PACIFIC JOURNAL OF MEDICAL SCIENCES

{Formerly: Medical Sciences Bulletin}

ISSN: 2072 – 1625

Pac. J. Med. Sci. (PJMS)

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ABSTRACT:

Gingival hyperplasia (GH) is an increase in the gingival height or mass due to proliferation and thickening of gingiva. Gingival hyperplasia represents an over-exuberant response to certain inflammatory and genetic factors, drugs, systemic diseases, neoplasms. Hereditary gingival fibromatosis (HGF) is a rare oral disease, affecting only one in 750,000 people. It is characterized by a slow and progressive enlargement of both maxilla and mandibular gingiva. It usually develops as an isolated disorder, but can be one feature of a syndrome. Drug induced gingival enlargement is frequently observed as a side effect with the use of several medications in the susceptible patients. These reports address the diagnosis, treatment and follow up of two separate cases of gingival enlargement.

Keywords: gingival hyperplasia, hereditary gingival fibromatosis, drug induced gingival enlargement.
Received: June 2011; Accepted: August 2011

INTRODUCTION

Gingival hyperplasia is the increase in size of the gingiva resulting from proliferation of its cellular elements [1]. The clinical features of gingival enlargement depend on the etiologic factors and the pathological processes associated with it. Gingival enlargements are broadly classified into inflammatory, fibrotic and combination of these two. The main etiologic factors are inflammation, genetic factors, drugs, systemic diseases and neoplasms [2,3]. Inflammatory gingival enlargement can be caused by prolonged exposure to dental plaque, which may occur due to poor oral hygiene. In puberty and pregnancy, hyperplasia of the gingival tissues may be due to poor oral hygiene, inadequate nutrition, or systemic hormonal stimulation [4]. Fibrotic gingival

enlargement is a non-inflammatory type of enlargement seen in hereditary and drug induced forms. It is neither hypertrophy nor hyperplasia. Increase in the intercellular matrix is responsible for the enlargement. Hereditary gingival enlargement is rather rare but the drug induced enlargement is much more common [5].

Gingival enlargement is the preferred term for all medication-related gingival lesions previously termed “gingival hyperplasia” or “gingival hypertrophy” [6]. Drug induced gingival enlargement is frequently observed as a side effect with the use of several medications in the susceptible patients. Drugs associated with gingival enlargement can be broadly divided into three categories: anticonvulsants, calcium channel blockers, immuno-suppressants [6].

Hereditary gingival fibromatosis (HGF) is a rare condition characterized by a proliferative fibrous overgrowth of the gingival tissues. It usually develops as an isolated disorder but can be one of the features of several multisystem syndromes [7].The syndromic characteristics most commonly seen in association with HGF are hypertrichosis, mental retardation, epilepsy [7]. HGF is an autosomal dominant disorder with a high degree of penetrance, although recessive forms are also described in literature [8]. Males & females are equally affected as a phenotype frequency of 1:175,000 and a gene frequency of 1: 350,000 [8]. Here we are presenting two case reports of gingival enlargement, one with hereditary cause and another induced by the drug amlodipine.

CASE REPORT 1

A 21year old female presented with a complaint of excessive swelling of the gums and bleeding while brushing. The swelling caused difficulties in mastication and phonation and significant esthetic problem. Besides this no other complains of pain, inflammation, discharge or halitosis were present. Patient remembered having the enlargement since childhood. She did not give a history of taking any drugs known to cause gingival enlargement. The patient gave a history of her brother (35 years) having a similar gingival enlargement, which was later confirmed on his examination. Intra oral examination of the patient revealed uniform, generalized and severe gingival overgrowth involving buccal and lingual tissues of both mandibular and maxillary arches with morphologically normal teeth. The tissue covered the crowns of the teeth till middle 1/3 rd (Figure. 1). The gingival surface was pink, firm, granular and pebbled with abundant stippling. No acute inflammatory signs were present. Moderate local deposits were present. Routine blood investigations showed normal values. Panoramic radiograph revealed bone resorption, more severe in respect to the mandibular teeth’s indicating periodontitis

(Figure. 2). A incisional biopsy was performed and it was confirmed as gingival fibromatosis. Thus a diagnosis of hereditary gingival fibromatosis was made based on absence of drug history, positive familial history, clinical and histopathological features. Gingivectomy procedure with periodontal pack placement was done over duration of two weeks. Instructions were given to the patient to strictly maintain the oral hygiene. After the last gingivectomy procedure the patient returned for post surgical follow up after one month (Figure. 3). Patient was advised for routine scaling and oral prophylaxis procedure once in every six months.

The ethical clearance for the publication of the case report was obtained from the concerned authority.

CASE REPORT 2:

A 40 year old female patient reported to our department with a chief complaint of enlarged gums in the upper and lower front and back teeth region noticed since 1 year. Initially there was small bead-like nodular enlargement of the gums that gradually progressed to the present size covering almost the entire front teeth. Enlargement was associated with intermittent pus discharge, bleeding and difficulty in chewing food. Her past medical history revealed that the patient was on Amlodipine 5 mg taking once daily since 2 years. On intraoral examination, marginal, attached and interdental gingival enlargement was well appreciated covering almost coronal one-third of maxillary and mandibular teeth and is extending to the lingual and palatal mucosa. Gingiva was pink in colour with erythematous area in relation to maxillary left lateral incisor and has lobulated surface. Margins of the gingiva were rolled out with normal gingival scalloping. On palpation, gingiva was firm and resilient in consistency. Hypertrophied areas were painless and did not bleed on touch. Poor oral hygiene status of the patient was assessed from the presence of local irritating factors contributing to the mild inflammatory component of the gingival enlargement (Figure. 4). Patient was subjected to complete hemogram and all the parameters were found to be within normal range. Orthopantomograph revealed complete set of dentition with generalized horizontal bone loss (Figure. 5). On the basis of the patient's history and clinical features, a clinical diagnosis of amlodipine induced gingival overgrowth (AIGO) was made. Patient was subjected to gingivectomy procedure and was recalled for follow up after a month (Figure. 6). Patient's physician was consulted regarding drug substitution or withdrawal of the drug. Patient was instructed to maintain good oral hygiene with the use of chlorhexidine oral rinses.

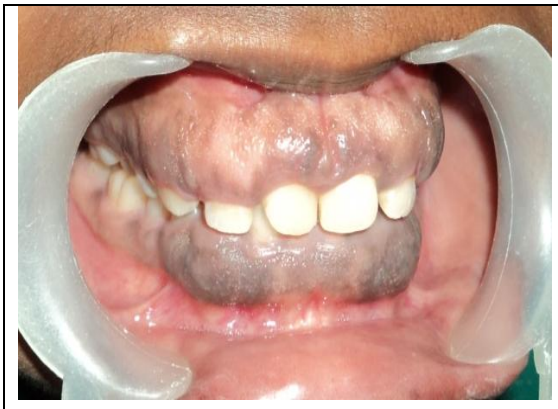

Figure 1: Hereditary gingival enlargement in relation to maxillary and mandibular gingiva.

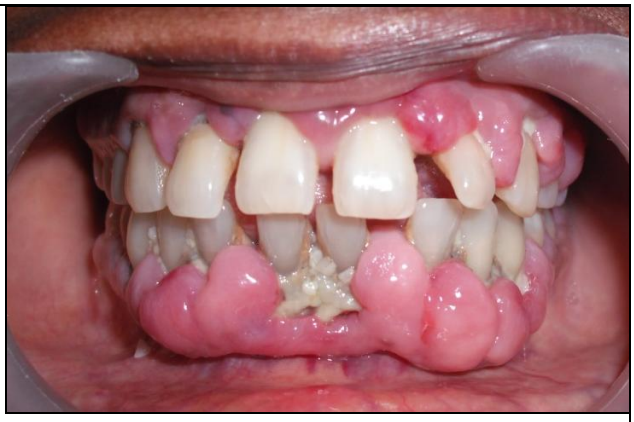

Figure 4: Amlodipine induced gingival enlargement of marginal,attached gingival,interdental papilla.

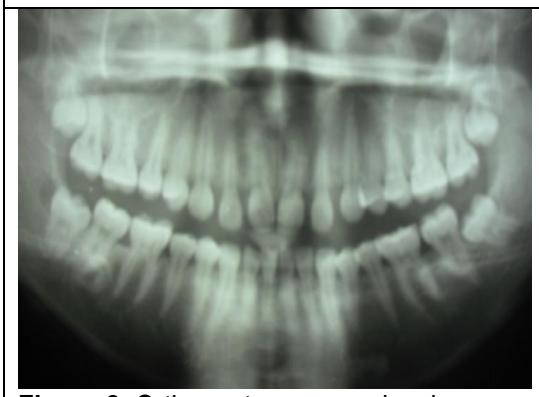

Figure 2: Orthopantomogram showing generalized horizontal bone loss in hereditary gingival enlargement.

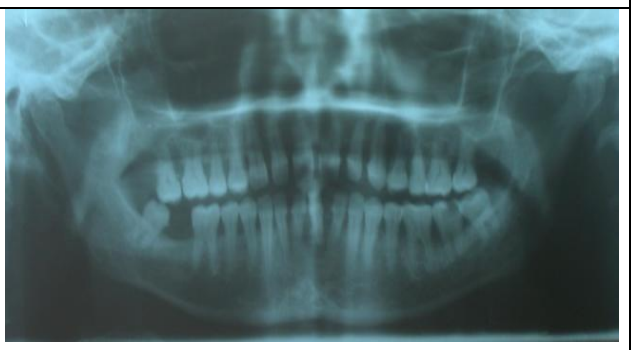

Figure 5: Orthopantomogram showing generalised horizontal bone loss in Amlodipine induced gingival enlargement.

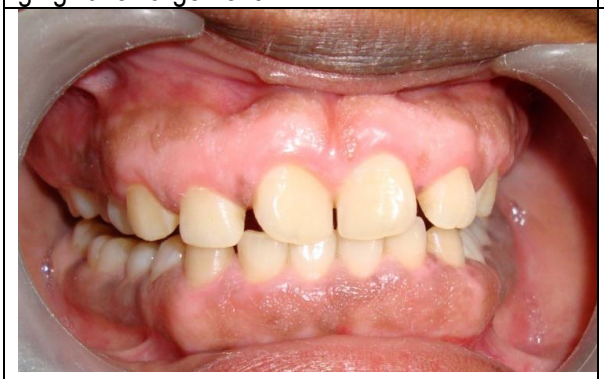

Figure 3: Post operative - after gingivectomy procedure in hereditary gingival enlargement

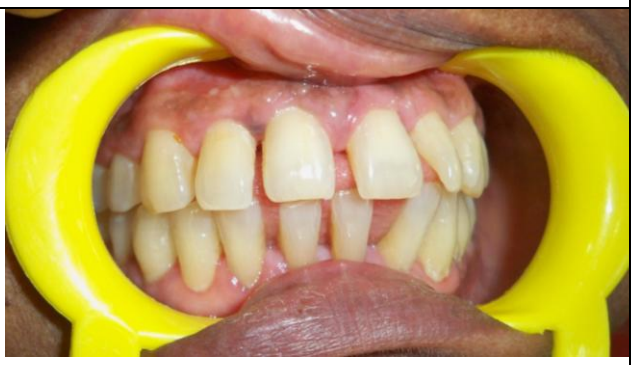

Figure 6: Post operative -after gingivectomy procedure in Amlodipine induced gingival enlargement.

DISCUSSION

This report documents two cases of gingival enlargements due to two different etiologies. Gingival enlargements have hereditary and acquired forms causes of which are inflammation, leukemia, use of medication such as phenytoin, cyclosporine & calcium channel blockers [9].

HGF can be inherited as an autosomal dominant or recessive condition. Autosomal dominance in a four generation pedigree with 50 of 105 at risk of developing gingival fibromatosis was reported by Bozzo et al [10]. According to Bitten court et al [11], this anomaly is classified in two types according to its form. The nodular form is localized and characterized by presence of multiple enlargements of gingival. The symmetric form is most common type and results in uniform gingival enlargement as was seen in the present case. The enlargement usually begins at time of eruption of permanent dentition and rarely develops with eruption of deciduous dentition. Fletcher reported that the enlargement progresses rapidly during "active" eruption and decreases with end of this stage [12]. He also stated that presence of teeth appears to be necessary for HGF to occur as the condition is not seen before eruption of teeth and disappears with loss of teeth. It is accepted that HGF is a disease of genetic origin. Some authors report increase in proliferation of fibroblasts, collagen synthesis and elevated matrix metalloproteinase's while others suggest a decrease in collagenase activity [13]. A gene locus for hereditary gingival fibromatosis has been localized to the 37CM genetic interval on chromosome 2p 21-p22 flanked by D2 S1788 and D2S441 [13].

Calcium channel blockers are considered potential etiologic agents for drug-induced gingival hyperplasia. Although the incidence of nifedipine-induced gingival hyperplasia is about 10%, very few reports of amlodipine-related gingival hyperplasia have been reported in the literature [6]. The prevalence rate of gingival enlargement in patients taking amlodipine is found to be 3.3% [14]. Because only a subset of patients treated with this medication will develop gingival overgrowth, it has been hypothesized that these individuals have subsets of fibroblasts with an abnormal susceptibility to the drug. It has been showed that fibroblast from overgrown gingiva in these patients are characterized by elevated levels of protein synthesis, most of which is collagen [15]. Most types of pharmacological agents implicated in gingival enlargement have negative effects on calcium ion influx across cell membranes, thus it has been postulated that such agents may interfere with the synthesis and function of collagenases, thereby inhibiting collagen degradation [16]. Several factors such as age, genetic predisposition, pharmacokinetic variables, and alteration in gingival connective tissue homeostasis, histopathology, ultrastructural factors, and inflammatory changes may influence the relationship between the drugs and gingival tissues.

CONCLUSION

The above case reports outline the two forms of gingival enlargements, their identification and diagnosis. Gingival hyperplasias have potential cosmetic implications and also provide new

niches for the growth of microorganisms, which is a serious concern for both the patients and oral diagnostician

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