

Unusual case of postpartum gingival enlargement: diagnosis and management

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Abstract

Gingival enlargement is a clinical condition that has been widely studied. Usually, it is related to specific local or systemic factors. However, it is difficult sometimes to find out a definite etiology, and treatment has to be done according to presenting clinical features. This article presents an unusual case of gingival enlargement that occurred after pregnancy without any clear underlying etiology. A female aged 31 years reported with gingival enlargement and mobility of teeth during lactation period. Gingival enlargement had started 2 months after child birth. All female sex hormones were found to be within normal limits. Karyotyping was also found to be normal, without any genetic alteration. Radiographic analysis revealed generalized severe crestal bone loss. After phase I periodontal therapy, enlargement was managed surgically under local anaesthesia. On histological examination of excised specimen, tissue was found to be hyperplastic. Although a definite etiology could not be ascertained, the treatment was successful and there has been no recurrence after one year post-treatment interval.

Keywords: Gingival Enlargement; Postpartum; Gingivectomy; Pregnancy; Hyperplasia

1. Introduction

Hormones exert significant influence in body physiology throughout life. Women in particular, experience variations in levels of different hormones under physiological conditions, including menstrual cycle, pregnancy, as well as in hormonal therapy (use of oral contraceptives). This variation significantly affects women's health, including the oral cavity. Hormonal changes occurring during pregnancy and puberty have been known to be associated with varying patterns of gingival enlargement as they potentiate the gingival inflammation in response to local irritants on tooth surface. A number of studies have demonstrated that the hormonal influence on the immune system contributes significantly to the etiology and pathogenesis of pregnancy gingivitis (Amar & Chung, 1994). Kornman & Loesche (1980) showed that during pregnancy there are increased levels of progesterone and oestrogen with increased proportions of *P. intermedia* among periodontal microbiota. In addition, O'Neil (1979) demonstrated that increased level of progesterone causes an increase in vascular permeability, number of polymorphonuclear leukocytes and levels of prostaglandin E₂ in the gingival sulcus. Willerhausen et al. (1991) showed that progesterone concentrations corresponding to those seen in the 3rd trimester of pregnancy, caused decreased synthesis of all glycosaminoglycans by human gingival fibroblasts, contributing to the inflammatory changes observed in the gingiva. Although a significant proportion of pregnant women suffer from pregnancy gingivitis, this condition is both self-limiting and transient. Gingival tissues return to their original healthy state postpartum when oestrogen and progesterone levels reach baseline values (Amar & Chung 1994).

However, when oestrogen and progesterone levels continue to remain high after pregnancy, then gingival overgrowth persists. This is unusual, and even more unusual is the occurrence of gingival overgrowth two to three months postpartum. This article presents a case of gingival enlargement that occurred during lactation, 2 months after childbirth.

2. Case report

A 31 years old female reported with generalized overgrowth of gingiva since last one month. Patient had delivered a healthy child three months back. It was a full term pregnancy and child birth was free of any complications. She reported to have experienced bleeding on brushing teeth during pregnancy. The gingival enlargement was noted two months postpartum and had gradually grown in size. The overgrowth was causing functional and esthetic problems for the patient. The patient had a known history of hypothyroidism and was taking medication for it since last 5 years. She had received routine medical evaluations during pregnancy and never taken any medication known to induce gingival overgrowth. There was no other relevant dental and medical history.

Intraoral examination revealed that the enlarged gingival tissue covered the entire crown like an apron and it was retractable. The severity of gingival enlargement on both the arches corresponded with a grade IV of Eva Ingle's classification of gingival overgrowth index (Ingle et al. 1999). The gingiva was non-pigmented, bulbous, soft and boggy in consistency (Figures 1-3).

Hard tissue examination revealed #17, #26, #27, #37, #47 missing teeth and grade III mobility in #16. The gingiva was probed at 4 sites (mesiobuccal, buccal, distobuccal and lingual/palatal) around

each tooth. Total 92 sites were probed out of which 76 sites were found with probing depth in the range of 8-12 mm. Panoramic radiograph showed generalized aggressive pattern of bone loss. There were no significant calcified accretions (Figure 4). The complete blood count with differential and comprehensive metabolic panel was normal. Genetic karyotyping done to rule out any type of genetic defect failed to reveal any relevant findings (Figure 5).



Fig. 1: Gingival Enlargement 2 Months after Pregnancy (Frontal View).



Fig. 2: Gingival Enlargement 2 Months after Pregnancy (Left Lateral View).



Fig 3: Gingival Enlargement 2 Months after Pregnancy (Right Lateral View).

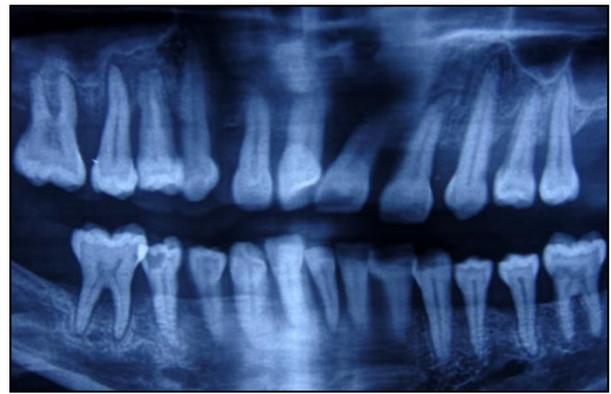


Fig. 4: Panoramic Radiograph Showing Severe Bone Loss.

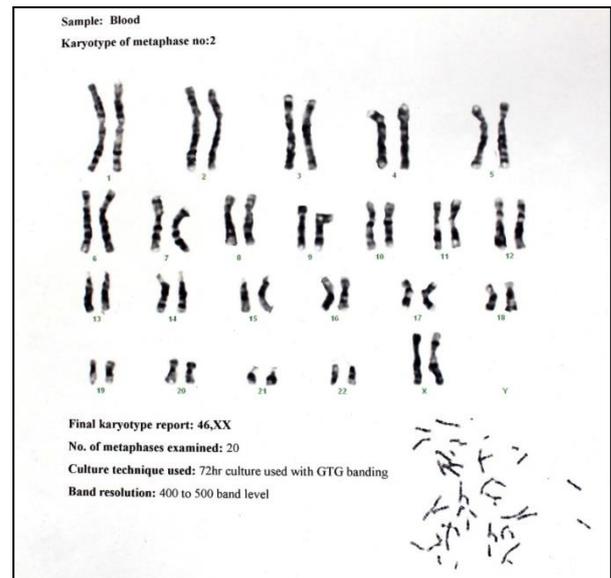


Fig. 5: Normal Karyotype of the Patient.

Treatment included patient education, motivation and oral hygiene instructions, followed by mechanical debridement (scaling and root planing) and periodontal surgery (internal bevel gingivectomy). Eight weeks interval was maintained between the non-surgical and surgical phases. In the meantime patient was placed on chlorhexidine (0.2%) mouth rinse to help reduce bacterial plaque and gingival inflammation. There was no improvement in gingival enlargement after non-surgical phase, but the gingiva appeared healthier as inflammation had reduced significantly.



Fig. 6: Mandibular Arch Immediately after Gingivectomy.



Fig. 7: Excised Tissue.



Fig. 8: Frontal View 1 Month after Gingivectomy.

Gingivectomy was performed on all the teeth (Figure 6). The gingival tissues were collected and submitted for histologic analysis (Figure 7). Ibuprofen (400 mg) twice daily was prescribed for 5 days. The patient was instructed to continue using the chlorhexidine mouth-rinse for 2 weeks after surgery. The post-surgical phase was uneventful in both arches.

Histologic examination revealed tissue covered with stratified squamous epithelium. The epithelium was mildly acanthotic with thick rete ridges. The submucosa consisted of highly collagenous fibrous connective tissue with focally dense infiltrate of mature lymphocytes and plasma cells. Scattered neutrophils were also noted. The histopathologic features were most consistent with the diagnosis of hyperplastic gingivitis.

Only few of the total sites bled upon probing at the post-surgical evaluation after 1 month. Overall, the gingival tissues appeared clinically healthy (Figure 8). Patient was instructed to maintain good oral hygiene. She was placed on a 3-months periodontal recall program and was followed up to 1 year after treatment. The mobility of teeth had considerably reduced. After 1 year of follow up, no recurrence of the gingival overgrowth was observed and patient was satisfied with the aesthetic outcome of treatment.

3. Discussion

The main etiologic factors for gingival enlargement are local irritation from dental plaque, selective drug therapy (Newman et al. 2012, Ahad et al. 2016) and familial or genetic conditions (Ramer et al. 1996). However, in the present case, the medical and dental history did not ascertain a direct cause. Since plaque control was fair, it is unlikely that this much enlargement and bone destruction resulted from chronic and untreated periodontal inflammation. As the enlargement occurred after pregnancy, the diagnosis of hereditary gingival hyperplasia had been ruled out. Hereditary gingival

hyperplasia is a genetic disorder which usually manifests much earlier in life and lesion appears more firm and fibrotic clinically. Patient was taking thyroxine since 5 years but there is no evidence in literature to prove that thyroxine contributes to gingival enlargement and also drug induced gingival enlargements usually occur in first three months after starting medication (Meraw & Sheridan, 1998).

In the present case, histopathologic features were most consistent with hyperplastic gingivitis. The histologic differential diagnosis for generalized gingival enlargement includes generalized chronic periodontitis, drug-induced gingival enlargement, granulomatous gingivitis, leukaemia, and hereditary gingival fibromatosis. Gingival fibromatosis has hyperplastic epithelium with long, thin rete ridges and submucosa of densely collagenized fibrous connective tissue. Scattered inflammatory cells may be seen occasionally but are usually absent (Bozzo et al. 2000). Drug induced gingival enlargement also consists of hyperplastic epithelium with highly collagenous fibrous connective tissue (Newman et al. 2012, Ahad et al. 2016). A chronic inflammatory cell infiltrate may be seen if secondary inflammation is present (Newman et al. 2012). Gingival hyperplasia as a result of leukemic infiltrate is an unusual situation. The submucosa shows destruction of the normal host tissue by a sheet of poorly differentiated cells. Diagnosis of ligneous periodontitis had been ruled out as there was absence of amyloid on histopathological examination.

Hormonal changes during pregnancy and puberty are known to aggravate gingivitis and gingival enlargement. Pregnancy accentuates the gingival response to plaque. Incidence of gingivitis in pregnancy varies from 50% to 100% (Amar & Chung, 1994). The recent history of pregnancy in our case offers pregnancy gingivitis a possible explanation for the hyperplastic gingiva. The peculiarity in the gingival enlargement in the case reported here is the time of onset, which was during lactation. The oestrogen and progesterone levels were normal at this time. The literature does not report any similar association postpartum or with lactation. In the present case, gingival enlargement also reported rapid increase in size and presented with underlying periodontal destruction. However, elevated hormonal levels characteristic of pregnancy usually do not affect periodontal attachment. Vittek et al. (1979) and Staffolani et al. (1989) reported that elevated levels of these hormones during a nine month period was insufficient to cause significant periodontal break-down, despite their reported effects on the epithelial barrier, vasculature and connective tissue matrix. Therefore it might be the case that patient was unaware of any periodontal problem, and periodontal destruction had started earlier and suddenly flared up postpartum, along with the occurrence of enlargement.

Although the true etiology of gingival enlargement could not be ascertained in this case, treatment based on presenting clinical features resulted in successful and aesthetically acceptable results. Completion of phase I therapy before surgery was crucial in reducing inflammation prior to surgical treatment.

4. Conclusion

In the present case of gingival enlargement, etiology remained a mystery as it had started after pregnancy when the hormonal levels were normal. Although combination of non-surgical and surgical therapy resulted in successful outcome, the etiopathogenesis of postpartum gingival enlargement needs to be explored so as preventive measures could be taken in susceptible patients.

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