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A CRITICAL INSIGHT INTO PERSISTENT GINGIVAL OVERGROWTH INDUCED BY PREGNANCY– A Rare Case Report

Periodontology

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ABSTRACT

Background: Gingival enlargement is a common clinical problem, finds a unique place in the literature associated with specific conditions. **Aim:** Our aim is to critically give an insight into a case reported with gingival enlargement initiated during first trimester but persisted even after normal child birth. **Methodology:** Diagnosis was made by history, signs and symptoms and with appropriate blood and hormonal investigations. After thorough phase 1 therapy, surgical excision of excessive residual tissue was carried out. **Result:** A complete resolution of local irritant and inflammation which resulted in a well contoured and aesthetically pleasing gingiva. **Conclusion:** Although a spontaneous reduction in the size of gingival enlargement commonly occurs following childbirth, complete elimination of residual inflammatory lesions requires the removal of all forms of local irritants followed by surgical approach if necessary

KEYWORDS

Pregnancy- induced, gingival enlargement, periodontal disease

INTRODUCTION:

Gingival enlargement is a common clinical entity but finds a unique place in the literature, because it has been associated with a variety of local and systemic factors so differential diagnosis becomes an important aspect for complete management of the lesion. Most of the causative factors lead to an unusual hyperplastic tissue response to chronic inflammation associated with local irritants such as plaque, calculus or bacteria.[1]

Hormonal changes can significantly potentiate the effects of local irritants on gingival connective tissue. In all forms of enlargements, good oral hygiene is necessary to minimize the effects of systemic factors; gingivoplasty or gingivectomy may be required, but should be done in combination with prophylaxis and oral hygiene instructions.[2]

Periodontal health in pregnant women has become a field of research since the 1960s, resulting in a flurry of studies to focus on it. Gingival inflammation associated with pregnancy has been initiated by dental plaque and exacerbated by endogenous steroid hormones.[3] Meanwhile, the bidirectional interaction between systemic conditions and periodontal status has been taken more seriously into consideration with the proposition of periodontal medicine since from the middle 1990s.[4]

Although it is mandatory to exclude the effects of previously existing periodontal inflammation and dental plaque in order to explore the sole effect of pregnancy on periodontal health,[5] works of research in this regard have rarely been performed. This case report summarizes the status of the changes of periodontium during pregnancy, especially the normal periodontium in order to elucidate the effect of pregnancy on the progress of gingival inflammation.

CASE REPORT:

A 36 year old female patient reported to Department of Periodontics, Mahatma Gandhi Post Graduate Institute of Dental sciences, Puducherry, with complaint of swelling of gums in her upper front teeth region since 3 years with bleeding on brushing and difficulty in chewing. Three years back she noticed bleeding while brushing, with mild swelling in the gums in her third month of pregnancy, which gradually increased in size till seventh month and it persisted even after normal child birth. She was also concerned about her aesthetics and bad breath. On intra oral examination revealed the presence of Grade II gingival enlargement (Figure-1) in relation to the maxillary anterior tooth region, with generalized bleeding on probing; probing depth of the maxillary anterior region was 6-9 mm with generalized probing depth of 3-5mm. The mobility was Grade I in 11 and 21; Grade II in 22. Plaque index was found to be poor on examination. The patient was systemically healthy and had not taken any medications known to provoke gingival enlargement. There was no relevant family history. Oral prophylaxis was performed after routine haematological investigations. There were no abnormalities in peripheral blood smear. In thyroid function test, levels of thyroxine (T4) and thyroid stimulating hormone (TSH) were found to be normal. After evaluation of Phase-I, a probing depth of 6-8 mm was found in maxillary anteriors (Figure - 2). After adequate local anesthesia, an internal bevel incision was given to remove excess fibrotic epithelial tissues and a full thickness mucoperiosteal flap was reflected in relation to maxillary anteriors. A thorough degranulation was done, haemostasis achieved, and the flap was sutured with 3-0 BBS using continuous sling suture (Figure-3). The patient reported after one week for suture removal and early healing was uneventful. The patient was re-instructed for maintenance of oral hygiene. Three months post operatively the gingival contour was normal and the patient was able to maintain her oral hygiene with no recurrence of growth on follow up (Figure-4).

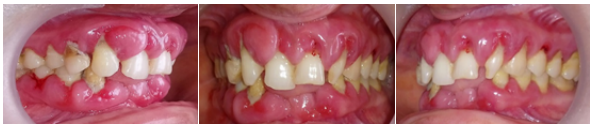

Figure -1: Pre – Operative images showing Grade II Gingival enlargement

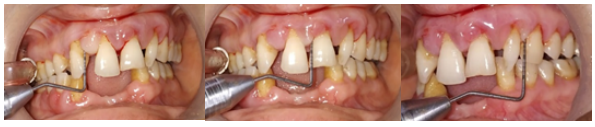

Figure -2: Photographs showing Probing depths of 6-8 mm after Phase 1

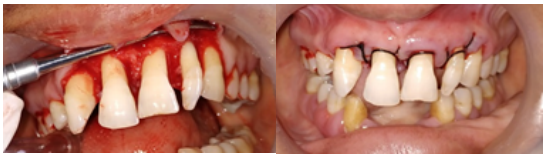

Figure -3: Photographs showing Full thickness flap reflection with degranulation done and Continuous sling suture

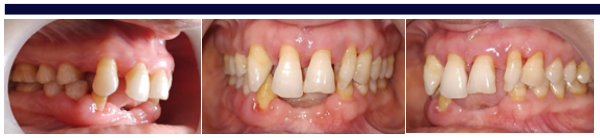

Figure -4: Photographs showing Post - Operative view (3 Months)

DISCUSSION:

Pregnancy is a physiological state that brings metabolic and immunological changes by an increase in secretion of sex hormones like Progesterone and Estrogen. The correlation between gingivitis and the quantity of plaque was greater after parturition than during pregnancy, which suggests that pregnancy induces other factors that aggravate gingival response to local irritants.[2]

During the second trimester of pregnancy, the progesterone and estrogen levels are increased by 10 and 30 times respectively than that of during menstrual cycle.[6] Estrogen is the main sex hormone responsible for stimulating the endometrial blood flow. On contrast, in gingiva and other non-periodontal intraoral tissues, more evidence has accumulated for progesterone affecting the local vasculature than for estrogen. Progesterone has been shown to reduce corpuscular flow rate, allowing for accumulation of inflammatory cells, increased vascular permeability and proliferation.[7]

Sex steroid hormones have also been shown to increase the rate of folate metabolism in oral mucosa. Since folate is required for tissue maintenance, increased metabolism can deplete folate stores and inhibit tissue repair.[8]

Several studies showed the presence of estrogen receptors on various immune cells as well as the presence of androgen receptors on T and B lymphocytes. Progesterone has been shown to stimulate the production of the inflammatory mediator prostaglandin E2, enhance the accumulation and chemotaxis of neutrophils, while low concentrations of estradiol have been demonstrated to reduce neutrophil chemotaxis.[9]

Human PDL cells possessed immunoreactivity towards estrogen receptors, specifically estrogenic effects in PDL cells are mediated via estrogen receptors beta (ER beta).[7] During the second trimester, alteration in subgingival microflora occurs, of which an increase in presence of Prevotella intermedia, which can substitute estrogen and progesterone for Vitamin K, an essential bacterial growth factor.[10]

Culture supernatants of these micro-organisms have been shown to synthesize steroid metabolizing enzymes and enhance the expression of 5α-reductase activity in human gingiva and in cultured gingival fibroblasts, resulting in the formation of 5α - dihydrotestosterone (DHT) from androgen substrate. The DHT can influence protein synthetic activity in these pathogens, for which there is a variety of applications. Some of these functions are: a) the formation of surface capsular protein contributing to their evasion of host elimination mechanisms, such as phagocytosis, by preventing opsonization b) persistence and dissemination with the host and c) interspecies aggregation and energy generation as a result of the electron transfers involved in these enzyme activities.[7]

Our case has occurred in first trimester, on the maxillary anterior teeth region. It usually regresses after pregnancy but in our case it persisted for three years. It was also noted that there is increased subgingival deposits with less supragingival deposits. In the present case, there was persistent hyperplastic gingival tissue which was interfering with the patient's ability to maintain proper oral hygiene and was causing serious aesthetic problems even after thorough phase I therapy, so surgical reduction of excessive fibrotic tissue was carried out to obtain a proper gingival contour that facilitates good oral maintenance by the patient.

CONCLUSION:

Pregnancy does not cause the condition, but altered tissue metabolism in pregnancy due to hormonal imbalance, accentuates the response to local irritants i.e., plaque and calculus aggravates hyperplasia. Since there are a large number of diseases, medications, and physiologic states reported in association with gingival overgrowth, the diagnostic work-up must be pursued in a logical, step-wise approach.

In all forms of gingival enlargements, good oral hygiene is necessary to minimize the effects of systemic factors. Although a spontaneous reduction in the size of gingival enlargement commonly occurs following childbirth, complete elimination of residual inflammatory lesions requires the removal of all forms of local irritants followed by surgical approach if necessary.

CONFLICT OF INTERESTS:

There is no conflict of interests to declare.

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