

Unilateral Gingival Enlargement - An Unusual Case Report

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Abstract: *An unusual case of a 9 years old female patient presented to the department of Paedodontics in National Ribat University/ Khartoum-Sudan, with chief complaint of moderate, unilateral gingival enlargement confined to the right side of oral cavity. The inflamed edematous enlarged gingiva covered more than 2/3 of the crown surfaces. The diagnosis of unusual non-specific gingival hyperplasia was made, based on clinical and histological features. Surgical excision and conventional periodontal therapy reduced the size of enlargement. This enlargement was considered due to exaggerated gingival response to local irritation from plaque and calculus, leading to overgrowth of periodontopathic bacteria. A careful recording of the history and results of clinical examination and investigations were noted to achieve diagnosis.*

Keywords: Gingival hyperplasia, Unilateral

1. Introduction

Gingiva is part of oral mucosa which surrounds the neck of the teeth like a collar. Healthy gingiva is important for the proper functioning of oral cavity and esthetics. Gingival enlargement or hyperplasia is one of the gingival diseases in which there is increase in the cellular matrix. The overgrowth might be caused by several etiological factors, such as the administration of specific drugs (e.g., cyclosporin, nifedipine, phenytoin), the autosomal-dominant or the autosomal-recessive inheritance as an isolated feature, and/or as a syndromic manifestation (Poulopoulos A, Kittas D, Sarigelou, 2011).

The enlargement of the gingiva may be localized to one papilla or may involve several or all of the gingival papillae throughout the mouth. Gingival enlargements are quite common and may be either inflammatory, non-inflammatory or a combination of both (R. Rajendran and B. Shivpathasundharam, 2007).

Inflammatory enlargement of gingiva usually results from prolonged chronic inflammation of the gingival tissue. In most cases, the enlargement results because of local irritations such as poor oral hygiene, accumulation of dental calculus or mouth breathing and represents a variation in host tissue response to dental plaque accumulation. Gingival enlargement may cause discomfort, interfere with speech or chewing, result in halitosis and it may look unsightly (Newman, Takei and Klokkevold, 2006 and Pandit I.K. and Pandit N, 2000).

The enlargement may be associated with one or more teeth, involve one or more quadrant(s), or may be generalized. The lingual or palatal gingivae are typically increased in thickness when compared to the buccal side. The etiology is thought to be familial or idiopathic. The familial variation may occur as an isolated finding or be associated to one of several hereditary syndromes e.g., Zimmermann-Laband,

Murray-Puretic-Drescher, Rutherford, multiple hematomas, and Cross syndrome. The exact cause of this abnormality is unknown, but it is thought to be an autosomal dominant pattern of inheritance in most cases. However, autosomal recessive examples have also been noted (Sandhu SP, Kakar V, Gogia G, and Narula SC., 2009).

2. Case Report

A 9 year-old girl presented to the Department of Paedodontics with the chief complaint of swelling in the gums around the right upper and lower back teeth region and inability to chew food from right side. History revealed that the swelling present six months with no associated symptoms and gradually increased to its present size. The patient came for treatment only when the swelling started interfering with mastication. Patient gave history of unilateral mastication. There was family history of consanguineous marriage.

No relevant medical history of intake of drugs, nutritional deficiency or a family history indicating any underlying genetic mechanism was recorded. General physical examination and extraoral examination was done. The extraoral findings revealed enlarged gingiva. The overlying skin appeared to be normal. Right submandibular lymphadenopathy was noticed and there was absence of abnormalities in extraoral examination.

On intraoral examination, gingival hyperplasia was seen from mesial of first premolar to distal of first molar on the right side, both in maxillary and mandibular arch. The right side of the oral cavity was unaffected. The enlargement seemed to be progressing around the first molars. The enlargement involved the marginal, attached and the interdental gingiva. Both facial and lingual/palatal gingiva was affected. The enlarged gingiva was smooth but firm, exhibited no change in the color /altered surface characteristics and extended on to the occlusal surface of

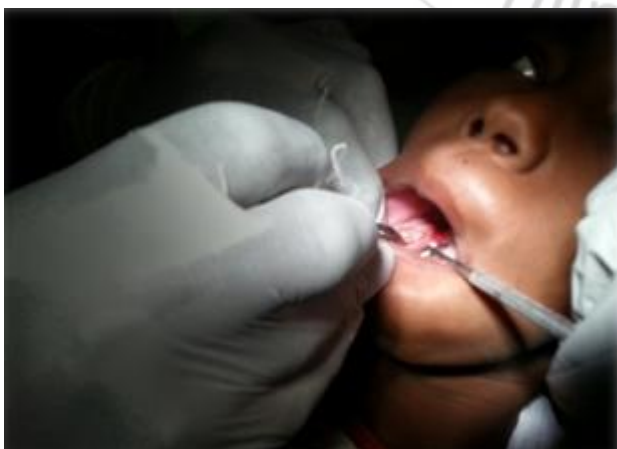
maxillary and mandibular first molars with bleeding and no significant pain for the patient and probing revealed little subgingival calculus. The jaws appeared distorted because of the bulbous enlargement of the gingiva.

Routine hematological investigations revealed hemoglobin count of 11gm % and a differential leukocyte count of polymorphonuclear leukocytes (neutrophils) 61%, lymphocytes 32%, monocytes 5% eosinophils 2%, and a random blood sugar level of 98 mg/dl. Assessment of sex hormones does not reveal any abnormality. Radiographic examination (OPG and IOPA X-rays) revealed no gross bone loss.



After assessing the complete status of the patient gingivectomy was done and tissue sample was sent for histopathological examination. Histopathological examination showed hyperkeratotic stratified squamous epithelium and underlying tissue infiltrated with mixed inflammatory cells associated with fibrosis.

Healing was uneventful; sutures were removed after one week. Based on the investigatory results, final diagnosis acute on chronic non specific inflammation. Gingivectomy of maxillary and mandibular arches has been done and healing was uneventful. Patient is under periodic follow up and there were no signs of recurrence and she was advised to maintain a scrupulous oral hygiene and was kept under regular observation.



3. Discussion

Gingival hyperplasia characterized by slowly progressive, non-hemorrhagic, fibrous enlargement of maxillary and mandibular gingiva. Clinically, the onset is consistent with the eruption of permanent dentition. Overgrowth can be observed varying in extent and severity. The excess gingival tissue may cover partial or whole crown, resulting in diastemas, teeth displacement, retention of primary teeth, or impacted teeth. The hyperplastic gingiva is usually normal in color, with firm consistency and heavy stippling (Long He and Fei-Yun Ping, 2012).

Gingival enlargement is usually caused by local conditions such as poor oral hygiene, food impaction, or mouth breathing. Systemic conditions such as hormonal changes, drug, or tumor infiltrates may complicate the process or even set the stage for the development of unfavorable local conditions that lead to food impaction and difficulty with oral hygiene. When edema, vascular engorgement, and

inflammatory cell infiltration predominate, gingival enlargement is referred to as inflammatory gingival hyperplasia. When the enlarged gingivae consist largely of dense fibrous tissue as a consequence of chronic inflammation or other causes, the condition is referred to as fibrotic gingival hyperplasia. The term "chronic hyperplastic gingivitis" is often used for either process (R. Rajendran and B. Shivpathasundharam, 2007 and Greenberg and Glick, 2005)

The involved tissues are glossy, smooth, and edematous and bleed readily. A fetid odor may result from the decomposition of food debris and from the accumulation of bacteria in these inaccessible areas. Loss of interseptal bone and drifting of the teeth occur in long standing cases of inflammatory enlargement. These changes are commonly referred to as gingivitis or periodontal disease when the process involves the loss of gingival attachment and the subsequent loss of interproximal bone (Greenberg and Glick, 2005). Gingival hyperplasia can be hereditary condition and investigations are in evolution to establish the genetic linkage associated with it (Hart TC, Pallos D, Bozzo L, Almeida OP, Marazita ML, O'Connell JR, et al., 2000).

The autosomal dominant form is often associated with hypertrichosis, corneal dystrophy, nail defects, deafness and craniofacial deformities whereas in the autosomal-recessive form with facial anomalies, hypertelorism have been observed but most forms are without defects, other than gingival enlargement. Consanguinity has been in the recessive form. Clinical abnormalities most commonly with gingival enlargement are hirsutism, epilepsy, oligodontia, mental retardation, nystagmus, strabismus, cataract, soft tissue tumors and enlarged facial bones (Sharma S, Goyal D, Shah G, Ray A. 2012).

In the present case, initially its not appeared to be associated with one of the syndromes. The relevant fact for such assumptions was consanguineous marriage of parents. the exact causative factor could not be identified. However, the plaque and calculus deposited on tooth surface could be the cause of chronic irritation of gingival tissues resulting in its proliferation. The etiologic agent for chronic inflammatory gingival enlargement is prolonged exposure to dental plaque, calculus and the factors that favor plaque accumulation and retention include poor oral hygiene, anatomic abnormalities and improper restorations. Clinically the involved gingiva appeared to be firm without spontaneous bleeding. The chronic nature of the lesion made the gingiva fibrotic. With the history of six months duration the enlargement was considered to be an exaggerated response of gingiva to local irritation complicated by periodontal involvement. Histologically, the gingival hyperplasia is mainly due to an increase and thickening of mature **collagen bundles** in the connective tissue stroma. Microscopic appearance of fibro-epithelial hyperplasia with inflammatory cell component in the connective tissue is suggestive of non-specific gingival enlargement. Such deformities in gingiva interfere with normal food excursion, collect food debris and plaque comprising of periodontopathic bacteria which is believed to prolong and aggravate the disease process resulting in bone loss and root resorption (Pandit I.K. and Pandit N, 2000).

The most prevalent types of gingival overgrowth in children are drug-induced gingival overgrowth, hereditary gingival fibromatosis (HGF), and neurofibromatosis I (von Recklinghausen disease) (Aikaterini Doufexi et.al, 2005). The constant increase in the tissue mass can result in delayed eruption and displacement of teeth, arch deformity, spacing and migration of teeth (McDonald R.E. and Avery D.R., 2000).

4. Conclusion

The treatment of gingival enlargement depends on the clinical, radiographic and histopathologic assessment supported by hematologic and hormonal investigations. Surgical excision with maintenance of proper oral hygiene rarely leads to the recurrence of the condition, So in cases of gingival enlargement, case history and investigations are mandatory to identify the underlying causes or associated syndromes. In the present case no such findings were noticed.

The above mentioned findings can only be coincidental findings or can be unidentified syndrome till now. This aspect requires further study to confirm the same. Even the sex hormones were in the normal ranges which excludes the chances of pubertal gingival enlargement. By the above information the reason for the enlargement was not clear but after the Gingivectomy patient is comfortable with no signs of recurrence.

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