

The effect of hormonal changes observed in female patients on periodontium

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ABSTRACT

In women, physiological processes such as puberty, the menstrual cycle, pregnancy, oral contraceptive use, and menopause cause significant fluctuations in estrogen, progesterone, and androgen levels, increasing the inflammatory response in gum tissues and affecting the course of periodontal disease. Numerous studies in the literature have shown that hormonal changes directly affect periodontal cells, modulate inflammatory cytokine levels, and shape tissue destruction and healing processes. Furthermore, it has been emphasized that during these hormonal periods, there is an increase in the frequency of conditions such as gingivitis, periodontitis, and pyogenic granuloma seen in women. In periodontology practice, it is clear that variable hormonal conditions must be taken into account in treatment and preventive approaches for female patients. This study aimed to evaluate the effects of physiological hormonal processes in women on periodontal tissues.

Keywords: Hormonal changes, periodontal diseases, women, estrogen, pregnancy, menopause

PERIODONTAL DISEASE

Periodontal diseases are among the most common health problems in society. The most well-known of these diseases are gingivitis and periodontitis. However, both diseases have different subtypes, which are classified according to factors such as age of onset, clinical symptoms, rate of progression, types of pathogenic microorganisms, and systemic effects. Gingivitis is an inflammatory condition that affects only the gums, without accompanying attachment or alveolar bone loss. Early symptoms of this disease include increased gingival crevicular fluid and bleeding during probing. In some cases, gingivitis can progress to periodontitis. Periodontitis is a disease in which attachment and bone loss occur due to inflammation of the tissues supporting the teeth. Although it is usually chronic, factors such as diabetes, smoking, and stress can affect the host's response to bacterial plaque and increase the rate of disease progression.¹

Periodontal Disease Etiology

The fundamental cause of periodontal disease is the disruption of subgingival microbiota balance over time due to an increase in disease-associated bacteria and a decrease in healthy bacteria. This change leads to a loss of homeostasis between the host and the microbiota.² The development of the disease is a gradual process, beginning with the adhesion and proliferation of both gram-negative and gram-positive bacteria on the tooth surface.³ Over time, these bacteria colonize the subgingival region and alter the environmental conditions. The resulting environment becomes favorable for the growth and colonization of anaerobic gram-negative bacteria, particularly the orange and red complex groups.⁴

The orange complex bacteria include *Prevotella intermedia* (*P. intermedia*), *Parvimonas micra*, and *Fusobacterium nucleatum* (*F. nucleatum*), while the red complex consists of *Porphyromonas gingivalis* (*P. gingivalis*), *Tannerella forsythia* (*T. forsythia*), and *Treponema denticola* (*T. denticola*) species.⁴ These bacteria are highly pathogenic and have the ability to secrete bacterial collagenases and other proteases. The release of these enzymes triggers a proinflammatory response in the host, causing damage to periodontal tissues.⁵

Etiological Factors Causing Periodontal Disease

The etiological factors causing periodontal disease are classified below:⁶

- Microbial dental plaque
- Local predisposing factors
- Systemic predisposing factors

Local predisposing factors:

- Dental calculus (Tartar)
- Materia alba
- Food debris
- Food impaction
- Dental anatomy
- Faulty restorations
- Malocclusion
- Mouth breathing
- Occlusal trauma

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Systemic predisposing factors:

- Hormonal imbalance
- Nutritional deficiency
- Blood disorders
- Genetic
- Immunological factors
- Metal intoxication
- Immune suppression
- Psychological factors

Periodontal Disease Pathogenesis

Recent studies have shown that bacterial plaque alone is not sufficient for the onset and progression of periodontal disease.⁷ The most important factor in the destruction of hard and soft tissues in periodontal disease is the activation of the host immune-inflammatory response against bacterial plaque.⁸ While gingivitis and mild periodontitis are common in the general population, advanced periodontitis is a rarer condition.⁹

Some individuals have been found to be more susceptible to periodontal disease, and this increased susceptibility has been linked to the host immune-inflammatory response that develops in periodontal tissues following the formation of bacterial plaque.¹⁰ The microbial threat posed by subgingival plaque triggers the host immune-inflammatory response by triggering the excessive production of inflammatory cytokines [interleukins, tumor necrosis factor- α (TNF- α)], prostanoids (prostaglandin E₂), enzymes (matrix metalloproteinases-MMPs), and reactive oxygen species (ROS), thereby triggering the host immune-inflammatory response.¹¹ These inflammatory mediators play a significant role in the destruction of periodontal hard and soft tissues.¹²

Pathogenesis stages in periodontal disease:⁶

- Colonization
- Invasion
- Tissue destruction
- Healing and fibrosis

Colonization: Plaque accumulates on the tooth surface and is soon colonized by microorganisms. Plaque grows and develops through bacterial proliferation and the apical migration of motile bacteria.⁶

Invasion: Microorganisms and their products invade the depths of the connective tissue, even reaching the alveolar bone surface.⁶

Tissue destruction: Microorganisms and their products are observed when they penetrate tissues, either directly or through host-dependent effects.⁶

Healing and fibrosis: Antigens from plaque bacteria stimulate T and B lymphocytes, causing them to multiply. Thus, the humoral and cellular immune response begins.⁶

DISTRIBUTION OF PERIODONTAL DISEASE BY GENDER

Many studies examining the relationship between periodontal status and gender have indicated that the prevalence and severity of periodontal disease are higher in men.¹³

This difference between genders has been attributed more to oral hygiene habits than to genetic factors.¹⁴

A study conducted at the Faculty of Dentistry, Van Yüzüncü Yıl University, found that 53.5% of the 1,758 individuals who visited the clinic were women (941) and 46.5% were men (817).

The results of this study show that men are at higher risk for periodontal disease.¹³

Similarly, a study conducted at Atatürk University indicated that men are more prone to periodontal diseases and that this is largely due to smoking, low oral hygiene awareness, and hormonal differences.¹⁵

There is strong evidence that men have a higher prevalence and severity of destructive periodontal disease than women with similar gum disease.¹⁵

One study found that gingivitis is more common in women than in men, while periodontitis is more common in men than in women.¹⁶ This result may be related to higher smoking rates and poor oral hygiene (high PI, Gi, and SK values) among men.¹⁷ Similarly, some studies emphasize that women may be more susceptible to periodontal diseases during certain periods due to hormonal changes (such as pregnancy and menopause).¹⁸

PREVALENCE OF PERIODONTAL DISEASE IN WOMEN

The prevalence of periodontal disease in women may be higher than in men due to factors such as hormonal changes, pregnancy, menopause, and birth control pill use. Hormonal fluctuations, especially during adolescence, pregnancy, and menopause, cause the gum tissue to become more sensitive to inflammation. Therefore, it has been stated that women's risk of developing periodontal disease increases significantly during these specific periods.¹⁹

A study conducted among patients attending the Necmettin Erbakan University Faculty of Dentistry found that the prevalence of periodontal disease was higher in women than in men. The study examined a total of 799 patients, including 510 women and 289 men, and found that 56% of women had signs of periodontal disease.²⁰

Studies provide significant evidence that hormonal changes in women increase the risk of periodontal disease. The pregnancy period, in particular, is a time when the incidence of gum inflammation (gingivitis) increases. It has been emphasized that the decrease in estrogen levels after menopause increases the risk of bone loss and creates conditions conducive to the development of periodontal disease.²¹

EFFECTS OF HORMONAL CHANGES IN WOMEN ON PERIODONTAL TISSUES

The periodontium is a tissue complex consisting of the gingiva surrounding and supporting the tooth, the periodontal ligament, cementum, and alveolar bone. It is also responsible for the nutrition of the cells surrounding the tooth and the transmission of nerve impulses.²² The homeostasis of the periodontium involves a multifactorial relationship in which the endocrine system plays an important role. Hormones play an important role in the human body, and women in particular undergo many hormonal changes throughout their lives. Biological changes such as puberty, menstruation,



pregnancy, menopause, and oral contraceptive use have suggested a possible link between sex steroid hormones and periodontal health.²³ Clinical observations have revealed that periodontal tissues may be target tissues for androgens, estrogens, and progestins due to the combination of hormone localization, receptors, and metabolism. Although the etiology of periodontal endocrinopathies varies, it has been suggested that periodontal pathologies may be related to the effect and interaction of steroid sex hormones on cells found in the periodontium.²⁴

The currently accepted classification of periodontal disease acknowledges that endogenous steroid sex hormones have an effect on the periodontium.

These effects generally manifest as gingival symptoms.²⁵

Studies suggest that changes in the periodontium may be related to fluctuations in hormones.²⁶

Hormones

Sex steroid hormones: Estrogens, androgens, and progestins are known as sex steroid hormones. These hormones are lipid molecules with a common cyclopentanoperhydrophenanthrene skeleton. They are generally derived from 27-carbon cholesterol through sequential removal of carbon atoms and hydroxylation reactions, and are secreted by the adrenal cortex and gonadal cells. Steroid synthesis is the process by which the cholesterol molecule is irreversibly converted through sequential reactions into molecules belonging to the pregnane, androstan, and estrone families. Sex steroid hormones are synthesized in specific tissues and released directly into the bloodstream to reach their site of action. The tissues and cells they affect are called “target tissues”; the gingiva is one such target tissue. In target tissues, hormones can exert effects such as regulating the rate of metabolic pathways, stimulating or suppressing the synthesis and release of other hormones, and altering the production of non-hormonal compounds.²³

Androgens: All natural androgens are based on a 19-carbon tetracyclic hydrocarbon skeleton known as androstan.²³ Androgens exert a number of important effects in the body; these include roles in spermatogenesis and the development of secondary sexual characteristics in male puberty.²⁷ Androgens are essentially found in two main types: gonadal androgens, which include dihydrotestosterone (DHT), and adrenal androgens, known as dehydroepiandrosterone (DHEA). Adrenal androgens can be converted into hormones such as testosterone and estrogen in the circulation. This conversion is the primary source of estrogen in men and postmenopausal women. Testosterone, one of the most potent androgenic hormones, is synthesized in the Leydig cells of the testes, the theca cells of the ovaries, and the adrenal cortex.²⁸

The gingiva is one of the target tissues for androgen hormones. The presence of androgen receptors has been detected in fibroblasts found in periodontal and gingival tissues.

In the presence of testosterone, increased matrix synthesis has been observed in periodontal cells, and it has been reported that the number of testosterone receptors in fibroblasts increases in inflamed and hypertrophic gingiva.²⁹

In their studies, Kasasa and Soory³⁰ reported that androgen metabolism increased in response to interleukin-1 (IL-1) in chronically inflamed gingival and periodontal ligament

tissues. They also noted that DHT concentrations stimulated by insulin-like growth factor increased.

Parkar and colleagues³¹ demonstrated that increased DHT concentrations gradually reduced interleukin-6 (IL-6) production in gingival cells isolated from healthy individuals and gingivitis patients.

Similarly, Gornstein and colleagues³² detected androgen receptors in both gingival and periodontal ligament fibroblasts and observed that androgens reduced IL-6 production in cells possessing these receptors.

IL-6 is a cytokine that plays an important role in tissue destruction during periodontal disease and is secreted by many cells, including oral fibroblasts. These findings suggest that testosterone may have anti-inflammatory effects on the periodontium and that androgens may protect the periodontium through their positive anabolic effect on periodontal cells, their negative effect on the production and presence of inflammation markers, and their ability to inhibit osteoclastic activity.³²

Effects of androgens on periodontal tissues:

- Suppression of prostaglandin secretion
- Proliferation and differentiation of osteoblasts
- Reduction in IL-6 production during inflammation
- Increased matrix synthesis in periodontal ligament fibroblasts and osteoblasts.³²

Estrogen and progesterone: Estrogen and progesterone are the primary hormones that cause physiological changes in women throughout various stages of life, beginning at puberty. Estrogen initiates pubertal development in women, while androgens, together with estrogen, regulate the menstrual cycle and suppress follicle-stimulating hormone release from the anterior lobe of the pituitary gland.³³ Estrogen stimulates the secretion of watery mucus in the cervix by imparting acidophilic properties to cells. This process reduces the viscosity of the mucus while increasing its elasticity. In addition, estrogens increase the synthesis of coagulation factors associated with vitamin K, reducing the production of antithrombin III, which increases the tendency of blood to clot. Estrogen also affects water and salt retention in the kidneys. Estradiol is the most potent estrogen during the premenopausal period and is synthesized in peripheral tissues such as the ovaries, testes, and placenta.³³

Biological functions of estrogen:

- Development of secondary sex characteristics
- Uterine development
- Luteinizing hormone secretion from the anterior pituitary gland
- Peripheral and axial skeletal development³³

Clinical observations have revealed that individuals with low estrogen levels experience more gum inflammation compared to those with normal levels.³⁴ This suggests that estrogen may modulate inflammatory mediators by affecting prostaglandin production. Furthermore, circulating estrogen levels are thought to play a critical role in maintaining periodontal health.



In their studies investigating the relationship between periodontal disease and hormones, Plancak and colleagues³⁵ found that estradiol levels were lower in patients with advanced periodontitis compared to healthy individuals.

Effects of estrogens on periodontal tissues:

- Absence of gingival inflammation despite increased plaque amount³⁴
- Decreased keratinization while increasing epithelial glycogen³⁶
- Increased cellular proliferation in blood vessels³⁷
- Stimulation of PMNL phagocytosis activity³⁸
- Inhibition of PMNL chemotaxis³⁹
- Suppression of leukocyte production from bone marrow⁴⁰
- Inhibition of proinflammatory cytokines⁴¹
- Suppression of T-cell-mediated inflammation⁴⁰
- Proliferation of gingival fibroblasts⁴²
- Stimulation of gingival connective tissue synthesis and maturation⁴²

The biological activity of progesterone is primarily observed during the luteal phase of menstruation and during pregnancy.

Progesterone:

- Is necessary for the continuation of pregnancy
- Reduces the effect of insulin
- Stimulates the hypothalamic respiratory center
- Increases body temperature during ovulation
- Increases sodium excretion by the kidneys.²³

Effects of progesterone on periodontal tissues:

- Increases vascular dilation and permeability²⁸
- Weakens the anti-inflammatory effects of glucocorticoids⁴³
- Increases prostaglandin production⁴⁴
- Increases PMNL and prostaglandin E₂ levels in gingival crevicular fluid⁴⁴
- Inhibition of collagen synthesis in periodontal ligament fibroblasts²⁷
- Suppression of gingival fibroblast proliferation²⁶
- Decreased repair potential due to altered collagen production⁴⁵
- Increased metabolic breakdown of folate, which is necessary for tissue repair⁴⁵

The second phase is the luteal phase. During this phase:

- The developing corpus luteum synthesizes estradiol and progesterone.
- Estrogen rises to 0.2 ng/ml, progesterone to 10 ng/ml. This creates a suitable endometrium for a fertilized egg.
- The corpus luteum ruptures.
- Ovarian hormone levels decrease.
- Menstruation occurs.

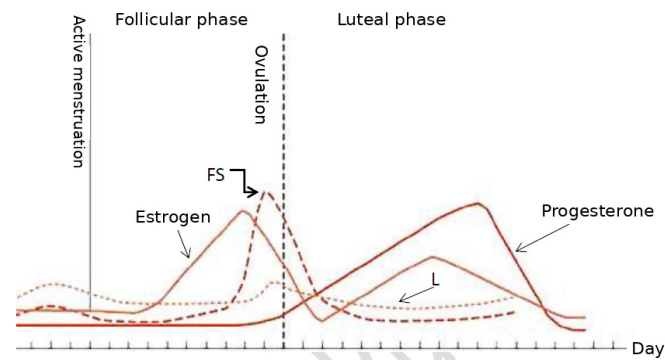


Figure 1. A graph showing hormone levels in women throughout the menstrual cycle.⁴⁶

Various scientific data indicate that ovarian hormones increase inflammation in gingival tissues and intensify the response to local irritants. Increases or imbalances in sex hormones may cause gingival inflammation to worsen. In addition, these hormones may have indirect effects on periodontal tissues by modulating the functions of immune system cells.⁴⁶

Progesterone alters collagen synthesis and structure in gum tissues, increases vascular permeability and folate metabolism, and regulates the immune response. Throughout the menstrual cycle, progesterone levels rise starting in the second week, peak approximately ten days later, and show a marked decline just before menstruation. This hormone promotes the production of prostaglandins, which regulate the body's inflammatory response. PGE₂, in particular, is one of the main mediators released by monocytes and found at higher levels in inflamed gums. Progesterone enhances the chemotaxis of polymorphonuclear cells, while estradiol suppresses this process. On the other hand, testosterone has been reported to have no significant effect on the chemotaxis of polymorphonuclear leukocytes. Physiological, experimental, and clinical studies have revealed significant differences in immune response between women and men.⁴⁶

During menstruation, gingival tissues may become more edematous. In some individuals, erythema observed in the gums prior to menstruation may be a sign of an approaching cycle. In addition, an increase in gingival exudate and, consequently, noticeable changes in tooth mobility may be observed. At the onset of menstruation, osteitis cases that may develop after tooth extraction have been reported to be observed more frequently. Although a slight increase in platelet count and prolonged clotting time is observed during this process, no significant hematological changes have been detected in blood values.⁴⁶

The peak levels of progesterone during the luteal phase may increase the incidence of recurrent aphthous ulcers, herpes labialis, and candidal infections in some women. Furthermore, progesterone's effect on relaxing the esophageal sphincter may increase susceptibility to gastroesophageal reflux disease (GERD). GERD can manifest with symptoms such as heartburn, vomiting, and chest pain, and in advanced cases, it may be associated with conditions such as coughing, hoarseness, sore throat, gingivitis, and asthma.⁴⁶

Approximately 7-10 days before menstruation, progesterone levels reach their peak, which may cause premenstrual syndrome (PMS). It has been reported that levels of certain neurotransmitters such as enkephalin, endorphin, gamma-aminobutyric acid (GABA), and serotonin are lower in



women experiencing PMS. This can lead to symptoms such as depression, emotional sensitivity, sudden mood swings, difficulty concentrating, and memory impairment. In addition, it has been observed that individuals are more sensitive to treatments during this period, nausea reflexes increase, and the pain threshold decreases. PMS occurs in 70% of menstruating women, but only 5% are clearly diagnosed.⁴⁶

Due to gum bleeding and increased sensitivity associated with the menstrual cycle, periodontal examination should be performed with care. Although periodontal control intervals vary according to individual needs, follow-up every 3-4 months is recommended in problematic cases. Antimicrobial mouthwashes may be an option in cases of cycle-related inflammation; however, unnecessary interventions should be avoided by first evaluating the individual's oral hygiene habits.⁴⁶

Surgical procedures should be postponed until after the menstrual cycle, if possible, in patients with a history of excessive bleeding during menstruation or who are at high risk of bleeding after surgery. Considering the risk of anemia in such patients, it is recommended that the necessary blood tests be performed and consultation with relevant specialists be provided before surgical intervention.⁴⁶

During PMS, many women experience:

- Fatigue
- Increased cravings for sweet and salty foods
- Bloating in the abdominal area
- Swelling in the hands and feet
- Headaches
- Breast tenderness
- Nausea or feeling nauseous
- Gastrointestinal discomfort and other physiological symptoms.⁴⁶

PREGNANCY

During pregnancy, significant physiological changes occur in both the mother and the baby. During this process, changes in the mother's immune system can increase susceptibility to infections, and periodontal infections are among these conditions.⁴⁷ Although there is no definitive evidence, it has been suggested that periodontal infections may be associated with adverse pregnancy outcomes such as preterm birth, low birth weight, gestational hypertension, preeclampsia, gestational diabetes, and miscarriage (abortus).⁴⁸

Effects of Pregnancy on Periodontal Tissues

Hormonal imbalances and decreased immune response during pregnancy can increase the response of periodontal tissues to irritation, affecting the clinical and biological characteristics of infections.⁴⁹ However, hormonal changes alone are not expected to cause gingivitis; bacterial plaque and gum inflammation are also necessary for this.⁵⁰ In many cultures, there is a widespread belief that every pregnancy causes tooth loss in the mother. Although epidemiological studies on the relationship between pregnancy and tooth loss have yielded different results, there is a general consensus that the prevalence and severity of gingivitis increase during pregnancy.⁵¹

Changes in estrogen and progesterone levels during pregnancy cause changes in the composition of the subgingival microflora. During this period, the ratio of anaerobic microorganisms to aerobic microorganisms increases. Some periodontal pathogens, such as *P. intermedia*, *Bacteroides* species, and *Campylobacter rectus* (*C. rectus*), have been found to be present at higher levels during pregnancy. This increase in pathogens affects the interaction between the periodontal microflora and the host, creating a higher susceptibility to periodontal tissue damage.⁴⁹

Machado and colleagues⁵² reported that oral bacteria such as *F. nucleatum*, *P. gingivalis*, *A.a.*, *T. denticola*, *C. rectus*, and *T. forsythia* can cross into the pregnant uterus regardless of the presence of clinical periodontitis, leading to local inflammation and adverse pregnancy outcomes. They emphasized that high levels of these pathogens in pregnant individuals may increase the risk of adverse pregnancy outcomes and that early diagnosis and additional care are important for controlling these microorganisms.

Although the exact mechanism of the increase in gingival inflammation observed during pregnancy is not fully understood, changes in neutrophil function, modifications in the cellular and humoral immune systems, hormone-induced changes in cellular physiology, and local effects on the microbial flora are thought to play an important role in this process.⁴⁹

Pregnancy gingivitis: Pregnancy gingivitis is a condition that can be seen in 30 to 100 percent of pregnancies and was first described by Pinard in 1877.⁵³ It is characterized by redness, swelling, gum enlargement, and increased bleeding tendency. Although histologically similar to classic gingivitis, its etiological factors differ.⁵⁴ Periodontal health status prior to pregnancy may be a determining factor in the rate and severity of the disease. While the anterior regions and interproximal areas are more frequently affected, increased swelling can lead to increased pocket depth and tooth mobility. The gums are the area most affected by this process, followed by the tongue, cheek mucosa, and palate, which may also show changes.⁵³

Pregnancy tumor: Pyogenic granuloma, also known as pregnancy tumor, pregnancy epulis, or granuloma gravidarum, occurs in 0.2% to 9.6% of pregnancies, usually during the second and third months of pregnancy.⁵³ This condition develops as a result of hormonal changes that occur during pregnancy. The effect of estrogen on macrophages, leading to an increase in vascular endothelial growth factor, local irritants, and bacterial factors cause this vascular lesion to grow.⁵⁵ Histologically, it is a vascular lesion resembling granulation tissue and exhibiting high proliferative properties.⁵⁶

Pyogenic granulomas seen during pregnancy usually shrink and disappear spontaneously after birth as hormonal balance returns to normal. In this respect, they differ from other pyogenic granulomas.⁵⁷

The lesion may be flat or lobulated, with an exophytic structure; it may be erythematous, pedunculated, or sessile. Although it is mostly localized in the gingiva, it can also develop on the tongue, palate, and buccal mucosa.⁵⁸ It has a fragile surface prone to bleeding and may be pink, red, or purple in color. This color variation may differ depending on the age of the lesion, the level of vascularization, and venous circulation.⁵⁸ Bone loss is not usually observed in pregnancy-related pyogenic granuloma.⁵³



If the lesion is not bleeding, does not affect chewing function, and gradually shrinks after birth, only regular follow-up is recommended instead of surgical intervention.⁵⁵ However, pyogenic granulomas removed surgically may recur due to incomplete surgery or poor oral hygiene.⁵⁷

Clinical and microbial changes in periodontal tissues during pregnancy:

- Increased gingival probing depths
- Increased gingival inflammation
- Increased gingival crevicular fluid
- Increased bleeding on probing
- Increased tooth mobility
- Increased incidence of pyogenic granulomas
- Increased number of periodontal pathogens, particularly *P. gingivalis* and *P. intermedia*.⁵⁹

Effects of Periodontal Disease on Pregnancy

Numerous epidemiological studies examining the relationship between periodontal infections and adverse pregnancy outcomes have demonstrated a statistically significant association between the two conditions.⁶⁰ However, some studies have reached opposite conclusions, suggesting that periodontal disease has no significant effect on pregnancy outcomes. Although the exact cause of these conflicting findings is unclear, genetic and environmental factors that vary across populations are thought to be one of the key determinants of susceptibility to adverse pregnancy outcomes. Adverse pregnancy outcomes include preterm birth, very preterm birth, low birth weight, very low birth weight, miscarriage, and preeclampsia.⁶¹

Preterm birth is defined as birth occurring before the 37th week of pregnancy and is seen in approximately 10-15% of pregnancies.⁶² According to World Health Organization (WHO) data, the preterm birth rate was reported as 9.6% between 1997 and 2007.

Births occurring before the 32nd week of pregnancy are referred to as very preterm births.⁶³ Low birth weight was defined by the WHO in 1976 as a birth weight of less than 2500 g. A weight of less than 1500 g is referred to as “very low birth weight”.⁶⁴

Infection is reported to be one of the main factors in 30-50% of cases of preterm birth and low birth weight. Infections occurring in the cervical region of the uterus, in particular, significantly increase these risks.⁴⁹ Periodontal disease is an infectious condition caused by gram-negative anaerobic bacteria.⁶¹

Colins et al.⁶⁵ reported that periodontal infections may lead to low birth weight by triggering cellular immune mechanisms that produce cytokines such as interleukin-1 beta (IL-1 β), TNF- α , and prostaglandin E (PGE₂) through bacterial activation.

The relationship between periodontal disease, preterm birth, and low birth weight can be explained by three mechanisms:

- Dissemination of inflammatory products into the bloodstream
- The immune response of the mother/fetus to oral pathogens
- Dissemination of oral bacteria through the bloodstream.⁶⁶

In some cases, it has been observed that amniotic fluid infected by oral microorganisms can trigger premature birth. In this context, it is noted that bacteria such as *Streptococcus* species, *E. corrodens*, *F. nucleatum*, and *P. gingivalis* may play a role.⁴⁹

Clinical approach to the pregnant patient: An important component of periodontal examination is taking a detailed medical history.

Immunological changes that occur during pregnancy can cause an increase in blood pressure. In addition, considering the physiological interactions between the fetus and the mother, the physician must carefully evaluate the patient.⁴⁶

The medical history should focus particularly on:

- Pregnancy-related complications
- Previous miscarriages
- Cramps, vomiting
- Vaginal bleeding in the form of spotting or staining.⁴⁶

To accurately determine the patient's periodontal and dental treatment needs, communication with the obstetrician is essential, and information about the patient's general health status should be obtained.

It should be explained in detail that pregnant individuals are more prone to gingival inflammation, effective oral hygiene techniques should be taught, and they should be encouraged to seek regular care.

During pregnancy, tartar removal, polishing, and root planing procedures can be performed when necessary. When mouthwash use is recommended, alcohol-free formulations should be preferred.⁴⁶

Elective dental treatments should be limited to maintaining good oral hygiene and should be postponed as much as possible during the first and second half of the third trimester of pregnancy.

The first trimester is a period when the fetus is extremely sensitive to environmental factors due to the organogenesis process. In the latter half of the third trimester, the uterus becomes sensitive to external stimuli and the risk of premature birth may increase. The patient should not be seated for long periods of time because supine hypotensive syndrome may occur. The pressure exerted by the pregnant uterus on the inferior vena cava and pelvic veins causes a slowdown in venous return and a decrease in cardiac output, leading to hypotension. Women lying supine may experience dizziness, paleness, tachycardia, sweating, and nausea. In a semi-recumbent or supine position, the inferior vena cava is compressed by the weight of the uterus.

In supine hypotensive syndrome, turning the patient onto her left side is usually sufficient (**Figure 2**). This relieves pressure on the inferior vena cava, allowing blood to return from the pelvic area and lower extremities. A soft cushion approximately 15 cm thick (a rolled towel may be used) should be placed under the patient's right side when she is reclined for treatment.⁴⁶

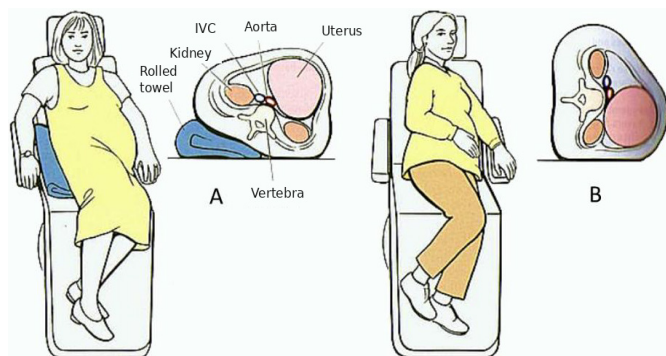


Figure 2. Patient position applied to prevent supine hypotensive syndrome⁴⁶

The second trimester is considered the most appropriate time for routine dental treatment in pregnant patients. During this period, the goal is to control existing diseases and prevent potential problems that may arise in the later stages of pregnancy.

Major oral and periodontal surgeries should be postponed until after delivery. However, in cases of pregnancy tumors causing pain, difficulty chewing, or suppuration with bleeding, a biopsy or excision of the lesion may be necessary before delivery if deemed necessary.⁴⁶

THE EFFECT OF ORAL CONTRACEPTIVE USE ON PERIODONTAL TISSUES

Steroid-containing medications taken orally by women to prevent pregnancy are called oral contraceptives. These medications contain estrogen and progestin hormones and prevent pregnancy by temporarily suppressing fertility.⁶⁷ Combination oral contraceptives used for contraception are usually taken daily as a single dose from the 5th day of the cycle until the 25th day, with a total of 21 pills used per cycle. These pills contain a combination of estrogen and progestin. Considered one of the most effective methods of preventing pregnancy, these drugs should ideally contain the lowest amount of hormones necessary to prevent pregnancy.⁶⁸

Hormonal changes can increase susceptibility to gingivitis by affecting the host's biological environment.⁶⁹ For example, during pregnancy, a higher-than-expected increase in plaque-related inflammation may be observed compared to the level of plaque accumulation.⁷⁰ In addition, it has been reported that the immune response to subgingival microbiota and bacterial antigens changes during pregnancy.⁷¹

Along with the rise in hormone levels, there is also an increase in the proportion of bacteria such as *P. intermedia*, which can use estrogen and progesterone instead of vitamin K. During this process, an increase in clinical susceptibility to gingivitis is observed, while these changes generally return to normal after delivery.⁷²

Clinical studies have shown that women using hormonal contraceptive drugs experience more inflammatory reactions than those who do not use them. However, it has not been definitively determined whether these drugs directly affect attachment levels. Gingivitis associated with oral contraceptive use shares similar characteristics with plaque-induced gingivitis and gingivitis observed during pregnancy. A significant increase in gingival crevicular fluid may be observed during this process. Additionally, it has been reported that the severity of gingivitis is related to the dose of the medication, with inflammatory reactions being milder

at lower doses. In some cases, it may be necessary to change or discontinue the medication based on the severity of gum symptoms.

Clinical studies have shown that the use of oral contraceptive agents may increase gum changes during the premenstrual period but does not cause significant tissue growth.⁷⁴ Furthermore, it has been reported that reducing the dose of contraceptive drugs or discontinuing their use alleviates or prevents gum discomfort.⁷³ Each of these mechanisms contributes to a stronger inflammatory response in the gums to local irritants. Bacterial plaque accumulating at the gum line causes mild inflammation in individuals who do not use oral contraceptives, while the artificial increase in systemic progesterone levels in individuals who use oral contraceptives may cause this inflammatory response to become more pronounced.⁷⁴

Changes observed in the periodontium with oral contraceptive use:

- Increased inflammation against local irritants
- Increased plaque levels, pocket formation, bleeding, and attachment loss
- Increased numbers of *Candida*, *P. gingivalis*, *P. intermedia*, and *A.a* species in periodontal pockets.⁷⁴

Consequently, the use of oral contraceptives containing estrogen and progesterone may cause an increase in the incidence of gingivitis by inducing effects similar to the hormonal changes seen during pregnancy. Furthermore, long-term use of hormonal contraceptives has been reported to cause progression of attachment loss in periodontal tissues.⁷⁴

MENOPAUSE

The WHO defines menopause as "the permanent cessation of menstruation following the end of ovarian function".⁷⁵ Clinically, menopause is confirmed 12 months after the last menstrual cycle, while the menopausal transition period generally lasts 4 to 7 years.⁷⁶ Global data shows the average age of menopause to be 51.⁷⁷ It has been reported that this range varies between 49.3 and 51.4 years in developed countries and between 43.5 and 49.4 years in developing countries.⁷⁸ According to a study conducted in Türkiye, the average age of menopause onset was 35, while the highest average age was recorded as 55. According to data from the 2013 Turkish Population and Health Survey, 49% of women aged 48-49 had entered menopause.⁷⁹

Katz and Epstein⁸⁰ suggested that androgens, together with estrogens, may play a fundamental role in maintaining bone health. They attributed this to the inhibitory effect of estrogens on osteoclastic activity. In the postmenopausal period, symptoms such as increased risk of osteoporotic fractures, myocardial infarction, menstrual irregularities, hot flashes, and night sweats may be observed.

Menopause not only causes systemic changes but also affects oral tissues. Changes in the mouth generally occur as a result of the aging process and decreased estrogen levels. Decreased estrogen levels cause reduced epithelial keratinization and decreased salivary flow rate.⁸¹ During this period, some women may experience symptoms such as menopausal gingivostomatitis, dry mouth, pale or red gums, and bleeding during probing and brushing.⁸²



Various changes in oral health may occur in women during menopause. Burning sensation in the mouth, dryness, and impaired taste are among the common complaints during this period. A burning sensation felt in the normal-looking oral mucosa is called "burning mouth syndrome" and is common in postmenopausal women. The symptoms of this syndrome can range from mild discomfort to severe pain. Some oral diseases, such as lichen planus, candidiasis, and viral infections, can cause similar symptoms, but no significant pathological changes are observed on the mucosal surface in burning mouth syndrome.⁸³

Wardrop et al.⁸⁴ investigated the relationship between menopause and oral disorders in a study of 149 women. According to the study results, the prevalence of oral disorders was 43% in perimenopausal and postmenopausal women, while this rate was found to be 6% in premenopausal women.

One of the most significant health problems encountered during menopause is osteoporosis. Osteoporosis is characterized by a decrease in bone density, which leads to an increased risk of fractures. Both osteoporosis and periodontal diseases are among the major health problems in older individuals. Studies have suggested that ovarian dysfunction and increased incidence of periodontal disease may be associated with a decrease in mandibular bone density. Osteoporosis leads to a decrease in alveolar bone volume in the crest region, and this bone loss becomes even more pronounced in the presence of periodontal disease.⁸⁵

The fact that a large proportion of patients with desquamative lesions are middle-aged and predominantly female (80%), along with the higher prevalence of diseases such as benign mucous membrane pemphigoid and lichen planus in women, suggests that sex steroid hormones may be associated with certain desquamative lesions. It has been reported that such lesions can be successfully managed with exogenous estrogen therapy.²³

Clinical and Microbiological Changes in the Periodontium During Menopause

In recent years, hormone replacement therapy (HRT) has been recognized as an effective method for managing symptoms associated with menopause. This treatment plays an important role in preventing postmenopausal osteoporosis by reducing bone mass loss. Furthermore, it has been suggested that HRT may contribute to the preservation of periodontal health in postmenopausal women.⁸⁶

Clinical symptoms associated with menopause:

- Decreased epithelial keratinization
- Reduced salivary flow, burning sensation in the mouth, dry mouth, and bad taste
- Increased bleeding during probing and brushing
- Increased alveolar bone loss due to increased osteoporosis
- Increased incidence of desquamative lesions has been reported.⁸⁶

HRT may contribute to the preservation of periodontal health during the postmenopausal period.⁸⁷ It has been suggested that estrogen supplementation may regulate periodontal tissue destruction by helping to reduce matrix metalloproteinases such as MMP-8 and MMP-9, as well as cytokines involved in bone resorption.⁸⁸ Some clinical studies have shown that HRT

has positive effects on tooth preservation and alveolar bone density.⁸⁹ In addition, it has been noted that the incidence of gingival bleeding is lower in women using HRT.⁹⁰

CONCLUSION

This review aims to examine the effects of hormonal changes observed in women on periodontal tissues. Numerous studies have shown that hormonal fluctuations during adolescence, the menstrual cycle, pregnancy, oral contraceptive use, and menopause affect the course of periodontal disease by increasing gingival inflammation. The literature has concluded that there is an increase in the frequency of conditions such as gingivitis, periodontitis, and pyogenic granuloma during hormonal periods. Furthermore, it has been emphasized that periodontal diseases during pregnancy may be associated with adverse pregnancy outcomes such as preterm birth and low birth weight. It is necessary to consider hormonal changes and raise awareness in this regard for the preservation of periodontal health and management of treatment processes in female patients. These findings are expected to contribute to the development of more effective and individualized treatment strategies for female patients in clinical practice.

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Conflict of Interest

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