UNILATERAL GINGIVAL ENLARGEMENT - A CASE REPORT
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Abstract
A 14 year old female patient suffered from unusual unilateral gingival enlargement on left side along with localized aggressive periodontitis. The enlarged gingiva covered more than 2/3 of the crown surfaces. Marked inflammatory edematous tissue with predominance of lymphocytes was observed histologically. The diagnosis of unusual nonspecific gingival enlargement along with localized aggressive periodontitis 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 and alveolar bone loss.

Key words: Gingival enlargement, Localized aggressive periodontitis

Introduction
The gingival tissues in the healthy mouth almost completely fill the interproximal spaces between teeth, beginning near the contact area and extending apically and laterally in a smooth curve. However, there is frequently an increase in the size of the gingiva so that soft tissue overfills the inter-proximal spaces, balloons out over the teeth and protrudes into the oral cavity. 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.[1]
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.[2,3] Inflammatory enlargement could be localized or generalized. Unilateral involvement of gingiva is rarely noticed in patients with inflammatory hyperplasia.

Case report
A 14 year old female patient reported in the dept. of oral & maxillofacial pathology with a complaint of unilateral swelling of gingiva since one year. The patient gave history of gradual enlargement of the swelling which was initially red and spongy with spontaneous bleeding. She stopped chewing food from the affected side because of discomfort, pain and bleeding. On general examination the patient was slightly under built for her age. No relevant medical history of intake of drugs, nutritional deficiency or a family history indicating any underlying genetic mechanism was recorded.
On clinical examination the extraoral findings revealed incompetent lips and the enlarged gingiva could be palpated over left cheek. The overlying skin appeared to be normal. Left submandibular lymphadenopathy was noticed. Intraoral examination showed a diffuse, nodular enlargement of gingiva extending from lateral
incisor to second molars in both arches on left side. The teeth were almost completely covered and displaced. The color of the gingiva appeared to be normal. It was firm and nontender on palpation. Slight bleeding on provocation was noted. Calculus deposits were seen on exposed tooth surfaces. (Fig.1&2)

Radiographically crestal bone loss with displacement of teeth could be appreciated. From this clinical and radiographic examination a provisional diagnosis of inflammatory gingival enlargement was made and the patient was subjected to a thorough medical examination. Patient was found to be anemic (Hb 8 gm %) and hormonal investigations were normal. The medical and family history was non-contributory.

After assessing the complete status of the patient, excision of the enlarged gingiva in both the arches was done under general anaesthesia. The patient was advised to maintain a scrupulous oral hygiene and was kept under regular observation.

The histopathology of the tissue showed a hyperplastic parakeratinized stratified squamous epithelium with fibrocellular connective tissue. The connective tissue showed variable areas of cellularity, collagen fibers, blood vessels and a moderate degree of chronic inflammatory cell infiltrate. These features were suggestive of inflammatory gingival hyperplasia. (Fig.3)

The patient was reviewed for recurrence at regular intervals with clinical and radiographic examination and after a year of surgical treatment no evidence of recurrence was noticed.

**Discussion**

Gingival enlargement, the currently accepted terminology for an increase in the size of the
gingiva, is a common feature of gingival disease. This is strictly a clinical description of the condition and avoids the erroneous pathologic connotations of terms used in the past such as hypertrophic gingivitis or gingival hyperplasia. Gingival enlargement can be caused by a number of various stimuli, and treatment is based on the underlying cause and pathologic changes.\textsuperscript{[2]}

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 therapy, 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.\textsuperscript{[1,4]}

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 peri-odontal disease when the process involves the loss of gingival attachment and the subsequent loss of interproximal bone.\textsuperscript{[4]}

In this case the exact causative factor could not be identified, further more, no clue as to the unilateral involvement could be established. 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.\textsuperscript{[2]}

Clinically the involved gingiva appeared to be firm without spontaneous bleeding. The chronic nature of the lesion made the gingiva fibrotic. Radiographically interproximal bone loss could be appreciated which is a sign of periodontal involvement. With the history of one year duration the enlargement was considered to be an exaggerated response of gingiva to local irritation complicated by periodontal involvement. The pubertal age of the patient also favored the response of gingiva to irritation. Histologically, the gingival hyperplasia is mainly due to an increase and thickening of mature collagen bundles in the connective tissue stroma. The nodular appearance can be attributed to the thickened parakeratinized epithelium. Microscopic appearance of fibro-epithelial hyperplasia with chronic 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.\textsuperscript{[3]}

The most prevalent types of gingival overgrowth in children are drug-induced gingival overgrowth, hereditary gingival fibromatosis (HGF), and neurofibromatosis I (von Recklinghausen disease).\textsuperscript{[5]} Diffuse gingival enlargement is also found to be associated with syndromes like Cross syndrome, Rutherford syndrome, Ramen syndrome, Zimmerman Laband syndrome and Juvenile hyaline syndrome.\textsuperscript{[6]}

It has been
suggested that gingival enlargement may be due to nutritional and hormonal factors, but these have not been completely substantiated. The constant increase in the tissue mass can result in delayed eruption and displacement of teeth, arch deformity, spacing and migration of teeth.\textsuperscript{[7]}

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.

References
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