



Research Paper

## Periodontal Therapy in Female Patients – A Review

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Received 20 December, 2014; Accepted 31 December, 2014 © The author(s) 2014. Published with open access at [www.questjournals.org](http://www.questjournals.org)

**ABSTRACT:-** Throughout a woman's life cycle, the hormonal influences that take place can affect the therapeutic decision making in Periodontics. Periodontal and oral tissue responses may be altered as a result of the local factors and the hormonal influence on them, creating diagnostic and therapeutic dilemmas the advent of new research has provided keener appreciation of these unique systemic influences on oral, periodontal, and implant tissues. Oral health care professionals have greater awareness and capabilities of dealing with the various responses of the tissue during the reproductive process. Therefore it is imperative that the clinician recognizes, customizes, and appropriately alters the periodontal therapy according to the individual woman's needs based on the stage of her life cycle.

Various Periodontology books, articles from journals have been referred for compiling the effects, management of such effects on the periodontium.

**Keywords:-** Menses, Puberty, Osteoporosis, Oral contraceptives

**Key Messages:** Various oral and periodontal manifestations seen during the life cycle of females during puberty, pregnancy, oral contraceptive usage, menopause as an influence of hormonal changes is a very important from the point of periodontal treatment to maintain a healthy periodontium.

### I. INTRODUCTION

Main aim of any periodontal treatment is to establish a healthy tissue so that it becomes maintainable by the patient. However at times there are various contributing factors that exaggerate the existing periodontal disease. One such is observed in female patients where, as a result of hormonal influences seen during their life cycle at puberty, pregnancy, on usage of oral contraceptives, menopause the already existing periodontal disease worsens resulting in loss of periodontium. This article mainly deals clinical manifestations and management of such within the oral cavity.

The matter is compiled from various Periodontal books dealing with such topics as well as journals who have published research and review work on the same.

Thus Periodontal manifestations in a female's life are seen during these phases of:

#### 1. Puberty

##### Oral and periodontal manifestations

Puberty occurs between the average ages of 11 to 14 in most girls. The production of sex hormones (estrogen and progesterone) increases, and then remains relatively constant during remainder of the reproductive phase. Also the prevalence of gingivitis increases, without an increase in the amount of plaque. Gram-negative anaerobes, especially *Prevotella intermedia*, have been implicated in association with puberty gingivitis.

Kornman and Loesche (1979) postulated that these anaerobic organisms may use ovarian hormones as a substitute for vitamin K growth factor. Levels of black pigmented Bacteroides, especially Prevotellaintermedia (formerly known as Bacteroidesintermedius), are thought to increase levels of gonadotropic hormones in puberty. Capnocytophaga species also increase in incidence as well as proportion. These organisms have been implicated in the increased bleeding tendency observed during puberty.

**Clinical feature:**

During puberty, periodontal tissues may have an exaggerated response to local factors. A hyperplastic reaction of the gingiva may occur in areas where food debris, materia-alba, plaque and calculus are deposited. The inflamed tissues become erythematous, lobulated, and retractable. Bleeding may occur easily from the gingival tissues when mechanical debridement of the local irritants is carried out. Histologically the appearance of the gingiva is consistent with inflammatory hyperplasia.

The clinician should recognize the intraoral effects of chronic regurgitation of gastric contents on intraoral tissues which is a common complaint in the puberty phase. This age group is also susceptible to eating disorders, namely, bulimia and anorexia nervosa. Perimylosis (smooth erosion of enamel and dentin) typically on the palatal surfaces of maxillary anterior teeth varies with duration and frequency of eating disorder.

**Management**

1. During puberty education of the parent or the care giver and patient is a part of successful periodontal therapy.
2. Preventive care, including a vigorous program of oral hygiene is also vital. Proper brushing technique and use of other oral hygiene aids is recommended.
3. Milder gingivitis cases respond well to scaling and root planing, with frequent oral hygiene reinforcement.
4. Severe cases of gingivitis may require:
  - a. Scaling and root planning
  - b. Microbial culturing
  - c. Antimicrobial mouthwashes
  - d. Local site delivery
  - e. Antibiotic therapy
5. Periodontal maintenance appointments may need to be more frequent when periodontal instability is noted.
6. Also enlargement of the parotid glands has been estimated to occur in 10% to 50% of patient who binge and purge (vomit). Therefore diminished salivary flow rate may also be present, which will increase oral mucous membrane sensitivity, gingival erythema and caries susceptibility

**1. Menses**

**Oral and Periodontal Manifestations**

During the reproductive years, the ovarian cycle is controlled by the anterior pituitary gland which secretes Follicle stimulating hormone (FSH) and Luteinizing hormone (LH) are produced from anterior pituitary gland. Ongoing changes in the concentration of the gonadotropins and ovarian hormones occur during monthly menstrual cycle.

The monthly reproductive cycle has two phases. The first phase is referred to as follicular phase. Levels of FSH are elevated, and estradiol (the major form of estrogen) is synthesized by the developing follicle and peaks approximately 2 days before ovulation. The second phase is called luteal phase. The developing corpus luteum synthesizes both estradiol and progesterone. Estrogen peaks at 0.2 ng/ml and progesterone at 10.0 ng/ml to complete the rebuilding of the endometrium for implantation of the fertilized egg. If fertilization and implantation does not occur the corpus luteum involutes, ovarian hormone level drops and menstruation ensues.

It has been postulated that ovarian hormones may increase inflammation in gingival tissues and exaggerate the response to local irritants. The main potential effects of these hormones on the periodontal tissues can be summarized as:

1. Estrogen affects salivary peroxidases, which are active against a variety of microorganisms by changing the redox potential. (Kimura et al. 1983)
2. Estrogen has stimulatory effects on the metabolism of collagen and angiogenesis. (Sultan et al. 1986)
3. Estrogen can trigger autocrine or paracrine polypeptide growth factor signalling pathways, whose effects may be partially mediated by the estrogen receptor itself. (Chau et al. 1998)
4. Estrogen and progesterone can modulate vascular responses and connective tissue turnover in the periodontium, associated with interaction with inflammatory mediator. (Soory 2000)

5. Progesterone has been associated with increased permeability of the microvasculature altering the rate and pattern of collagen production in the gingiva.
6. Progesterone plays role in stimulating the production of prostaglandins that mediate the body's response to inflammation.
7. PGE<sub>2</sub> is one of the major secretory products of monocytes and is increased in inflamed gingiva. (Pack AR, Thomson 1982)

### **Management**

1. Increased gingival bleeding and tenderness require closer periodontal monitoring, periodontal check up within 3 to 4 months recommended.
2. For patient with history of excessive postoperative haemorrhage or with excessive menstrual flow, scheduling surgical visits after cyclic menstruation is prudent.
3. If anaemia is common, an appropriate consultation with the physician and recent laboratory test is needed.
4. The clinician should be aware that NSAIDs, infection and acidic food exacerbate Gastroesophageal Reflux Disease (GERD). Patient taking over-the counter antacids, H<sub>2</sub> receptor antagonist and proton pump inhibitors may be GERD patients. These medications interact with some antibiotics and antifungals so review of their pharmacology is necessary.

## **II. ORAL CONTRACEPTIVE USAGE**

Contraceptives utilize synthetic gestational hormones (estrogen and progesterone), to reduce the likelihood of ovulation/implantation. (Guyton 1987). Women using oral contraceptives show elevated plasma levels of several clotting factors, related to the dose of estrogen. Less dramatic but similar effects to pregnancy are sometimes observed in the gingiva of hormonal contraceptive users.

### **Special Features**

An exaggerated response to local irritants is seen in patients on oral contraceptives. Oral contraceptive associated gingival inflammation may be chronic (Lindhe & Bjorn 1967, Lynn 1967, Groen et al. 1968, el-Ashiry et al. 1971, Knight & Wade 1974, Pankhurst et al. 1981). Investigators suggest several mechanism for exaggerated response like, increased vascular permeability, altered microvasculature, synthesis of prostaglandin. Hyperplastic gingival tissue, more formation of exudate, increase in bacteroides species and an increased incidence of localized osteitis following extraction of mandibular third molars is seen (Catellani 1979). OC's are also correlated with gingival melanosis.

Knight & Wade (1974) did not find any significant differences in Plaque Index and Gingival Index scores and attachment level between women using oral contraceptives and controls but noted that women using these for more than one and a half years tended to have higher Gingival Index scores and more loss of attachment. No significant influences on the periodontal clinical parameters were noticed when comparing oral contraceptives to non-medicated control groups (Moshchil et al. 1991)

### **Management**

1. Medical history
2. Patient should be informed about the oral and periodontal side effects of OCs.
3. Good meticulous home care and compliance with regular periodontal maintenance.
4. Scaling and root planning at regular 6monthly intervals.

## **III. PREGNANCY**

### **Oral and Periodontal Manifestations**

The link between pregnancy and periodontal inflammation has been known for many years. In 1778, Vermeeren discussed tooth pains in pregnancy. In 1818, Pitcairn described gingival hyperplasia in pregnancy. In 1877, Pinard recorded the first case of pregnancy gingivitis.

During pregnancy, the increased levels of sex steroid hormones are maintained from the luteal phase which results in implantation of the embryo, until parturition. Pregnant women, near or at term, produce large quantities of estradiol (20 mg/day), estriol (80mg/day) and progesterone (300 mg/day).

Gingival inflammation initiated by plaque, and exacerbated by these hormonal changes in the second and third trimester of pregnancy, is referred to as pregnancy gingivitis. Parameters such as gingival probing depths (Hugoson 1970, Miyazaki et al. 1991), bleeding on probing (Miyazaki et al. 1991) and crevicular fluid flow (Hugoson 1970) were found to be increased.

Robinson & Amar (1992) reviewed the influence of pregnancy on the oral cavity and described 4 oral pathological conditions that included

- a) Pregnancy gingivitis, b) pregnancy granuloma, c) periodontitis and d) dental caries.

### a) **Pregnancy Gingivitis**

In 1877, Pinard recorded the first case of “pregnancy gingivitis”. Epidemiological studies of pregnancy gingivitis showed a prevalence ranging from 35% (Hasson 1966) to 100% (Lundgren et al 1973). It is characterized by erythema, edema, hyperplasia, and increased bleeding. Histologically the description is the same as gingivitis. The anterior region of the mouth is affected more often, and interproximal sites tend to be more involved. (De Liefde 1984)

Increased tissue edema leads to increased pocket depths and relate to transient tooth mobility. Anterior site inflammation may be exacerbated by increase in mouth breathing because of pregnancy rhinitis.

In a study of 130 pregnant women, Machuca et al. (1999) demonstrated gingivitis in 68% of the population, ranging from 46% in technical executives to 88% in manual workers. Cross-sectional studies examining pregnant and postpartum women have shown that pregnancy is associated with significantly more gingivitis than at postpartum, despite similar plaque scores (Silness & Loe 1963).

A more recent study of a rural population of Sri Lankan women (Tilakaratne et al. 2000) showed increased gingivitis of varying degrees of significance amongst all the pregnant women investigated, compared with matched non-pregnant controls. There was a progressive increase in inflammation with advancing pregnancy which was more significant in the second and third trimester of pregnancy, despite the plaque levels remaining unchanged. At the third month after parturition, the level of gingival inflammation was similar to that observed in the first trimester of pregnancy. This suggests a direct correlation between gingivitis and sustained, raised levels of gestational hormones during pregnancy, with regression during the postpartum period.

In investigations by Cohen et al. (1969) and Tilakaratne et al. (2000) the values for loss of attachment remained unchanged during pregnancy and three months postpartum.

### **Effects on the microbiota**

There is an increase in the selective growth of periodontal pathogens such as *Prevotella intermedia* in subgingival plaque during the onset of pregnancy gingivitis at the third to fourth month of pregnancy. The gestational hormones act as growth factors, by satisfying the naphthoquinone requirement for bacteria. These findings were also confirmed by Muramatsu & Takaesu (1994) who showed that from the third to fifth month of pregnancy, the number of gingival sites which bled on probing corresponded with the percentage increase in *Prevotella intermedia*.

A 55-fold increase in the proportion of *P. intermedia* has been demonstrated in pregnant women compared with non-pregnant controls (Jensen et al. 1981), implying a role for gestational hormones in causing a change in microbial ecology in the gingival pocket.

### b) **Pregnancy granuloma/Pregnancy tumor/ and epulis gravidarum**

A pregnancy tumour is an pedunculated, sessile fibro-granulomatous lesion which sometimes develop during pregnancy. A combination of the vascular response induced by progesterone and the matrix stimulatory effects of estradiol, contribute to the development of pregnancy granulomas, usually at sites with pre-existing gingivitis. The lesions often occur in the anterior papillae of the maxillary teeth and usually do not exceed 2 cm in diameter. They can bleed when traumatized and their removal is best deferred until after parturition, when there is often considerable regression in their size (Wang et al. 1997).

Surgical removal of the granuloma during pregnancy can result in recurrence due to a combination of poor plaque control and hormone mediated growth of the lesion. Careful oral hygiene and debridement during pregnancy are important in preventing its occurrence. (Wang et al. 1997)

Laser surgical excision of the lesion is recommended, as opposed to scalpel, for less postsurgical bleeding (Pick et al 1987). Surgical removal is usually performed after parturition. However, if the lesion causes functional problems or appears to have deleterious effects on the adjacent periodontium, it can be safely removed under local anesthesia throughout a normal pregnancy, preferably during the second trimester.

### **Clinical Management**

1. A Careful medical history of the patients should be taken and the obstetrician should be contacted if required, to discuss her medical status, periodontal needs and the proposed treatment plan.
2. Plaque control
  - a. Scaling, polishing and root planning may be performed whenever necessary.
3. Use of prenatal fluoride has been an area of controversy.
4. Dental treatment should be avoided in the first and last phase of the third trimester. Second trimester is the safest period for dental treatment.
5. Safety precaution should be taken while taking dental radiographs. (eg- patient protection by using lead apron, etc.)
6. Certain drugs which cross the placental barrier should be avoided.

(eg- tetracyclines, metronidazole )

**Elective Dental Treatment**

Pregnant women need to be educated on the consequences of pregnancy on gingival tissues and should be thoroughly motivated in plaque control measures, with professional treatment as and when required. They are likely to be more comfortable to receive dental treatment during the second trimester than in the first or third trimester of pregnancy, although emergency treatment is permissible at any stage during pregnancy (Amar & Chung 1994).

**Medications**

Any form of medication during pregnancy must only be used if the gravity of the condition being treated outweighs the consequences. Amongst the antibiotics, tetracycline, vancomycin and streptomycin can contribute to staining of teeth and ototoxic and nephrotoxic effects during 4-9 months of pregnancy; erythromycin, penicillins and cephalosporins are relatively safer, but any medication must only be administered in consultation with the patient's obstetrician. (Lynch et al. 1991)

The classification system established by US FOOD AND DRUG ADMINISTRATION IN 1979 to rate fetal risk levels associated with many prescription drugs provides safety guidelines (table I).

Table 1 FDA pregnancy risk factor definitions <sup>20</sup>	
Category	Definition
A	Controlled studies in women fail to demonstrate a risk to the fetus in the first trimester (and there is no evidence of risk in later trimesters), and the possibility of fetal harm appears remote.
B	Either animal reproduction studies have not demonstrated a fetal risk but there are no controlled studies in pregnant women or animal reproduction studies have shown an adverse effect (other than a decrease in fertility) that was not confirmed in controlled studies in women in the first trimester (and there is no evidence of risk in later trimesters).
C	Either studies in animals have revealed adverse effects on the fetus (teratogenic, embryocidal, or other) and there are no controlled studies in women or studies in women and animals are not available. Drugs should be given only if the potential benefit justifies the potential risk to the fetus.
D	There is positive evidence of human fetal risk, but the benefits of use in pregnant women may be acceptable despite the risk (for example, if the drug is needed in a life-threatening situation or for a serious disease for which safer drugs cannot be used or are ineffective).
X	Studies in animals or human beings have demonstrated fetal abnormalities or there is evidence of fetal risk based on human experience, or both, and the risk of the use of the drug in pregnant women clearly outweighs any possible benefit. The drug is contraindicated in women who are or may become pregnant.

A list of Local anesthetic drugs during pregnancy (table II).

DRUG	FDA CATEGORY	DURING PREGNANCY
<b>LOCAL ANESTHETICS</b>		
LIDOCAINE	B	YES
MEPIVACAINE	C	USE WITH CAUTION
PRILOCAINE	B	YES
BUPIVACAINE	C	USE WITH CAUTION
ETIDOCAINE	B	YES
PROCAINE	C	USE WITH CAUTION
ARTICAINE	B	YES

A list of Analgesics during pregnancy (table III).

DRUG	FDA CATEGORY	DURING PREGNANCY
<b>ANALGESICS</b>		
ASPIRIN	C/D, THIRD TRIMESTER	CAUTION AVOID
ACETAMINOPHEN	B	YES
IBUPROFEN	B/D, THIRD TRIMESTER	CAUTION AVOID
CODEINE	C	USE WITH CAUTION
HYDROCODONE	B	USE WITH CAUTION
OXYCODONE	B	USE WITH CAUTION
PROPOXYPHENE	C	USE WITH CAUTION

A list of antibiotic administration during pregnancy (table IV).

DRUG	FDA CATEGORY	DURING PREGNANCY	RISKS
PENICILLINS	B	YES	DIARRHEOA
ERYTHROMYCINE	B	YES	JAUNDICE
CLINDAMYCINE	B	YES WITH CAUTION	DRUG CONC. IN FETAL BONE, SPLEEN, LUNG
CEPHALOSPORINS	B	YES	LIMITED INFORM.
TETRACYCLINE	D	AVOID	DEPRESSION OF BONE GROWTH, TOOTH DISCOLORATION
CIPROFLOXACIN	C	AVOID	CARTILAGE EROSION
METRONIDAZOLE	B	AVOID	CARCINOGENIC
GENTAMICIN	C	CAUTION	OTOTOXICITY
VANCOMYCINE	C	CAUTION	LIMITED INFORMATION
CLARITHROMYCINE	D	AVOID	LIMITED INFORMATION

The amount of drug excreted in breast milk is usually not more than 1% to 2% of the maternal dose therefore it is highly unlikely that most drugs have any pharmacologic significance for the infant. The mother should take prescribed drugs just after breastfeeding and then avoid nursing for 4 hours or more if possible to decrease drug concentration in breast milk. Dental Drug Administration during Breastfeeding are mentioned in table V

DRUG	DURING BREASTFEEDING
LIDOCAINE	YES
MEPIVACAINE	YES
PRILOCAINE	YES
BUPIVACAINE	YES
ETIDOCAINE	YES
PROCAINE	YES
ARTICAINE	YES
ASPIRIN	AVOID
ACETAMINOPHEN	YES
IBUPROFEN	YES
CODEINE	YES
HYDROCODONE	NO DATA
OXYCODONE	YES
PROPOXYPHENE	YES
PENICILLINS	YES
ERYTHROMYCINE	YES
CLINDAMYCINE	YES WITH CAUTION
CEPHALOSPORINS	YES
TETRACYCLINE	AVOID
CIPROFLOXACIN	AVOID
METRONIDAZOLE	AVOID
GENTAMICIN	AVOID
VANCOMYCINE	AVOID
CLARITHROMYCINE	AVOID

#### IV. MENOPAUSE AND OSTEOPOROSIS

##### Oral and Periodontal Manifestations

During menopause there is a decline in hormonal levels due to decreased ovarian function. This is characterized by tissue changes such as desquamation of gingival epithelium and osteoporosis which may be attributed to hormone deficiency. **Osteopenia and Osteoporosis**

Osteoporosis and osteopenia are characterized by reductions in bone mass and may lead to skeletal fragility and fracture. In 1994 the World Health Organization defined osteoporosis as a bone mineral density level more than 2.5 standard deviations below the mean of young, normal women.

Osteopenia is a reduction in bone mass due to an imbalance between bone resorption and formation, favoring resorption and resulting in demineralization and osteoporosis.

It has been demonstrated that women with early onset of menopause have a higher incidence of osteoporosis and significantly lower bone mineral density. (Kritz-Silverstein & Barrett-Connor 1993). A third of women over age 60 are affected by postmenopausal osteoporosis. (Baxter 1987)

The changes involved in **postmenopausal osteoporosis** are a reduction in bone density, affecting its mass and strength without significantly affecting its chemical composition. The circulating levels of estrogen have been shown to have an influence on alveolar bone density in postmenopausal women. (Payne et al. 1997). Estrogen is responsible for both arterial and venous effects, while progesterone affects arterial changes.

#### **Relationship between systemic and Mandibular bone density**

It has long been postulated that mandibular bone density may be indicative of systemic bone mineral density. Kribbs et al. (1983) addressed this relationship in both normal and osteoporotic women. Von Wewern et al. (1994) reported that 12 osteoporotic subjects with a history of fractures had less mandibular bone mineral content as measured by dual photon absorptiometry than 14 normal women.

More recent studies in postmenopausal women have indicated that a relationship may exist between osteoporosis and recession (Mohammad AR et al. 1996). 70 post-menopausal women with periodontitis have been studied by Wactawski-Wende et al. (2000) to test the hypothesis that systemic bone mineral density is related to periodontitis. Positive and significant correlations were seen between alveolar bone loss and bone mineral density.

#### **Oral changes seen in Osteoporotic patients**

1. Inflammatory changes in gingiva
2. Thinning of oral mucosa
3. Burning mouth
4. Gingival recession
5. Xerostomia
6. Altered taste sensations
7. Ridge resorption

#### **Effect of osteoporosis upon periodontium**

1. Poor wound healing: less attachment formation (von Wewern et al. 1994)
2. Reduced bone mineral content in the jaws (von Wewern et al. 1994, Payne et al. 1999)
3. Increase of periodontitis and tooth loss (Mittermayer et al. 1998)

#### **Common strategies for treatment of osteoporosis and periodontal disease**

In order to maximize the likelihood that bone mass is maintained over a lifetime, load-bearing exercise (bruxism, clenching) should be treated. Like periodontal disease, smoking is a major risk factor for osteoporosis and avoidance of smoking or smoking cessation contributes to osseous health.

**Hormone replacement therapy (HRT)** is designed to replace estrogen after menopause since this immediate post-menopausal period is a time of rapid loss of bone mineral density. Use of **sodium fluoride**, as well as **vitamin D** metabolites to correct malabsorption of calcium has been shown to be of some value in established osteoporosis. **Alendronate** has been shown to inhibit loss of bone density and decrease the risk of fracture without disturbance of bone healing observed in earlier drugs. (Papapoulos SE 1996). Few studies have directly assessed the relationship between periodontal disease and its sequelae in women receiving hormone replacement therapy. Jacobs et al. (1996) found a significant but moderate correlation. Nordey in 1993 reported no difference in clinical attachment level or alveolar bone loss. However, estrogen replacement therapy was associated with less gingival bleeding in women aged 50 to 64, as compared to an age-matched control group.

Two longitudinal studies were done to determine if hormone replacement therapy reduced the number of lost teeth in post-menopausal women. These were a Three year study of 42,171 postmenopausal women in the Nurses Health Cohort and the 10-year study of 3,921 women living in a retirement community in the Leisure World Cohort. Leisure World Cohort taking estrogen experienced a 36% reduction in tooth loss, and the Nurses Health Cohort showed an inverse relationship between hormone replacement therapy and loss of teeth after correcting for smoking and age.

## V. CONCLUSION

Thus the female body has a series of reaction to the hormonal changes seen within. Improper oral hygiene and hormonal imbalance seen during different life cycle of female patients exaggerates the oral tissues response to the plaque and other local factors thus worsening the condition. Prevention is better than cure, hence necessary precautions need to be taken at the earliest once the condition of the patient is known.

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