

Influence of female sex hormones on periodontium: A case series

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Abstract

Dental plaque is the primary etiologic factor for the periodontal diseases. Although pathogenic bacteria in dental plaque are necessary for the incidence of periodontal disease, but a susceptible host is as important. The susceptibility of the host can be modified by various systemic factors with hormones level being one. The periodontium shows an exaggerated inflammatory response to plaque modified by female sex hormone during puberty, pregnancy, in women taking oral contraceptives and at the postmenopausal stage. This paper presents such few cases where periodontium is influenced by variation in sex steroid hormones of female during different phases of their life time and to discuss how much a same hormone at different age and stage shows an exaggerated gingival response to plaque.

Key words: Female hormones, periodontium, postmenopause, pregnancy, puberty

INTRODUCTION

The variation in the level of female sex hormones estrogen and progesterone are responsible for various physiological changes in females at specific phases of their life. These changes not only affect other parts of the body, but also have significant influence on oral tissues as the receptors for estrogen and progesterone have been demonstrated in the gingiva, on the periosteal fibers, scattered fibroblasts of the lamina propria and also on periodontal ligament fibroblasts and osteoblasts proving the direct action of sex hormones on periodontal tissues. Sex steroid hormones directly and indirectly exert influences on cellular proliferation, differentiation and growth in target tissues. Estrogen can influence the cytodifferentiation of stratified squamous epithelium as well as the synthesis and maintenance of fibrous collagen.^[1] The action of the hormones on these

cells changes the effectiveness of the epithelial barrier to bacterial insult and collagen maintenance and repair. These hormones may alter immunologic factors and responses, including antigen expression and presentation, cytokine production as well as the expression of apoptotic factors and cell death. Progesterone, in particular, has been shown to stimulate the production of the inflammatory mediator, prostaglandin E2 and enhance the accumulation of polymorphonuclear leukocytes in the gingival sulcus and down regulates interleukin 6 production by human gingival fibroblasts.^[2] Estrogen and progesterone also has effect on the microcirculatory system, producing the following changes: Swelling of endothelial cells and pericytes of the venules, adherence of granulocytes and platelets to vessel walls, formation of microthrombi, disruption of

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perivascular mast cells, enhanced vascular permeability and vascular proliferation.^[3] As a result to these effects, an exaggerated inflammatory response of periodontal tissues may be seen during puberty, pregnancy and at the postmenopausal stage with the variation in the level of sex steroid hormone at different lifetimes. The paper presents such few cases and reviews how much a same hormone at different age and stage shows an exaggerated gingival response to plaque.

CASE REPORTS

Case report 1

A 15-year-old girl reported to the Department of Periodontics with a complain of swollen gums, which initiated 2 years back and gradually progressed to the extent that it caused difficulty in mastication and speech. She had no significant family history, medical history, and denied of taking any medication, which could have induced gingival enlargement. When examined intraorally, the gingiva was extensively enlarged, edematous, soft, friable, reddish pink in color with patches of melanin pigmentation and had a shiny surface [Figure 1a]. Bleeding occurred easily on probing. Moderate deposits of plaque and calculus were observed. Lab investigations for complete blood counts and hormone level for circulating estradiol, stimulating follicular hormone, luteinizing hormone were advised, which came out to be within normal limits with hormone level coinciding with the pubertal age. A diagnosis of pubertal gingival enlargement was made as the enlargement was initiated at around puberty. The patient was motivated and educated for taking proper oral hygiene measures and given treatment with thorough oral prophylaxis followed by gingivectomy full mouth in multiple sittings [Figure 1b]. An excised sample was given for histopathological examination

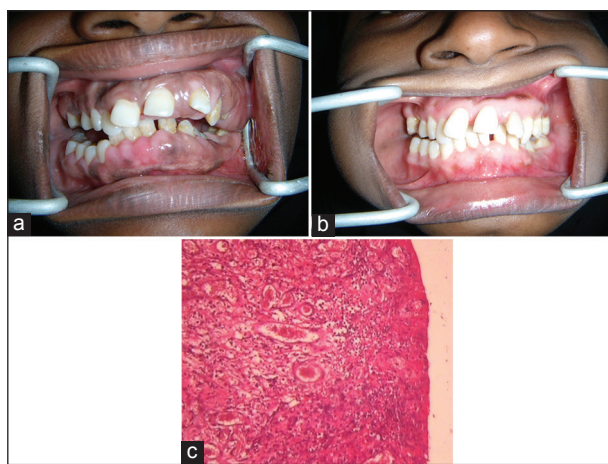


Figure 1: (a) Pubertal gingival enlargement before treatment. (b) Pubertal gingival enlargement after treatment. (c) Histopathological picture

which showed parakeratinized stratified squamous epithelium with densely arranged collagen fibers in the underlying connective tissue. Marked inflammatory edema and predominance of lymphocytes in addition to other inflammatory cells were observed all suggestive of inflammatory enlargement [Figure 1c].

Case report 2

A 37-years-old pregnant female patient reported to the Department of Periodontics with a complaint of swelling gum in upper front teeth and difficulty in eating food. She was in her 6 months of pregnancy. The patient did not take any medication in the past few months, not even her regular iron and calcium supplements probably because of her poor socioeconomic status and unawareness. An intraoral examination revealed grade 4 gingival enlargement in relation to 12, 13 with generalized inflammation and bleeding on probing [Figure 2a]. Her plaque index was very poor. The patient was diagnosed of having chronic generalized periodontitis with a localized pregnancy tumor. Full mouth scaling was performed in multiple short appointments. The patient was motivated for the maintenance of good oral hygiene and regular use of a toothbrush. The excision of the growth was deferred until postpartum. On recall visit, after pregnancy, the hyperplastic tissue that persisted was excised completely [Figure 2b] by a diode laser and sent for histopathological examination. The report showed hyperplastic stratified squamous epithelium with underlying connective tissue stroma showing increased collagen fibers and intense inflammatory cell infiltrate in a diffuse pattern suggestive of inflammatory gingival enlargement [Figure 2c].

Case report 3

A female patient with age 54 years reported with a complaint of burning sensation in her mouth. Her



Figure 2: (a) Pregnancy tumor before treatment. (b) Pregnancy tumor after treatment. (c) Histopathological picture

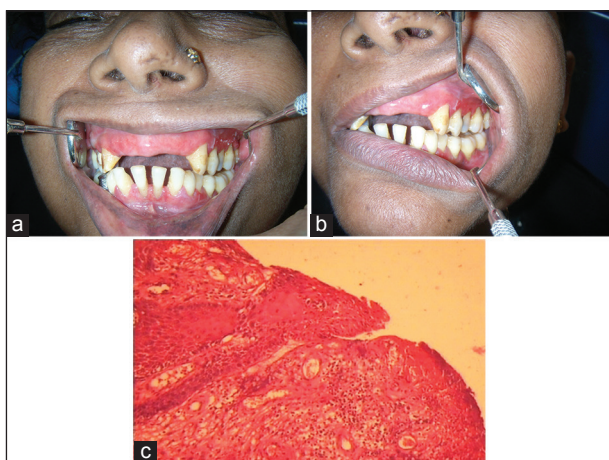


Figure 3: (a) Postmenopausal gingivostomatitis before treatment. (b) Postmenopausal gingivostomatitis showing changes in gingival epithelium. (c) Histopathological finding

medical history revealed no significant finding. According to her personal history, she entered her menopausal stage 5 years back. On intraoral examination, the gingiva appeared dry, shiny, erythematous and less keratinized [Figure 3a]. Patches of red and white lesions with areas of ulceration were seen on gingival and buccal mucosa [Figure 3b]. Bleeding on probing was present, and the plaque deposits were fair. Her upper anterior teeth were missing. A gingival swab was taken for a histopathological examination that showed atrophy of the germinal and prickle cell layers of the epithelium and areas of ulceration [Figure 3c]. Although her clinical finding was quite similar to desquamative gingivitis but correlating the histopathology, history, age and gender, and the postmenopausal stage where reduced circulating estrogen level might have affected the epithelial changes, the patient was diagnosed with postmenopausal gingivostomatitis with burning mouth syndrome. The patient was given phase I therapy where complete scaling was done and was motivated to maintain strict oral hygiene. Use of an extra soft tooth brush using “toe” or “heel” of the brush was recommended seeing the fragility of the mucosa. A corticosteroid gel was prescribed to relieve the symptoms. The patient was recalled after 1-month, but she failed to keep further appointments.

DISCUSSION

Pubertal gingivitis

Puberty is a complex process of sexual maturation, and it is responsible for changes in physical appearance and behavior that are related to increased levels of the sex steroid hormone estradiol in females. A peak prevalence of gingivitis has been determined at 12 years, 10 months in females^[4] which are characterized by the hyperplastic

reaction leading to gingival enlargement and onset of exuberant inflammation of the gingiva. This clinical presentation is believed to be related, at least in part, to an alteration in the subgingival microflora during this period. The bacterial counts increase in number, and there is a prevalence of certain bacterial species such as *Prevotella intermedia* (Pi) and *Capnocytophaga*. Pi has been shown to possess the ability to substitute estrogen and progesterone for menadione (Vitamin K) as an essential growth factor.^[5] This may explain the association between increased estrogen concentrations and the elevated counts of Pi. Together, estrogen affecting gingival vasculature and *Capnocytophaga* species which often increases during puberty, have been associated with the increased bleeding tendency observed during this period.^[6]

Pregnancy induced gingivitis and pregnancy tumor

Pregnancy results in significant endocrine alterations where both progesterone and estrogen are remarkably elevated. Pregnant women are more prone to inflammation and gingival bleeding. Pregnant women may also experience localized gingival enlargement resembling pyogenic granulomas. These lesions also known as pregnancy tumor have been described as a painless, exophytic mass that has either a sessile or pedunculated base extending from the gingival margin or, from the interproximal tissues in the maxillary anterior region predominantly.^[7] The pregnancy tumor bleeds easily and may range in color from purplish red to deep blue, although most commonly is red in color with small fibrin spot. Tooth mobility, pocket depth, and gingival fluid are also increased in pregnancy. The levels of sex steroid hormones in saliva increases during pregnancy resulting in alterations in the microbial populations which may contribute to these pathologic changes. Kornman and Loesche^[8] reported that the ratio of subgingival bacterial anaerobes-to-aerobes increased, as well as proportions of *Bacteroides melaninogenicus*, Pi and *Porphyromonas gingivalis*. Pi and *P. gingivalis*, can use female sex hormones as a source of nutrients. The immunologic changes might also be responsible for periodontal pathologic conditions observed during pregnancy.^[9] The increased synthesis of prostaglandin E2 observed when estradiol and progesterone are present in higher concentrations, occurs during pregnancy may also contribute to these pathologic changes.^[10] Effect of these hormones on microvasculature is responsible for marked hemorrhagic tendency during pregnancy.

Postmenopausal gingivostomatitis

The menopause triggers a wide range of changes in women's bodies, and the oral cavity is also affected. The absence of ovarian sex steroids mainly estrogen has been related

to a worsening in gingival health. An increase in gingivitis, periodontal disease, and tooth loss has been reported.^[11] Patients may also complain of dry mouth because of decreased salivary secretion as well as burning sensation of the mouth and tongue. Taste sensation may change causing frequent complaints of a metallic taste. Some women develop senile atrophic gingivitis in which an abnormal paleness of gingival tissue develops. Other people develop a condition known as gingivostomatitis which is characterized by gingivae that are dry and shiny, bleed easily and range in color from abnormally pale to erythematous. The signs and symptoms of postmenopausal gingivostomatitis are somewhat comparable to those of chronic desquamative gingivitis.^[12] Histopathology, antibody detection by direct and indirect immunofluorescence and the absence of other dermatological conditions may help in ruling out desquamative lesions. Studies have suggested that estrogen deficiency leads to an increase in the immune function. Low estrogen production after menopause is associated with increased production of Interleukin 1, 6, 8, 10, tumor necrotic factor α , granulocyte colony stimulating factor as estrogen inhibits the expression of inflammatory cytokines important in bone resorption and may contribute to more intense gingival inflammation during periodontitis and subsequent oral bone loss resulting in clinical attachment loss and tooth loss. Reduction in epithelial keratinization and collagen formation in connective tissue results in thinning of oral mucosa and difficulty with removal partial prosthesis.^[13]

Management

To reduce the incidence of hormone influenced periodontal diseases all local irritants should be removed, and meticulous plaque control be maintained. Patients should be educated regarding the profound effects of the sex hormone on periodontal tissues and oral tissues as well as the consistent need for home and office removal of local irritants. Milder form of gingivitis cases responds well to scaling and root planing. Severe cases, where enlargement occurs, requires surgical excision. For menopausal gingivitis hormone replacement therapy can be included. A number of studies have suggested that the symptoms of postmenopausal gingivitis and risk of postmenopausal tooth loss is reduced by estrogen replacement, but mostly patients fail to comply with the therapy because of the fear of cancer, irregular bleeding, and other minor side-effects.^[14] Given in low dosages, benzodiazepines, tricyclic antidepressant or anticonvulsants may be effective in patient with burning mouth syndrome.^[15]

CONCLUSION

Female sex hormones are implicated in the changes in periodontal conditions. The gingival inflammation is exacerbated during puberty, pregnancy and at the postmenopausal stage. The cases presented have shown how much a same hormone at different age and stage shows an exaggerated inflammatory response to the plaque. Strict oral hygiene maintenance is of prime importance for the patient because it is the dental plaque that leads to incidence and prevalence of disease while the level of hormone modifies the response.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Amar S, Chung KM. Influence of hormonal variation on the periodontium in women. *Periodontol* 2000 1994;6:79-87.
2. Lapp CA, Thomas ME, Lewis JB. Modulation by progesterone of interleukin-6 production by gingival fibroblasts. *J Periodontol* 1995;66:279-84.
3. Krejci CB, Bissada NF. Women's health issues and their relationship to periodontitis. *J Am Dent Assoc* 2002;133:323-9.
4. Sutcliffe P. A longitudinal study of gingivitis and puberty. *J Periodontol Res* 1972;7:52-8.
5. Kornman KS, Loesche WJ. Effects of estradiol and progesterone on *Bacteroides melaninogenicus* and *Bacteroides gingivalis*. *Infect Immun* 1982;35:256-63.
6. Gusberti FA, Mombelli A, Lang NP, Minder CE. Changes in subgingival microbiota during puberty. A 4-year longitudinal study. *J Clin Periodontol* 1990;17:685-92.
7. Sills ES, Zegarelli DJ, Hoschander MM, Strider WE. Clinical diagnosis and management of hormonally responsive oral pregnancy tumor (pyogenic granuloma). *J Reprod Med* 1996;41:467-70.
8. Kornman KS, Loesche WJ. The subgingival microbial flora during pregnancy. *J Periodontol Res* 1980;15:111-22.
9. Loe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontol Scand* 1963;21:533-51.
10. Offenbacher S, Odle BM, Van Dyke TE. The use of crevicular fluid prostaglandin E2 levels as a predictor of periodontal attachment loss. *J Periodontol Res* 1986;21:101-12.
11. Krall EA, Dawson-Hughes B, Hannan MT, Wilson PW, Kiel DP. Postmenopausal estrogen replacement and tooth retention. *Am J Med* 1997;102:536-42.
12. Klokkevold PR, Mealey BL, Carranza FA. Influence of systemic diseases and disorders on the periodontium. In: Newman MG, Takei HH, Carranza FA, editors. *Carranza's Clinical Periodontology*. 9th ed. Philadelphia, Pennsylvania: Saunders; 2003. p. 214.
13. Massler M. Oral manifestations during the female climacteric (the postmenopausal syndrome). *Oral Surg Oral Med Oral Pathol* 1951;4:1234-43.
14. Björn I, Bäckström T. Drug related negative side-effects is a common reason for poor compliance in hormone replacement therapy. *Maturitas* 1999;32:77-86.
15. Grushka M, Epstein JB, Gorsky M. Burning mouth syndrome. *Am Fam Physician* 2002;65:615-20.