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Let us forget about the doom and gloom the best is yet to come - case report of idiopathic gingival fibromatosis.

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Abstract

Gingival enlargement is defined as the ballooning out of gingiva over the teeth; it may be localized or generalized. The etiology can be multifarious - due to the influence of bacterial plaque, systemic diseases or conditions, genetic predisposition (hereditary or familial), drugs or of unknown origin. When the etiology is unknown, it is considered as idiopathic gingival enlargement. The patient may end up in psychosocial humiliation or even mistreated by friends or relatives due to ghastly appearance of the overgrown gingiva. Here, we report a case of gingival enlargement in a 8-year-old female. The maxillary posterior teeth were scarcely discernible as the enlargement was enormous. The case was diagnosed as idiopathic gingival enlargement based on history, clinical and histolopathological findings. Conventional gingivectomy was performed. The patient was on the maintenance phase, and it proved effective as no recurrence was noted. Surgical correction, coupled with consistent oral hygiene practices, notably ameliorated the disease condition. This not only enhanced aesthetic appearance but also fostered better social acceptance for the patient.

Keywords: Gingival Fibromatosis, Idiopathic Overgrowth, Gingivectomy, Enlargement, Genetic.

Introduction

Idiopathic gingival fibromatosis is a relatively rare genetic heterogeneous conditions characterized by recurring gingival enlargement of unknown origin, which may have autosomal dominant or recessive traits of inheritance. When an inherited genetic predisposition has been recognized, it is referred as "hereditary gingival fibromatosis".[1] It typically manifests as either an isolated disorder or as part of a multisystem condition. It is a slowly progressive, benign, painless, localized or generalized overgrowth of the keratinized gingiva.[2] Clinically, the gingival tissue is pink, non-hemorrhagic with a firm and fibrotic consistency. This kind of overgrowth can potentially cause malocclusion due to prolonged retention of primary dentition, delayed eruption of permanent dentition resulting in esthetic and functional handicaps.[3] Usually, the onset coincides with the eruption of permanent dentition, nevertheless it may be associated with deciduous dentition as well.

Case Report

A 8-year-old female patient reported to the Department of Periodontics, with the complaint of swelling of gums of the left upper side of the mouth for 2 months, and the patient gave a history of rapid increase in the size of swelling. The patient noticed gradually increasing swelling that was painless, but interfered with chewing. The patient had delayed eruption of permanent teeth. Developmental milestones and other systems of child were within normal parameters and non-contributory to the condition. General evaluation revealed normal physical appearance and psychomotor skills. Extra-oral features were normal and the lips were competent. The patient had unilateral mastication habit. Intra-oral findings revealed the left maxillary and mandibular posterior teeth were completely involved covering almost the entire clinical crown [Figures 1 and 2]. The swelling was irregular, pale pink, firm and fibrotic in consistency, devoid of stippling with diffuse involvement of marginal, papillary and attached gingiva. Generalized bleeding on probing was present. 64, 65, 26, 73, 74, 75 teeth were clinically submerged. Deep pseudopockets along with attachment loss of upto 4mm were present in 64, 65 and 26 region.

Investigation

Panoramic radiograph revealed the presence of succedaneous permanent teeth 23, 24, and 25 with the root resorption of primary teeth 63, 64 and 65. Complete hemogram revealed that normal blood profile with the exception of reduced hemoglobin level (8 g/dl). Histopathological investigation of the biopsy tissue demonstrated dense bundles of collagen fibers along with intervening aggregation of chronic inflammatory cells and compressed blood vessels [Figure 3]. The overlying epithelium was hyperplastic, parakeratinized, stratified squamous with long and thin rete ridges.

Treatment

After obtaining consent from the parents, phase I therapy was carried out. Meticulous oral hygiene instructions were advised. The patient was recalled after 2 weeks for reevaluation of oral growth. Two weeks later, the surgery was performed quadrant wise under local anesthesia [Figure 4]. The procedure involved excision of gingiva using external bevel gingivectomy technique, followed by the application of periodontal dressing. The patient was prescribed with antibiotics and anti-inflammatory drugs for a three-day period. Additionally, the use of 0.2% chlorhexidine mouthwash was advised. After one week, the patient reported for the removal of the periodontal dressing. Subsequently, the patient was scheduled for recall therapy, initially every three months

[Figure 5 and 6], and later yearly follow-up appointments. The patient appeared with satisfactory functionality and esthetics of oral cavity.

Discussion

The congenital hypertrophy of the gum is also known as hereditary gingival enlargement, gingival elephantiasis, gingival gigantism, symmetrical fibroma of the palate, gingival hyperplasia, gingival overgrowth and congenital macrogingiva.[4] The pronounced swelling resulting from extensive gingival enlargement leads to functional challenges and an unappealing aesthetics. Involvement of attached gingiva and associated teeth disappearance occurs in post-extraction period.[5] Attachment loss and bone loss occurs with poor oral hygiene, secondary to inflammation. Some cases are also associated with aggressive periodontitis.[6] Before diagnosing the condition as idiopathic, it is crucial to eliminate other possible causes of gingival enlargement.[7] Clinical presentation of this condition differs due to a variety of genetic mutation – autosomal dominant or autosomal recessive inheritance or a sporadic type of genetic mutation.[8] So far, loci for isolated hereditary growth factor have been identified on chromosome 2 (GINGF on 2p21-22 and GINGF3 on 2p22.3-p23.3), chromosome 5 (GINGF2 on 5q13-5q22) and chromosome 11, (GINGF4 on 11p15).[9]

Excess fibroblast proliferation or disturbed collagen turnover resulted in an excessive collagen fiber of connective tissue.[10] The collagen excess can be due to deficiency of growth factors and increased cross linking. Higher expression of prolyl 4- hydroxylase, increased exogenous or autocrine production of transforming growth factor – beta (TGF- β) and interleukin-6 (IL-6) production increases the synthesis and reduces the proteolytic activities of fibroblasts, may favour accumulation of the extracellular matrix. In individuals with Son of Sevenless – 1 (SOS-1) gene mutation, the Ras- MAPK pathway may be affected; activation of this pathway increases the expression of type 4 collagen, along with connective tissue growth factor (CTGF), TGF-β, and decreases the expression of matrix metalloproteinases (MMP) that degrades the extracellular matrix components.[5]

Treatment depends on the severity of the enlargement, minimal gingival enlargement may be treated with phase - 1 therapy, whereas gingival enlargement interfering with occlusion may require phase – 2 therapy. Furthermore, the effects of unilateral mastication such as compromised facial growth and development, potential for malocclusion and dento-facial deformities, and periodontal problems must not be overlooked.

Conclusion

Idiopathic gingival enlargement is an enlargement of unknown etiology, typically of genetic predisposition. The rapidity of enlargement is very impending to the normal functions and aesthetics of the patient. Although the recurrence cannot be predicted, long-term follow-up is mandatory for maintaining form and function. A stringent maintenance of good oral hygiene may improve the disease condition.

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Legend Figures

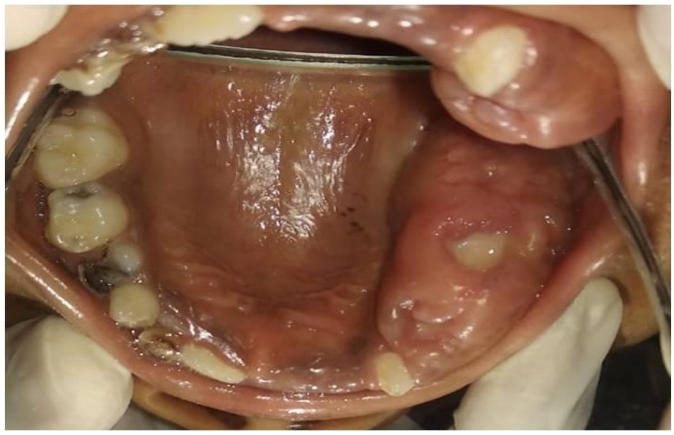

Figure 1: Pre-operative maxillary occlusal view

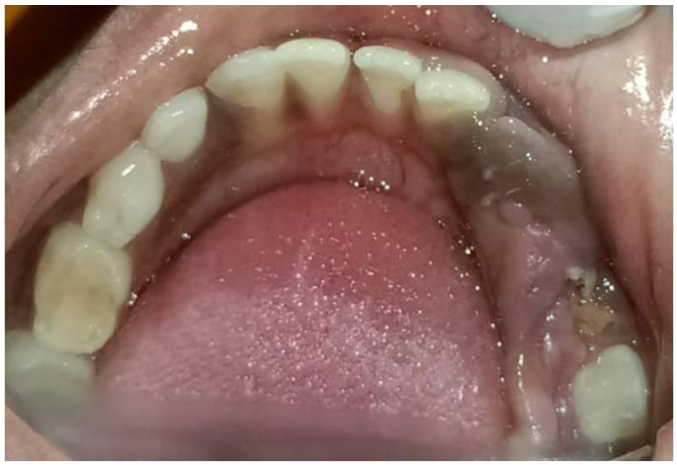

Figure 2: Pre-operative mandibular occlusal view

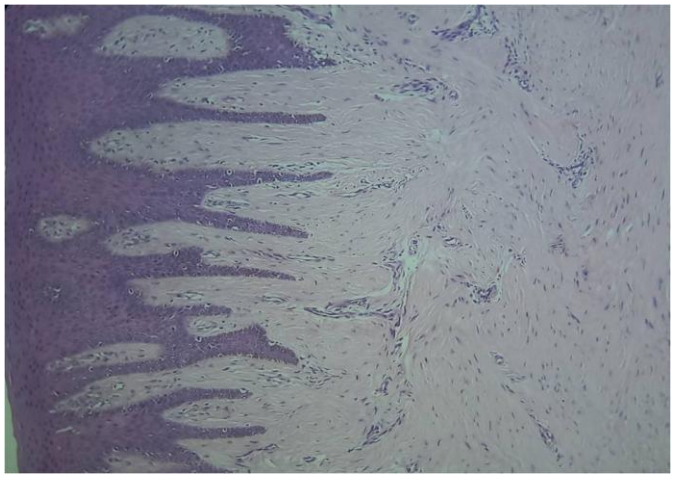

Figure 3: Histopathological picture showing epithelial and connective tissue hyperplasia with dense fibrosis and inflammatory cells

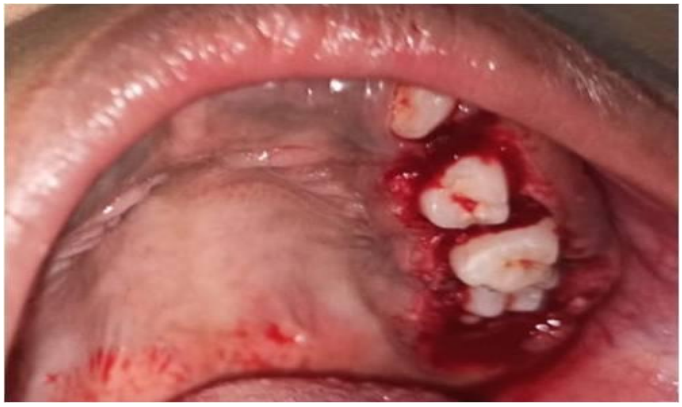

Figure 4: Intraoperative view

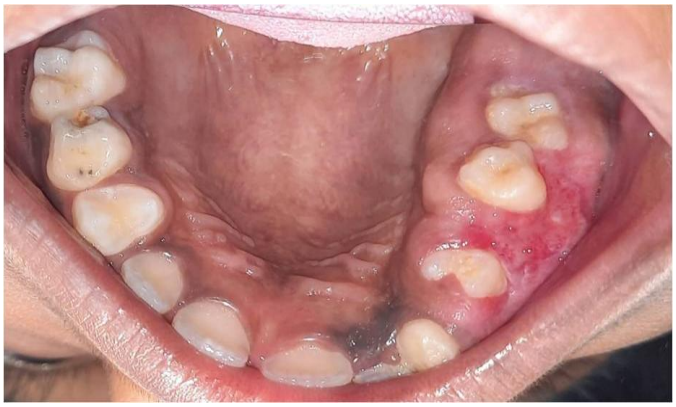

Figure 5: Post-operative (3 months) maxillary occlusal view

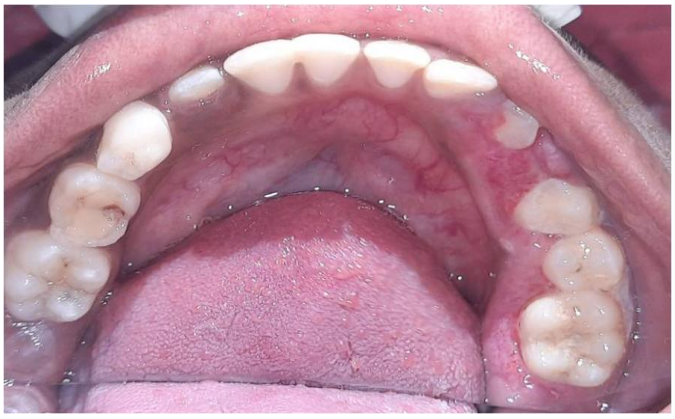

Figure 6: Post-operative (3 months) mandibular occlusal view