Role of Endogenous Female Sex Hormones On The Periodontium

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Introduction

Many systemic conditions can be considered as the risk factors or risk indicators for adverse periodontal diseases. Most significant advances in our understanding of pathogenesis of periodontitis are host immune responses. It can be altered, deficient or exaggerated to bacterial pathogens and may lead to more severe forms of disease. Systemic diseases alter the host tissues and physiology which impair host's barrier function and immune response to perio-pathogens creating opportunity for destructive periodontal diseases.

Currently accepted periodontal disease classification acknowledges the infiuence of endogenously produced female sex steroid hormones on the periodontium. These effects are primarily seen as gingival manifestations. Under the broad category of dental plaque-induced gingival diseases that are modified by systemic factors, those associated with the endocrine system are classified as puberty, menstrual cycle, or pregnancy-associated gingivitis.

Female sex steroid hormones are neither necessary nor sufficient to produce gingival changes by themselves. However, they may alter periodontal tissue responses to microbial plaque, and thus indirectly contribute to periodontal disease progression.(1)

Periodontal Menifestations Related To Female Sex Steroid Hormone:

Puberty:

Puberty is often accompanied by an exaggerated response of the gingiva to plaque.(1) Pronounced inflammation, edema, and gingival enlargement result from local factors that might ordinarily elicit a comparatively mild gingival response as shown in following clinical picture.

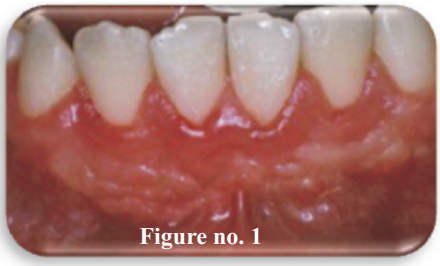

Figure no. 1

As adulthood approaches, the severity of the gingival reaction diminishes, even when local factors persist. However, complete return to normal health requires removal of these factors. Although the prevalence and severity of gingival disease are increased in puberty, gingivitis is not a universal occurrence for all adolescents, with good oral hygiene, it can be prevented. Gingivitis in puberty, with edema, discoloration, and enlargement of the entire gingival margin and papillary areas is commonly seen around the mandibular incisors.

Menstruation:

During the menstrual period, the prevalence of gingivitis increases. Some patients may complain of bleeding gums or a bloated, tense feeling in the gums in the days preceding menstrual flow. The exudate from inflamed gingiva is increased during menstruation, suggesting that preexisting gingivitis is aggravated by menstruation, but the crevicular fluid of normal healthy gingiva is unaffected.(2) Tooth mobility does not change significantly during the menstrual cycle.(3) The salivary bacterial count is increased during menstruation and at ovulation up to 14 days earlier.(4)

Pregnancy:

Gingival changes in pregnancy were described as early in the late 1800s, even before any knowledge about hormonal changes in pregnancy was available. As with other systemic conditions, pregnancy itself does not cause gingivitis. Gingivitis in pregnancy is caused by microbial plaque, just as it is in non-pregnant women. The hormonal changes of pregnancy accentuate the gingival response to plaque and modify the resultant clinical picture (fig. no. 2). No notable changes occur in the gingiva during pregnancy in the absence of local factors.

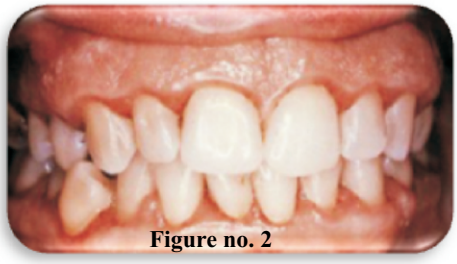

Figure no. 2

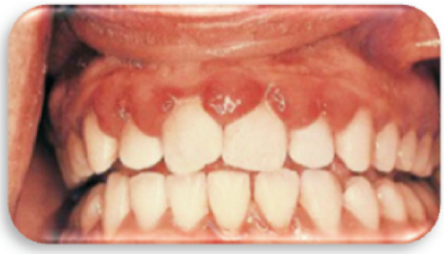

Figure no. 3

The reported incidence of gingivitis in pregnancy in well-conducted studies varies from 50% to 100%. (5) Pregnancy affects the severity of previously inflamed areas but does not alter healthy gingiva. Impressions of increased incidence may be created by the aggravation of previously inflamed but unnoticed areas. Tooth mobility, pocket depth, and gingival fluid are also increased in pregnancy. (6) The severity of gingivitis is increased during pregnancy beginning in the second or third month. Patients with mild chronic gingivitis that attracted no particular attention before the pregnancy become aware of the gingiva because previously inflamed areas become enlarged, oedematous, and more notably erythematous. Gingivitis becomes more severe by the eighth month and severity decreases during the ninth month of pregnancy. (5) Plaque accumulation follows a similar pattern. Some investigators report that the greatest severity is between the second and third trimesters.(7) The correlation between gingivitis and the quantity of plaque is greater after parturition than during pregnancy, which suggests that pregnancy introduces other factors that aggravate the gingival response to local factors.

Partial reduction in the severity of gingivitis occurs by 2 months postpartum, and after 1 year the condition of the gingiva is comparable to that of patients who have not been pregnant. Tooth mobility, pocket depth, and gingival fluid are also reduced after pregnancy. In a longitudinal investigation of the periodontal changes during pregnancy and for 15 months postpartum, no significant loss of attachment was observed. (7)

Pronounced ease of bleeding is the most striking clinical feature. The gingiva is inflamed and varies in color from a bright red to bluish red. The marginal and interdental

gingivae are edematous, pit on pressure, appear smooth and shiny, are soft and pliable, and sometimes present a raspberry-like appearance. The extreme redness results from marked vascularity, and there is an increased tendency to bleed. The gingival changes are usually painless unless complicated by acute infection. In some cases the inflamed gingiva forms discrete "tumorlike" masses, referred to as pregnancy tumors.

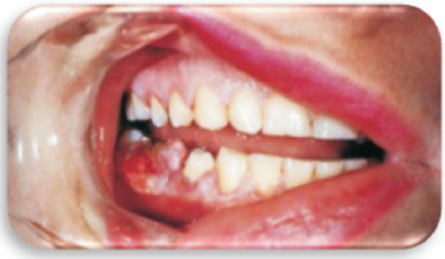

Figure no. 4

Microscopically, gingival disease in pregnancy appears as nonspecific, vascularizing, and proliferative inflammation. Marked inflammatory cellular infiltration occurs, with oedema and degeneration of the gingival epithelium and connective tissue. The epithelium is hyperplastic, with accentuated rete pegs, reduced surface keratinization, and various degrees of intracellular and extracellular oedema and infiltration by leukocytes. Newly formed engorged capillaries are present in abundance.

The possibility that bacterial-hormonal interactions may change the composition of plaque and lead to gingival inflammation has not been extensively explored. Kornman and Loesche (8) reported that the subgingival flora changes to a more anaerobic flora as pregnancy progresses. P.intermedia appears to be the only microorganism that increases significantly during pregnancy. This increase appears to be associated with elevations in systemic levels of estradiol and progesterone and to coincide with the peak in gingival bleeding. It has also been suggested that during pregnancy, a depression of the maternal T-lymphocyte response may be a factor in the altered tissue response to plaque.

The aggravation of gingivitis in pregnancy has been attributed principally to the increased levels of progesterone, which produce dilation and tortuosity of the gingival microvasculature, circulatory stasis, and increased susceptibility to mechanical irritation, all of which favor leakage of fluid into the perivascular tissues. A marked increase in estrogen and progesterone occurs during pregnancy, with a reduction after parturition. Animal studies with radioactive estradiol have demonstrated that the gingiva is a target organ for female sex hormones. The severity of gingivitis varies with the hormonal levels in pregnancy.

It has also been suggested that the accentuation of gingivitis in pregnancy occurs in two peaks:

-During the first trimester, when there is overproduction of gonadotropins.

-During the third trimester, when estrogen and progesterone levels are highest.

Destruction of gingival mast cells by the increased sex hormones and the resultant release of histamine and proteolytic enzymes may also contribute to the exaggerated inflammatory response to local factors.

Hormonal Contraceptives:

Hormonal contraceptives aggravate the gingival response to local factors in a manner similar to that seen in pregnancy and when taken for more than 1.5 years, increase periodontal destruction.(9) Although some brands of oral contraceptives produce more dramatic changes than others, no correlation has been found to exist on the basis of differences in progesterone or estrogen content in various brands. Cumulative exposure to oral contraceptives apparently has no effect on gingival inflammation or oral debris index scores.

Menopause:

During menopause the usual rhythmic hormonal fluctuations of the female cycle are ended as estradiol ceases to be the major circulating estrogen. As a result, females can develop a gingivostomatitis. This condition occurs during menopause or in the postmenopausal period. Mild signs and symptoms sometimes appear, associated with the earliest menopausal changes. Menopausal gingivostomatitis is not a common condition. The term used for its designation has led to the erroneous impression that it invariably occurs associated with menopause, whereas the opposite is true. Oral disturbances are not a common feature of menopause.

The gingiva and remaining oral mucosa are dry and shiny, vary in color from abnormal paleness to redness, and bleed easily. Fissuring occurs in the mucobuccal fold in some women, and comparable changes may occur in the vaginal mucosa. Microscopically, the gingiva exhibits atrophy of the germinal and prickle cell layers of the epithelium and, in some patients, areas of ulceration. The patient complains of a dry, burning sensation throughout the oral cavity, associated with extreme sensitivity to thermal changes; abnormal taste sensations described as "salty," "peppery," or "sour"; and difficulty with removable partial prostheses.

Conclusion:

It is clear that endogenous sex steroid hormones play significant roles in modulating the periodontal tissue responses and may alter tissue responses to microbial plaque and thus directly may contribute to periodontal disease. A better understanding of periodontal changes to varying hormonal levels throughout life can help the dental practitioner in the diagnosis and treatment. The influence of sex hormones on periodontal wound healing is still largely unclear. Further research is needed to improve the understanding of how endogenous sex steroid hormones influence the periodontal wound healing.

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