



Major clinical and systemic impacts of periodontal diseases in pregnant: a systematic review

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Abstract

Introduction: Periodontal disease (PD) represents several pathological changes that occur in the periodontium, that is, the tissues surrounding the teeth. These include the periodontal ligament, cementum, alveolar bone, and gingiva. During pregnancy, women enter a period in which oral health should be a priority. During this period, estrogen and progesterone levels, their sex hormones, potentially increase over time and are detrimental to tissue metabolism. **Objective:** It was to conduct a systematic review of the risk of periodontal disease in pregnant women. **Methods:** The research was conducted from June to July 2026 in the Scopus, Embase, PubMed, Science Direct, and Scielo databases, using older gold-standard scientific articles up to 2025. Study quality was based on the GRADE instrument, and risk of bias was assessed using the Cochrane tool. **Results and Conclusion:** A total of 145 studies were found and submitted to eligibility analysis. The final sample contained 17 eligible studies, which were described in the systematic review. Most studies presented homogeneous results, with $X^2=70.5\% >50\%$. This study highlighted that periodontal disease in pregnant women may be one of the determinants of low birth weight. Studies were conducted to verify the existence of an association between maternal periodontal disease and low birth weight. The gestational period is when the occurrence of PD is more likely due to its hormonal and physiological changes. Pregnant women should be vigilant and take appropriate care, have prenatal care

with their dentist, and in addition to oral care, they should be aware of the risks of low birth weight and preterm birth. Infections in the genitourinary tract, such as PD, may be associated with the occurrence of low birth weight newborns through the same mechanisms as other maternal infections and preterm birth. Uterine contraction and cervical dilation, which trigger premature birth, can be caused by inflammatory stimuli, which can induce hyperirritability of the uterine smooth muscle. Infection and the resulting inflammatory process can damage the placenta, thus restricting fetal development. Throughout life, women experience various phases of hormonal fluctuations that, in addition to interfering with the reproductive system, influence other organs and systems, such as the periodontium, especially in the presence of preexisting plaque-induced gingival inflammation. During pregnancy, women are exposed to several significant hormonal changes, as the placenta produces high amounts of estrogen and progesterone, which, in turn, affect oral tissues. It was concluded that Black women over 40 are more likely to have PD, presenting a higher risk of poor fetal development.

Keywords: Periodontal disease. Inflammatory periodontal diseases. Pregnant women.

Introduction

Periodontal disease (PD) is a range of pathological changes affecting the periodontium—the

tissues surrounding the teeth—specifically the periodontal ligament, cementum, alveolar bone, and gingiva. Bacterial plaque is the primary determinant of the imbalance between defensive and aggressive forces acting upon the tooth's protective and supporting tissues, triggering a host response. Sex hormones act as influencing factors— affecting periodontal immune responses—due to changes in subgingival plaque. The disease manifests clinically when the patient already exhibits uncontrolled plaque accumulation [1-3].

Various factors determine periodontal disease in Brazil. In contrast, socioeconomic conditions are not determined by the gestational period itself; cultural factors, smoking, and a lack of knowledge regarding proper care can exacerbate the condition [1,2]. Evidence suggests an association between maternal PD and low birth weight, an effect that is intensified by low maternal education levels. Pregnancy is a time when oral health should be a priority. During this period, levels of the sex hormones estrogen and progesterone rise significantly, potentially impairing tissue metabolism. The prevalence of these change ranges from 35% to 100%, with severity increasing progressively up to the 36th week of gestation. The presence of bacteria is a key factor aggravating both the incidence of periodontal disease during this period and the severity of the disease once established [2,3].

Pregnant women require special attention to oral health for two main reasons: proper nutrition is essential, making tooth pain or mobility unacceptable, and periodontal infections can spread through the bloodstream, stimulating the production of inflammatory cytokines [4-8]. Pregnancy is a complex human experience involving both a social dimension— influenced by various external factors—and a biological dimension, which requires a balance between the physiological conditions necessary for fetal development and the maternal immune system [8,9].

In this context, oral hygiene routines—including plaque control and intensified oral care—can be performed during any trimester of pregnancy, given that pregnancy gingivitis is the most common condition; it is characterized by gums becoming highly vascularized, edematous, and sensitive. The gingival margin and surrounding tooth structure become erythematous and bleed frequently during brushing and chewing—a condition affecting between 50% and 100% of pregnant women [9]. Given this background, the present study conducted a systematic review regarding the risk of periodontal diseases in pregnant women.

Methods

Study Design

This study followed an international systematic review model, adhering to PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. Available at: <http://www.prisma-statement.org/?AspxAutoDetectCookieSupport=1>. Accessed on: July 22, 2026. It also adhered to the AMSTAR 2 (Assessing the Methodological Quality of Systematic Reviews) standards for methodological quality. Available at: <https://amstar.ca/>. Accessed on: July 22, 2026.

Search Strategy and Sources

The literature search process was conducted between June and July 2026 using Web of Science, Scopus, Embase, PubMed, LILACS, EBSCO, SciELO, and Google Scholar, covering scientific articles ranging from various past periods up to the present. The Health Sciences Descriptors (DeCS/MeSH) used were: "Periodontal disease," "Inflammatory periodontal diseases," and "Pregnant women," employing the Boolean operator "AND" between MeSH terms and "OR" between the search terms.

Study Quality and Risk of Bias

Quality was classified as high, moderate, low, or very low based on risk of bias, clarity of comparisons, precision, and consistency of analyses. Systematic reviews or meta-analyses of randomized clinical trials ranked highest, followed by randomized clinical trials. Low-quality evidence was attributed to case reports, editorials, and brief communications, in accordance with the GRADE instrument. Risk of bias was assessed using the Cochrane tool via funnel plot analysis (sample size versus effect size), employing Cohen's d test.

Results

Summary of Findings

A total of 145 articles were identified. Duplicate articles were initially excluded. Following this process, abstracts were evaluated, and further exclusions were made to remove articles unrelated to the study topic, resulting in 94 articles. A total of 52 articles were evaluated in full, and 17 were included and analyzed in this systematic review. Based on the Cochrane risk-of-bias tool, the overall assessment identified 16 studies with a high risk of bias and 26 studies that did not meet GRADE criteria (Figure 1). According to the GRADE instrument, the majority of studies showed homogeneity in their results, with $X^2 = 70.5\%$ ($>50\%$).

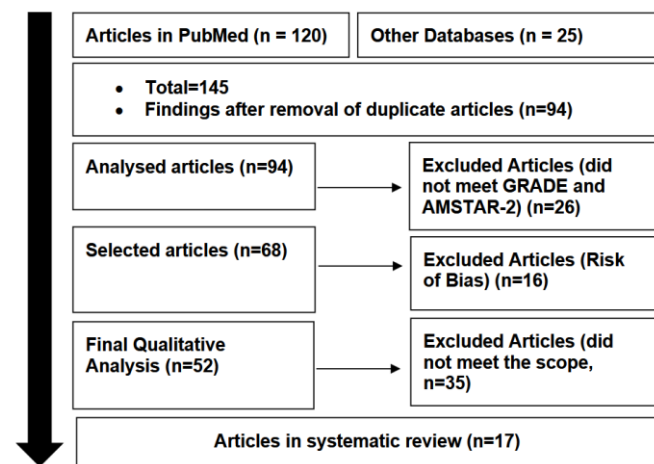


Figure 1. Flowchart showing the article selection process. Source: Own authorship.

Figure 2 presents the results regarding risk of bias across the studies using a funnel plot, showing the calculated effect size (magnitude of difference) based on Cohen's d. Precision (sample size) was determined indirectly as the inverse of the standard error ($1/\text{Standard Error}$). The plot exhibited a symmetrical pattern, suggesting no significant risk of bias among either studies with small sample sizes (lower precision)—shown at the bottom of the plot—or studies with large sample sizes—shown at the top.

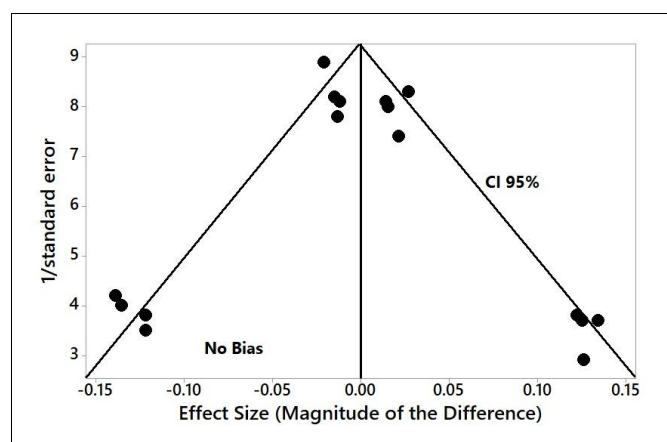


Figure 2. The symmetrical funnel plot suggests no risk of bias among studies with small sample sizes, shown at the bottom of the plot. Studies with high confidence and high recommendation levels are shown at the top of the plot (n=17 studies). Source: Own authorship.

Major Clinical Findings and Development

Preeclampsia is a blood pressure disorder characterized by the onset of proteinuria from the 20th week of gestation onwards. It is a leading cause of maternal and perinatal morbidity and mortality, affecting 5% to 8% of pregnant women in Brazil. Factors associated with preeclampsia include its etiology, inflammatory aspects, and its correlation with endothelial dysfunction. During the pseudovasculogenesis that occurs in pregnancy, processes such as trophoblast

migration and spiral arteriole remodeling—phenomena intended to establish a local low-resistance arteriolar system—appear to be impaired in women with preeclampsia. This local defect leads to the second phase of the disease, in which systemic maternal responses result in hypertension, proteinuria, and multi-organ alterations, primarily driven by endothelial dysfunction. The potential role of inflammatory cytokines in endothelial dysfunction has prompted researchers to investigate polymorphisms in the genes encoding them [6].

Although preeclampsia remains a condition that is not fully understood or widely discussed in society, several risk factors have been identified, such as primiparity, obesity, and renal disorders. However, recent years have seen evidence of the influence of infections on preeclampsia, including a possible link to periodontal disease. Pregnant women with preeclampsia exhibit elevated plasma levels of certain cytokines. These increases are thought to stem from a generalized inflammatory state, although the specific mechanisms underlying these changes in the pathogenesis of preeclampsia remain unclear [7].

Periodontal disease is a general term encompassing a range of pathological conditions affecting the periodontium. The periodontium consists of the tissues that line and surround the tooth—specifically the gingiva, alveolar bone, cementum, and periodontal ligament (fibers connecting the gingiva to the alveolar bone and cementum). There are numerous periodontal diseases, which can be categorized into two main groups: periodontitis and gingivitis. In periodontitis, changes occur in both soft and hard tissues (bone, periodontal ligament, and cementum). In gingivitis, only the soft tissues of the gingiva are affected [1-3].

When microorganisms adhere to the tooth and gingiva, the primary change that occurs is gingival inflammation. Under these conditions, the space between the gingiva and the tooth (the gingival sulcus) widens, and the sulcus (normally 2–3 mm deep) transforms into a pathological pocket. When the microbial flora associated with gingivitis is eliminated, the condition resolves, and the gingiva returns to normal [9,10].

If gingivitis remains untreated, the pathological process tends to extend to the hard tissues; these undergo gradual or abrupt alteration, resulting in periodontitis. Alveolar bone resorption and the loss of the fibers connecting the bone to the tooth (the periodontal ligament) are the primary changes associated with periodontitis. This bone resorption is referred to as attachment loss. Periodontal diseases are primarily classified into two groups: gingivitis and

periodontitis [11].

Periodontitis is a chronic, highly prevalent infection caused by Gram-negative bacteria; it is considered the second leading cause of dental pathology in the global population. It is defined as a “multifactorial chronic inflammatory disease associated with biofilm and characterized by the progressive impact on the tooth attachment apparatus.” As previously mentioned, the primary alteration in periodontitis is the resorption of alveolar bone and the loss of the fibers connecting the bone to the tooth (periodontal ligament). However, there are various types of periodontitis involving attachment loss, as shown below [1,2,11]:

- ❖ Localized Chronic Periodontitis: Generally slow-progressing, associated with bacterial plaque, and occurring at localized sites.
- ❖ Generalized Chronic Periodontitis: Slow-progressing, associated with bacterial plaque, and generally affecting the oral cavity.
- ❖ Localized Aggressive Periodontitis: Rapid progression and not necessarily associated with significant bacterial plaque accumulation.
- ❖ Generalized Aggressive Periodontitis: Slightly more rapid progression and not necessarily associated with significant bacterial plaque accumulation. Attachment loss detected at two or more non-adjacent interproximal sites; or attachment loss of 3 mm or more on the buccal or lingual/palatal surfaces of at least 2 teeth, not resulting from traumatic gingival recession; dental caries extending to the cervical region of the tooth; attachment loss on the distal surface of a second molar associated with third molar malposition or extraction; endo-periodontal lesion draining through the marginal periodontium; or occurrence of a vertical root fracture.

Periodontitis Stage

- ❖ Stage I: Initial Periodontitis. No tooth loss due to periodontitis.
- ❖ Stage II: Moderate Periodontitis. No tooth loss due to periodontitis.
- ❖ Stage III: Severe periodontitis with potential for additional tooth loss. Loss of ≤ 4 teeth due to periodontitis.
- ❖ Stage IV: Advanced periodontitis with extensive tooth loss and potential for tooth loss. Loss of ≥ 5 teeth due to periodontitis [12].

Periodontitis Grade

- ❖ Grade A: Heavy biofilm deposits with low levels of destruction. Bone loss % < 0.25 .
- ❖ Grade B: Destruction proportional to biofilm deposits. Bone loss % from 0.25 to 1.0.

- ❖ Grade C: Destruction exceeds expectations based on biofilm deposits; specific clinical patterns associated with periods of rapid progression and/or early-onset disease (lack of expected response to standard bacterial control therapies); bone loss > 1.0 .

Gingivitis is a periodontal disease characterized by inflammation of the gingiva, causing edema, redness (enantherma), bleeding, exudate, alterations in normal contours, and occasionally discomfort [12-14]. The microbiota consists predominantly of Gram-positive, aerobic, saccharolytic, and non-motile bacteria. Diagnosis is based on clinical inspection [1,3,4].

Plaque-associated gingivitis is caused by the accumulation of bacterial plaque or biofilm on the tooth surface, leading to gingival inflammation, followed by bleeding, swelling, and discomfort. Mineralized bacterial plaque is a concretion of bacteria—associated with almost all forms of gingivitis—comprising food debris, saliva, and mucus mixed with calcium and phosphate salts. Poor oral hygiene allows bacterial plaque to accumulate between the gingiva and the teeth; gingivitis occurs only in areas where both gingiva and teeth are present—without the presence of both, the condition is not classified as gingivitis. The irritation caused by bacterial plaque deepens the normal gingival sulcus between the gum and the tooth, creating pathological gingival pockets [1,4].

Biofilm-induced gingivitis can be classified according to its location and extent (localized or generalized) and its severity (mild, moderate, or severe). Percentages: Mild: less than 10% of sites; Moderate: 10% to 30% of sites; Severe: more than 30% of sites; Grade: Grade 1: 20% of sites; Grade 2: 40% of sites; Grade 3: 60% of sites [9]. Non-Plaque-Induced Gingivitis.

Gingivitis not associated with bacterial plaque is an inflammation of the gums—causing swelling, redness, and bleeding—triggered by factors other than biofilm. This led to the creation of a more comprehensive classification system that includes additional diseases and conditions. Regarding the epidemiology of preterm birth, a key point is that while some pregnant women are exposed to an infectious stimulus, that stimulus alone is not always sufficient to activate the inflammatory cascade and trigger preterm delivery. The first evidence supporting this concept came from animal studies. Endotoxins restrict fetal growth. Another study induced localized infections by subcutaneously injecting *Porphyromonas gingivalis*, which resulted in a reduction of fetal weight by more than 25%. Complications such as preterm birth, elevated intrauterine levels of PGE2 and TNF- α , increased amniotic fluid PGE2, and

miscarriage were also monitored and observed [15].

Gestational Changes and Their Potential Impact on Periodontics

Pregnancy is a process involving complex physical and psychological changes that profoundly affect healthy women. In the past, pregnancy was considered a barrier to dental treatment due to physiological changes that altered the patient's medical status. Physiological changes occurring during pregnancy include weight gain, supine hypotension, urinary frequency, respiratory function restriction, potential for hypoglycemia, and decreased heart rate. Syncope and nausea are also common during pregnancy [16].

The first trimester is the most critical period for the embryo, as various organs develop during this time, making it more vulnerable to teratogenic insults and miscarriage. Spontaneous miscarriages are more likely to occur during this period; therefore, whenever possible, dental procedures—especially invasive ones—should be avoided [17]. Furthermore, during pregnancy, elevated levels of sex steroid hormones are maintained—resembling the luteal phase—facilitating embryo implantation and sustaining the pregnancy until birth. A pregnant woman near or at term produces large quantities of estradiol (20 mg/day), estriol (80 mg/day), and progesterone (300 mg/day). Gingival inflammation initiated by bacterial plaque and exacerbated by hormonal changes during the second or third trimester of pregnancy is known as gestational gingivitis [17].

PD is a common term for chronic inflammatory conditions of bacterial etiology that cause gingival inflammation—specifically gingivitis—which may or may not progress over time to inflammation of the tooth-supporting tissues, known as periodontitis. This progression does not occur linearly but rather through episodic flare-ups [1,3,4]. Based on evidence regarding the hematogenous spread of cytokines and bacteria originating from periodontal infections, it is believed that an association exists between this condition and an increased risk of certain systemic disorders, particularly cardiovascular diseases and diabetes mellitus [8]. Cytokine levels may be elevated in the blood of patients with periodontal disease. Following periodontal treatment, increases in serum levels of IL-6 and TNF- α are observed. If cytokines remain confined to the periodontal pocket without entering the bloodstream, there is no cause for concern regarding a link between periodontal disease and systemic disorders. This process can occur in two ways: through the circulatory action of bacteria inducing systemic cytokine production, or through the dissemination of cytokines during the scaling of infected tissue [2,5,6].

Conclusion

It was concluded that the stage of pregnancy during which periodontal disease is present is a potential high-risk factor for low birth weight in the fetus. Dental care is essential for optimal oral health and extends through the period of pregnancy. Certain oral changes may be observed as early as the first trimester and can progress into the third, a period coinciding with a significant surge in the expectant mother hormone levels. The dentist should be part of the prenatal care team. During early appointments, the obstetrician should refer the pregnant woman to the team's designated oral health professional for a dental assessment and ongoing care. During this period, the dentist carries out preventive, educational, and interventional measures—determined after evaluating each case in collaboration with the obstetrician—while assessing the patient's specific risk factors, such as the stage of pregnancy and systemic health conditions. It is also worth noting that this approach impacts public finances, as it aims to reduce potential complications during and after childbirth, thereby decreasing the rate of neonatal hospitalizations.

CRedit

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Conflict of Interest

The authors declare no conflict of interest.

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Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

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Referências

- García-Martos JM, Valverde-Bolívar FJ, Campillo-López MT, Delgado-Rodríguez M. Association between periodontal disease and gestational diabetes: Systematic review and meta-analysis. *Prim Care Diabetes*. 2025 Feb;19(1):1-6. doi: 10.1016/j.pcd.2024.11.003.
- Ibraheem SA, Afolabi OA, Oveh R, Chukwuemeka A, Dabar A, Nwaokorie FO, Salako AO, Gbaja-Biamila TA, Owotade FJ, Akinsolu FT, Eleje GU, Ezechi OC, Foláyan MO. Periodontal diseases among pregnant adolescents and young women in Nigeria: a scoping review. *BMC Oral Health*. 2025 Jun 6;25(1):936. doi: 10.1186/s12903-025-06004-3.
- Montoya-Carralero JM, Ávila-Villasmil R, Sánchez-Pérez A, Jornet-García A, Terrer-Alonso E, Moya-Villaescusa MJ. Relationship between periodontal disease and preterm birth. A systematic review and meta-analysis. *Med Oral Patol Oral Cir Bucal*. 2024 Nov 1;29(6):e857-e865. doi: 10.4317/medoral.26830.
- Duque Duque A, Chaparro Padilla A, Almeida ML, Marín Jaramillo RA, Romanelli HJ, Lafaurie Villamil GI. Strategies for the prevention of periodontal disease and its impact on general health: Latin America and the Caribbean Consensus 2024. *Braz Oral Res*. 2024 Oct;38(suppl 1):e120. doi: 10.1590/1807-3107bor-2024.vol38.0120.
- Ashokan DB, Shekar MK, Dharani K, Mani E, Pampani P, Ravichandran B. Clinical evaluation of photobiomodulation therapy and local 1.2% atorvastatin gel as an adjunct to subgingival instrumentation in patients with stage II periodontitis: a randomized controlled trial. *Quintessence Int*. 2025 Jul 21;56(7):514-527. doi: 10.3290/j.qi.b6218534.
- Gadzhula NG, Cherepakha OL, Lezhnova OV. Efficiency of treatment of inflammatory periodontal diseases in pregnant women. *Wiad Lek*. 2021;74(5):1065-1068. PMID: 34090265.
- Cruz, Simone Seixas da et al. Doença periodontal materna como fator associado ao baixo peso ao nascer. *Revista de Saúde Pública* [online]. 2005, v. 39, n. 5 [Acessado 16 Dezembro 2021], pp. 782-787. Disponível em: <<https://doi.org/10.1590/S0034-89102005000500013>>. Epub 03 Feb 2006. ISSN 1518-8787. <https://doi.org/10.1590/S0034-89102005000500013>.
- Geisinger ML, Geurs NC, Bain JL, Kaur M, Vassilopoulos PJ, Cliver SP, Hauth JC, Reddy MS. Oral health education and therapy reduces gingivitis during pregnancy. *J Clin Periodontol*. 2014 Feb;41(2):141-8. doi: 10.1111/jcpe.12188. Epub 2013 Nov 19. PMID: 24164645.
- Ostrovskaya LI. Modern approaches to prevention of periodontal diseases in pregnancy: a review. *Wiad Lek*. 2019;72(1):89-94. PMID: 30796869.
- Petit C, Benezech J, Davideau JL, Hamann V, Tuzin N, Huck O. Consideration of Oral Health and Periodontal Diseases During Pregnancy: Knowledge and Behaviour Among French Pregnant Women. *Oral Health Prev Dent*. 2021;19(1):33-42. doi: 10.3290/j.ohpd.b875513. PMID: 33491376.
- Iheozor-Ejiofor Z, Middleton P, Esposito M, Glenny AM. Treating periodontal disease for preventing adverse birth outcomes in pregnant women. *Cochrane Database Syst Rev*. 2017 Jun 12;6(6):CD005297. doi: 10.1002/14651858.CD005297.pub3. PMID: 28605006; PMCID: PMC6481493.
- Grinin VM, Makeeva IM, Gosteva NS, Erkanyan IM, Solovyova OA. Struktura vospalitel'nykh zabolevanii parodonta i dinamika parodontal'nogo statusa beremennykh na protiazhenii gestatsionnogo perioda [The structure of inflammatory periodontal diseases and the dynamics of the periodontal status in pregnant women during the gestational period]. *Stomatologiya (Mosk)*. 2020;99(1):12-17. Russian. doi: 10.17116/stomat20209901112. PMID: 32125296.
- Hartnett E, Haber J, Krainovich-Miller B, Bella A, Vasilyeva A, Lange Kessler J. Oral Health in Pregnancy. *J Obstet Gynecol Neonatal Nurs*. 2016 Jul-Aug;45(4):565-73. doi: 10.1016/j.jogn.2016.04.005. Epub 2016 Jun 6. PMID: 27281467.
- Steffens JP, Marcantonio RAC. Classificação das Doenças e Condições Periodontais e Peri-implantares 2018: guia Prático e Pontos-Chave. *Revista de Odontologia da UNESP* [online]. 2018,

- v. 47, n. 4 [Acessado 17 Dezembro 2021] , pp. 189-197. Disponível em: <<https://doi.org/10.1590/1807-2577.04704>>. Epub Jul-Aug 2018. ISSN 1807-2577. <https://doi.org/10.1590/1807-2577.04704>.
15. Escobar-Arregoces F, Latorre-Uriza C, Velosa-Porras J, Roa-Molina N, Ruiz AJ, Silva J, Arias E, Echeverri J. Inflammatory response in pregnant women with high risk of preterm delivery and its relationship with periodontal disease: a pilot study. *Acta Odontol Latinoam*. 2018 Jun;31(1):53-57. English. PMID: 30056467.
16. Opacic J, Maldonado A, Ramseier CA, Laugisch O. Einfluss der Parodontitis auf Schwangerschaft und Geburt [Influence of periodontitis on pregnancy and childbirth]. *Swiss Dent J*. 2019 Jul 22;129(7-8):581-589. German. PMID: 31271020.
17. Bobetsis YA, Graziani F, Gürsoy M, Madianos PN. Periodontal disease and adverse pregnancy outcomes. *Periodontol* 2000. 2020 Jun;83(1):154-174. doi: 10.1111/prd.12294.