failed to reduce or eliminate the systemic conditions. However, Hunter ideas did encourage extensive research in the areas of microbiology and immunology. The Hunter theories are being revived today in light of recent research demonstrating links between oral and systemic health. Today's era of evidence-based medicine and dentistry provides an excellent environment in which to examine the possible relationship between oral infection and systemic disorders.

The first association between periodontal disease and preterm low birth weight babies was documented by Offenbacher and colleagues in 1996 using a case-control study design. The study by Offenbacher et al. [6] suggested that maternal periodontal disease could lead to a sevenfold increased risk of delivery of a preterm low birth weight infant. Human case-control studies have demonstrated that women who have low birth weight infants as a consequence of either preterm labor or premature rupture of membranes tend to have more severe periodontal disease than mothers with normal birth weight infants [7].

1.2 Pregnancy and its outcome

Every pregnant woman who is carrying a live baby in her womb wishes to deliver a healthy baby. There are numerous genetic, pathological, and environmental factors that can affect the growth and development of the baby in the womb. During the course of a normal pregnancy, a series of profound and dynamic physiological changes occur in both the mother and developing baby [8]. Pregnancy and parturition involve a complex series of molecular and biological events for mother and fetus. Pregnancy by itself does not cause periodontal diseases, but the hormonal changes during pregnancy accentuate the gingival response to plaque and modify resultant clinical picture.

Medical science aims at reducing the risk factors involved in the growth and development of a baby in the womb. Adverse pregnancy outcomes including preeclampsia, preterm delivery, intrauterine growth restriction, and fetal demise affect a significant number of pregnancies and are a major source of both maternal and neonatal morbidity and mortality. The Centers for Disease Control and Prevention (CDC) advocates that babies born with less than 5.5 pounds or 2.5 kg will be at risk of long-term health problems such as delayed motor skills, social growth, or learning disabilities. Babies born, at least 3 weeks, earlier than its due date have also risk for retarded growth and development [9]. Respiratory problems, vision and hearing loss, or feeding and digestive problems are other problems associated with preterm and low birth weight babies.

Adverse pregnancy outcomes (APOs) are serious events that every year cause the death or disability of many newly born infants worldwide [10]. The most common adverse pregnancy outcomes are represented by low birth weight (LBW), preterm birth (PTB), and preeclampsia (PE). Adverse pregnancy outcomes represent an important health issue which affects not only the infant but also the mother, and more than half a million women die each year from related causes. About 10–15% of maternal death during pregnancy is associated with PE and eclampsia, which could affect the liver, kidneys, brain, and the clotting system.

World Health Organization (WHO) in 1995 defined low birth weight (LBW) as any live birth of <2500 g and very low birth weight to be <1500 g. WHO defines preterm birth as any live birth at <37 weeks of gestation period [11, 12]. More than 33% of the infant mortality is attributed to the preterm low birth weight (PTLW), and surviving infants also have increased morbidity to congenital, neurological disabilities, and various developmental defects.

Little reduction in incidence of adverse pregnancy outcomes has occurred despite advances in technology, promotion of prenatal care, and continued scientific efforts. Investigations to detect the potential causative factors for adverse pregnancy outcome include infection and/or inflammation in the reproductive tract and at sites remote from the feto-placental unit. The relationship between adverse pregnancy outcomes and maternal periodontal infections has been studied extensively over the past 10 years, as periodontal infection is most prevalent in populations with highest risk of adverse pregnancy outcomes.

1.3 Maternal immunological changes during pregnancy

Previously, it was believed that there was little or no exposure of the mother to the immunologically foreign cells of the fetus. It is now clarified that there is considerable mixing of maternal and fetal cells, especially at the maternal-fetal interface.

One of the major alterations in the immune system during pregnancy is partial dampening of the mother's cell-mediated immune responses associated with T-helper type 1 (Th1) lymphocytes. Stimulated Th2 cells produce an array of cytokines, such as interleukin-4, interleukin-5, and interleukin-10, which suppress cell-mediated immune responses [13–15]. The mechanisms of this partial shift in the Th1/Th2 balance favoring Th2-mediated immune responses are not fully understood but are partly dependent on changes in progesterone, estrogen, and chorionic gonadotropin during pregnancy [8, 16].

Neutrophils in the peripheral circulation of pregnant women exhibit a significant reduction in myeloperoxidase, respiratory burst activities, and phagocytosis [8]. All of these inhibitory effects on neutrophils are most marked during the second and third trimesters [17, 18]. The postpartum readjustment of the mother's immune system occurs soon after birth, with rapid re-establishment of several Th1-associated and other pro-inflammatory host responses (**Table 1**). The phenomenon of return of immunological response after postpartum has been termed the "immune reconstitution syndrome" [15].

1.4 Gingival pyogenic granulomas in pregnancy

Pyogenic granuloma (PG) or pregnancy tumor is a non-specific inflammatory lesion of the skin and mucous membranes. PG occurs in both males and females as inflammatory lesion on skin or mucous membrane. PG occurs approximately 0.5–2.0% of pregnant women with gingival lesions developing in interdental gingiva (**Figure 1**). They are also called pregnancy tumors or granuloma gravidarum.

Components	Changes in host response
Innate immunity	
Monocytes and neutrophils	Effect on cellular immunity via enhanced phagocytosis and superoxide anion generation (respiratory burst); increased expression of CD14
Natural killer cells	Effect on cellular immunity via downregulation of cytotoxic activity by progesterone-induced blocking factor and IL-10; decreased IFN-c production
Complement	Effect on humoral immunity by increased C3, C4, and C1q levels and elevated levels of complement regulatory proteins including membrane cofactor protein (CD46), decay accelerating factor (CD55), and CD59
Acute-phase reactants	Effect on humoral immunity via increased levels of acute-phase reactants (e.g., fibrinogen and ceruloplasmin)
Adaptive immunity	
T cells	Effect on cellular immunity via enhanced Th2 (e.g., IL-4, IL-10) and Th3 (i.e., TGF-b) and suppressed Th1 (IFN-c, IL12) responses Effect on humoral immunity via increased T cell-dependent immunoglobulin production
B cells	Effect on cellular immunity via increased Th2-induced B-cell activity IL, interleukin; IFN, interferon; Th1, T-helper type 1 lymphocytes; Th2, T-helper type 2 lymphocytes; TGF, transforming growth factor

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Table 1.
Innate and adaptive immunity changes during pregnancy [8]

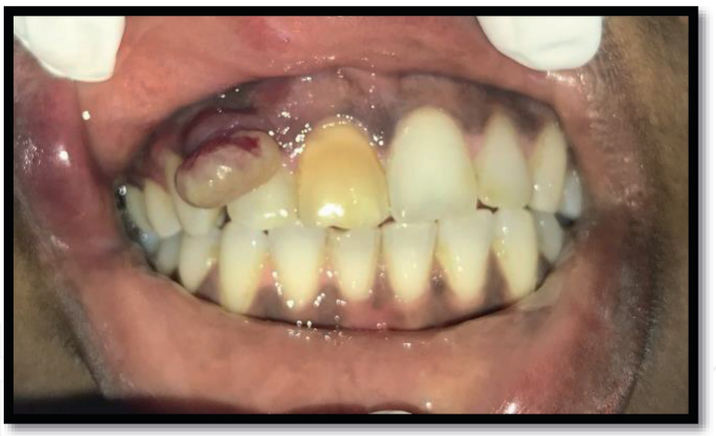

Figure 1.
A case of pyogenic granuloma in maxillary right lateral incisor region in a 9-month pregnant woman.

The lesion frequently presents as a rapidly growing gingival mass that may bleed profusely when touched. Based on histological features, it is a highly proliferative vascular lesion resembling granulation tissue. The etiological triggers for pyogenic granuloma are unknown; most lesions are associated with the presence of local irritants or trauma [19]. The pathogenesis of the lesion has been linked to female sex hormones, which stimulate increased local synthesis of angiogenic factors such as vascular endothelial growth factor and angiopoietin-2 [8]. Clinical complaints with pregnancy-associated pyogenic granulomas include gingival bleeding, tenderness, and esthetic problems. Treatment may include surgical removal, especially if the lesion is large and symptomatic [19, 20]. However, in many cases, the lesions undergo partial or complete resolution after delivery, especially if local irritants are removed [8].

1.5 Plaque-induced periodontal infections in pregnancy

Experimental gingivitis study of women during pregnancy and at 6 months postpartum showed that there was more gingival inflammation during pregnancy despite no significant differences in plaque scores. Cross-sectional studies indicate that 100% of women develop gingivitis between 3 and 8 months of their pregnancy, with a gradual decrease after parturition [21]. In some cases, the gingival inflammation is very severe and may be accompanied by gingival tenderness and profuse bleeding. Longitudinal studies have demonstrated that, during pregnancy, probing depths increase as the gingival inflammation increases. The increase in probing depths has been attributed to movement of the gingival margin in a coronal direction because of inflammation-induced swelling of the gingiva. Most authors have found that there is usually no permanent loss of clinical attachment [22, 23]. Individuals, especially those who have chronic periodontitis prior to becoming pregnant, tend to have increased rate of progression of periodontitis. Several standard cultural microbiological studies have shown that estrogen and progesterone changes associated with pregnancy have an effect on the composition of the subgingival microbiota. Some of the periodontal pathogens that apparently blossom under the selective pressure of pregnancy-associated steroids are *Prevotella intermedia*, *Bacteroides* species, and *Campylobacter rectus* [8].

Diverse array of pathogens that have the potential to cause periodontal tissue damage have been found in pregnant and parous women through microbiological studies using DNA probes [24, 25]. Several types of spirochetes, including *Treponema denticola*, as well as numerous gram-positive and gram-negative putative periodontal pathogens, are found in pregnant and nonpregnant women. Prominent gram-positive bacteria in this group are *Streptococcus intermedius*, *Parvimonas micra*

(formerly *Micromonas micros* and *Peptostreptococcus micros*), *Peptostreptococcus anaerobius*, *Staphylococcus aureus*, and *Actinomyces odontolyticus*. Frequently detected gram-negative organisms include *Porphyromonas gingivalis*, *Tannerella forsythia*, *C. rectus*, *P. intermedia*, *Prevotella nigrescens*, *Fusobacterium nucleatum*, *Eikenella corrodens*, *Selenomonas noxia*, *Enterococcus faecalis*, *Pseudomonas aeruginosa*, *Haemophilus influenzae*, and *Aggregatibacter actinomycetemcomitans*. [8] As the immunological changes associated with pregnancy include an increased susceptibility to intracellular pathogens, it is not surprising that survival of locally invasive bacteria such as *P. intermedia* and A. actinomycetemcomitans is enhanced during pregnancy [8].

Gingivitis and periodontitis are plaque-induced periodontal diseases, which are multifactorial infections involving innate and adaptive immune responses of the host toward tooth-associated microbial biofilms (plaque). Pregnant women undergo a lot of physiological and immunological changes during pregnancy. These changes in pregnancy have profound effects on the host-parasite interactions found in microbial infections. The exact mechanisms responsible for the increased gingival inflammation during pregnancy are not fully understood. It is clear that perturbations in neutrophil function, modifications in cellular and humoral immunity, hormone-induced changes in cellular physiology, and local effects on microbial ecology all play crucial roles [8].

1.6 Periodontal infections and gestational diabetes mellitus

Gestational diabetes mellitus (GDM) refers to the detection of glucose intolerance or raised blood glucose level, for the first time during pregnancy in a woman. Gestational diabetes occurs in approximately 7% of women during pregnancy, and it is a multifactorial disease. GDM has been associated with a long list of risk factors [26]. The increased blood sugar levels make the pregnant woman more susceptible for periodontal diseases.

Periodontitis and diabetes are both risk to each other, and studies have proved that increased diabetes was correlated with increased severity of periodontitis. Studies have been conducted on association of microorganisms and gestational diabetes, and contradictory results are obtained. Several study groups concluded that there appears to be an association between periodontal disease and gestational diabetes mellitus, but prospective studies with large enough sample sizes are required to confirm a relationship [8].

1.7 Preeclampsia in pregnant woman and periodontal infections

Preeclampsia is a condition characterized by hypertension, with blood pressure higher than 140/90 mmHg, and the patient also suffers from peripheral edema and proteinuria (i.e., urinary excretion of ±300 mg protein in 24 h) [27, 28]. Eclampsia occurs when there is a failure to control physiological abnormalities in a pregnant woman leading to convulsions, coma, and death of the mother.

Multiple factors are involved in the etiology of preeclampsia including infection, genetic susceptibility, immune responses, abnormal placentation secondary to hypoxia and impaired arterial remodeling, and a markedly enhanced systemic inflammatory burden. Increased risk of preeclampsia is seen with elevated serum levels of C-reactive protein; periodontal infections contribute to the increased C-reactive protein level [29, 30]. Therefore, it is biologically plausible that periodontal infections could play a part in the multifactorial etiology of preeclampsia. The link between periodontal disease and risk of preeclampsia is proved only in few populations and has not been confirmed in all populations [8].

1.8 Two-way relationship between periodontitis and pregnancy

Periodontal disease (PD) per se causes little clinical features and goes unnoticed until late in disease status. The tissue destruction is characterized by the formation of periodontal pocket that acts as reservoirs for bacterial colonization in the dento-gingival environment.

Multiple factors have been associated with preterm baby (PB) and/or LBW such as smoking, drug use, high or low maternal age, low socioeconomic strata, inadequate prenatal care, low maternal body mass index (BMI), hypertension, genitourinary tract infections, cervical incompetence, diabetes, low nutritional status, stress, and multiple pregnancies [31]. However, more than 50% of the cases do not show the presence of these risk factors and are still affected by PB and/or LBW [15]. The search continues for other causes including the presence of the chronic infectious diseases like periodontal infection.

The hypothesis that infection remote from the fetal-placental unit may influence PLBW has led to an increased awareness of the potential role of chronic bacterial infections elsewhere in the body. Periodontal disease is associated with a "chronic gram-negative infection" of the periodontal tissues which results in long-term local elevation of pro-inflammatory prostaglandins and cytokines [8] and an increase in the systemic levels of some of these inflammatory mediators [20]. Hence, periodontal disease has a potential to influence PLBW through an indirect mechanism, involving inflammatory mediators or a direct bacterial assault on the amnion [28].

Multiple factors have been associated with the delivery of preterm and low birth weight infants. The evidence suggests that an infectious etiology is the main cause for a large percentage of cases for preterm birth. Genitourinary tract infections, such as bacterial vaginosis, and inflammatory mediators resulting from such infections have been considered a biologically plausible pathway for preterm labor and premature rupture of the membranes. Alternatively, it was hypothesized that preterm low birth weight may be indirectly mediated through distant infections resulting in translocation of bacterial vesicles and lipopolysaccharide (LPS) in the systemic circulation. However, the exact mechanisms for the proposed relationship remain unclear. The periodontal infection is initiated by predominantly gram-negative, anaerobic, and microaerophilic bacteria that colonize the subgingival area. Host defense mechanisms play integral role in the pathogenesis of periodontal disease. It has been postulated that the association between periodontal disease and preterm low birth weight (PLBW) may have similar pathogenic mechanisms as other maternal infections [32]. Inflamed periodontal tissues produce significant amounts of pro-inflammatory cytokines, mainly interleukin 1 (IL-1b), IL-6, prostaglandin E2, and tumor necrosis factor-alpha (TNF-α), which may have systemic effects on the host, leading to premature rupture of membrane. Hence, periodontal disease has the potential to influence preterm low birth weight through an indirect mechanism involving inflammatory mediators or a direct bacterial assault on the amnion [8, 33] (**Figure 2**).

The inflammation and infection caused during the periodontal disease is not just limited to the oral cavity but also enters the systemic circulation. The systemic immune response gets activated due to the episodes of bacteraemia and dissemination of endotoxins from periodontal pockets. Systemic circulation may induce pro-inflammatory cytokine production due to the presence of bacteria or bacterial endotoxins in the systemic circulation. IL-6 and C-reactive protein that are released during chronic low-grade inflammation are further activated due to presence of cytokines in the systemic circulation. The endothelial dysfunction may result due to inflammatory response of endothelial cells. The immune response plays a pivotal role in maintaining a healthy equilibrium between the mother and fetus, during pregnancy. The specific immune response is shifted toward a Th2-type immune response, and the inflammatory

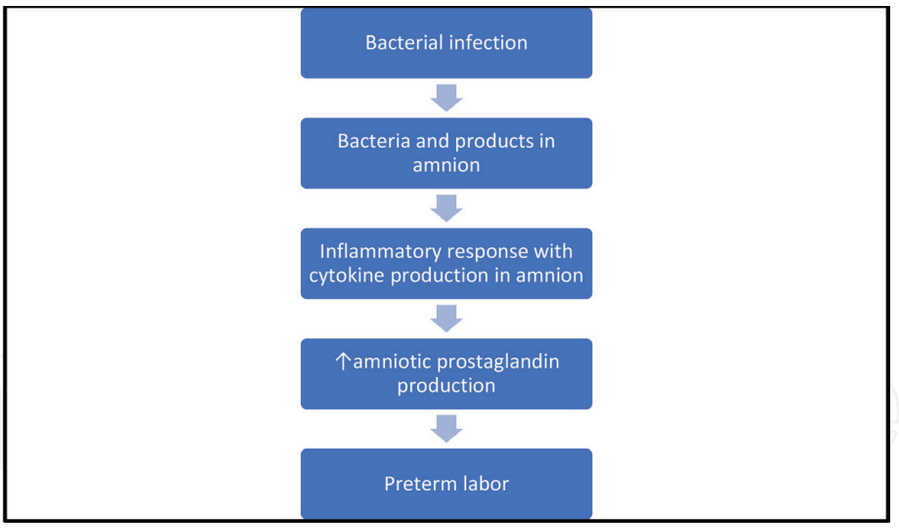

Figure 2.
Schematic representation of role of bacterial infection in preterm labor.

response is also activated, during a normal pregnancy. During pregnancy there is an increase in expression of activation markers on monocytes and granulocytes, differences in monocyte cytokine production, and increased circulating levels of pro-inflammatory cytokines and inflammatory markers, such as C-reactive protein.

Periodontitis sites and subjects harbor specific microorganisms or groups of microorganisms. *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola* are observed more frequently and/or in higher levels and proportions in periodontitis subjects, and *Actinomyces* genus are observed with periodontal health [34, 35]. *Fusobacterium nucleatum*, a bacterium, has been linked with adverse pregnancy outcomes. *F. nucleatum* is associated with periodontal infections and not observed during genital or uterine infections. The infection does not enter the womb through the genital tract; rather it enters the mother's bloodstream making its way down from the oral cavity. The liver produces C-reactive protein (CRP), an acute-phase reactant in response to the inflammatory cytokine's interleukin IL-6, IL-1, and tumor necrosis factor-alpha. Periodontal diseases are associated with raised level of circulating CRP levels and elevation of pro-inflammatory cytokines and prostaglandin [26, 29]. Adverse pregnancy outcomes have been associated with increase in CRP levels. The CRP levels are raised in elevated immunoglobulin G induced by bacterial species found in destructive periodontal diseases [8].

The absence of the mother's IgG antibody against organisms of the red complex is associated with an increased risk of premature birth of the baby. Mothers without a protective red complex IgG response coupled with a fetal IgM response to orange complex microbes had the highest rate of prematurity. This evidence suggests the concept that prematurity in pregnant women may be due to systemic dissemination of oral organisms that translocate to the fetus in the absence of a protective maternal antibody response and trigger preterm babies. The high prevalence of elevated fetal IgM to *C. rectus* among premature infants raises the possibility that this specific maternal oral pathogen may serve as a primary fetal infectious agent eliciting prematurity.

1.9 Effect of periodontal therapy on pregnancy outcomes

No definitive conclusions can be arrived about periodontal disease (PD) treatment during pregnancy. Attempts to improve oral health in women during

pregnancy have not reduced adverse pregnancy outcome (APO). No reduction in APOs was observed with standard PD therapy during pregnancy in several large clinical randomized controlled trials [36].

The dilemma of performing periodontal treatment during pregnancy to reduce the APOs has not been answered. Periodontal treatment even if undertaken during pregnancy will not be thorough and completely eradicate the disease process, due to fear of bacteraemia which may cause APO. Pre-conception period is most appropriate time for periodontal treatment. Periodontal treatment to create a healthy mouth before conception may reduce the occurrence of APOs. The local and systemic inflammation caused by periodontal pathogens may not be controlled by periodontal treatment during pregnancy. Periodontal treatment before pregnancy (for nulliparous women) or in the period between pregnancies (for multiparous women) may reduce APOs [37].

There was a deep-seated bias in the medical/dental community against nonsurgical periodontal interventions during pregnancy [38]. After long-term studies and analysis, the medical and dental fraternity is in a general agreement that pregnant patients can safely undergo dental cleaning [39]. Interventions to reduce the morbidity and mortality associated with preterm birth can be classified as primary, secondary, and tertiary. All interventions examined by existing studies on the effects of periodontal therapy on pregnancy outcomes can be classified as secondary interventions [38]. It has been known for many years that nonsurgical periodontal therapy is effective in reducing the increased amount of periodontal inflammation associated with pregnancy [33, 40, 41]. Data clearly show that this therapy is safe and does not trigger an increase in adverse pregnancy outcomes. It has not been shown that routine nonsurgical periodontal therapy decreases the incidence of these outcomes. In general, women assigned to the periodontal treatment groups showed statistically significant improvements in their periodontal assessments.

2. Conclusion

Pregnancy in woman brings about profound changes in innate and adaptive immunity of the mother and fetus; these changes play a major role altering the clinical course of a number of infectious diseases, including periodontal diseases. The severity of gingival and periodontal diseases increases during the course of normal pregnancy. Gingival inflammation and tissue response toward the microbial plaque is exaggerated during pregnancy due to the hormonal factors and is accepted by the scientific community. Pregnant women with previously existing periodontal disease will have increased destruction of the periodontal structures. The gingival changes observed during pregnancy return to normal limits immediately after delivery of the baby, if the local irritants are removed; this phenomenon is called as "immune reconstitution syndrome." Gestational diabetes which occurs in certain pregnant women can increase the risk for periodontal diseases, and it should be well controlled by treating gynecologist. Preeclampsia if not detected and treated can cause serious condition eclampsia leading to convulsions, coma, and death of the mother.

Large numbers of epidemiological studies suggest that periodontal infection is a modest risk factor for several adverse pregnancy outcomes. The studies conducted to link between periodontal diseases and adverse pregnancy outcomes have had contradictory results, as they were carried out in different sets of populations or with different study designs. It is better to consider periodontal disease as a risk factor for adverse pregnancy outcome, as thorough oral health maintenance helps the pregnant women attain a better oral health which is part of general health.

Controversies in the academic community regarding the treatment of periodontal problems have been eradicated. It is well accepted that oral prophylaxis and nonsurgical periodontal therapy can be rendered to pregnant women in the second trimester.

Periodontal diseases go unnoticed in the initial stages of disease process. The inflammatory load of periodontal disease can enter the systemic circulation and can be a risk factor for several host tissues and physiological activities. There is definite link between periodontal diseases and adverse pregnancy outcomes, through direct or indirect mechanisms. The direct action of perio-pathogenic organisms on amnion and indirect action through systemic circulation by production of inflammatory mediators can be risk for adverse pregnancy outcomes.

Conflict of interest

I have no "conflict of interest" in publishing this chapter.

Notes/thanks/other declarations

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