

A RARE CASE OF ADOLESCENT IDIOPATHIC GINGIVAL HYPERPLASIA

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ABSTRACT

There are a number of etiologic factors relevant to gingival hyperplasia. Generally, institution of proper oral hygiene is sufficient for maintaining normal healthy gingiva. Once established gingival hyperplasia can best be treated by resection of all gingival excess, using flaps and gingivectomies. Monthly periodontal check-ups (scaling and polishing) are scheduled to control the gingival inflammation. Cooperation between the periodontist and the patient remains vital for successful management of gingival hyperplasia.

Key words: *Gingival hyperplasia, idiopathic, gingivitis*

INTRODUCTION

Gingivitis is the most common inflammatory reaction of the gingiva. It is caused by colonization of bacterial plaque on tooth surfaces and subsequent invasion of the micro-organisms into the gingival sulcus. Affected gingival tissues are oedematous, soft in consistency, and may bleed when gently probed.^{1,2} In some pathological conditions, gingivitis caused by plaque accumulation can be more severe. Erythematous gingival enlargement may be due to poor oral hygiene, inadequate nutrition, or systemic hormonal stimulation.^{3,4,5}

Gingival overgrowth varies from mild enlargement of isolated interdental papillae to segmental or uniform and marked enlargement affecting one or both of the jaws. Gingival hyperplasia may be due to a number of etiologic factors alone or in combination. However, the majority of cases are of poorly defined etiology and reflect an abnormal response to chronic inflammation associated with local factors such

as plaque, calculus, bacteria, or other unknown factors.⁶ Other common causative factors attributed to gingival hyperplasia include hormonal changes and the use of certain drugs including phenytoin, cyclosporine, nifedipine, and other calcium channel blockers.^{7,11}

Enlargement has also been documented as one of the heralding signs of leukemia, especially the monocytic type. Prudent history and physical examination including biopsy must be considered for new onset of gingival enlargement in patients.

Gingival hyperplasia may also appear during childhood with no obvious association to any causative factor. This particular form of the disease has been identified as idiopathic gingival hyperplasia." In some patients a genetic link has been identified," while spontaneous disease expression is also common. The diagnosis of this disease then becomes a diagnosis of exclusion when no other identifiable causative factor can be identified.

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Gingival hyperplasia is a well-recognized pathologic entity of the periodontium that has obvious implications in the long-term maintenance and restoration of the dentition. The management and manifestations of the disease have been widely reported in the literature. However, little information exists for the surgical management of the severe form of idiopathic gingival hyperplasia

CASE HISTORY

A sixteen year old male reported to the Periodontology Department of Armed Forces Institute of Dentistry, Rawalpindi, with excessive swelling of the gums and propensity to bleed on brushing for the past six months. All the vital signs were within normal limits and the medical history was unremarkable with no apparent correlation with the existing pathology.

Family history of the patient revealed that his elder brother had a similar presentation and underwent treatment three years back. Subsequent to the treatment he had been disease free. The patient himself had been undergoing routine prophylaxis for management of his condition for the past two months. However he noticed that the swelling of his gums increased with the passage of time, in spite of the aforesaid modality of treatment.

CASE FINDING

Extra oral examination of the patient revealed normal symmetrical face with competent lips. Intra orally edematous gingival growth involving three quadrants (Figs-1, 2, 4), except the right maxillary quadrant was noticed (Fig-3). The hyperplastic gingiva extended both labially & lingually. Probing depths, plaque index and bleeding on probing were not recorded on account of gingival overgrowth. Mobility was detected using the Millers mobility index with class mobility on # 26 and class II mobility on # 36 and #46.

Full mouth radiograph (OPG) revealed generalized loss of supporting bone with localized areas of extensive bone loss around the posterior teeth in the maxillary right quadrant and the mandibular quadrants bilaterally (Fig-5). Wisdom teeth were unerupted with the exception of # 48 which was missing.

CASE MANAGEMENT

Preoperative coagulation profile, CBC and urine analysis were carried out and the patient was placed on doxycycline (100mg b.i.d) and metronidazole (200mg t.i.d) as well as chlorhexidine oral rinses three days prior to the surgical intervention.

An external beveled gingivectomy and gingivoplasty were preformed in the effected quadrants under local anesthesia (2% lignocaine with 1/100,000 epinephrine). Postoperatively surgical dressing (Coepack) was applied to the operated sites.

The patient tolerated the procedure well and was advised to continue with the medication prescribed preoperatively for four days; however, the oral rinse was to be continued till further orders.

Post operative period was unremarkable and the patient was recalled weekly for continued debridement. After four weeks his gingiva had a normal appearance and contour (Figs-7, 8).

HISTOLOGICAL FINDINGS

The biopsy of the resected tissues (Fig-6) revealed a nonspecific chronic inflammation.

DISCUSSION

Idiopathic gingival hyperplasia generalized or localized requires systemic management and local therapy. The onset of idiopathic gingival hyperplasia in susceptible individuals is insidious. It is asymptomatic, except in the presence of poor oral hygiene and dental plaque because patients may develop bleeding with tender and swollen gums. Patients with malpositioned teeth, periodontal disease, and poor oral hygiene are at risk of developing gingival hyperplasia. Severity varies depending on the oral/dental health prior to the beginning of therapy; however, not all patients with poor oral hygiene develop gingival hyperplasia

Gingival enlargement occurs primarily on the labial gingival mucosa and in between the teeth (interdental papillae). Potential risk factors for gingival enlargement include poor oral hygiene, periodontal disease, periodontal pocket depth, gingival inflammation and degree of dental plaque.¹⁴ Panorex (panoramic view) radiographs are indicated prior to



Fig 1. Preoperative left lower quadrant



Fig 2. Preoperative left upper quadrant



Fig 3. Preoperative upper jaw



Fig 4. Preoperative lower jaw



Fig 5. OPG shows extensive boneloss in three quadrants except right upper



Fig 6. Excised portion of gingiva



Fig 7. Post operative site after one week



Fig 8. Post operative site upper left quadrant

treatment to evaluate the status of the periodontal tissues or any compromised teeth.

Histologic changes are similar in gingival enlargement that is caused by either Phenytoin or Cyclosporine. The term gingival hyperplasia is a misnomer because enlargement does not result from an increase in the number of cells but rather an increase in extracellular tissue volume. A highly vascular connective tissue occurs histologically with focal accumulation of inflammatory cells, primarily plasma cells. The overlying epithelium is of variable thickness, irregular, and multilayered. Acanthosis and parakeratosis with pseudoepitheliomatous proliferation have been reported. Immunohistologic studies have demonstrated an increase in the number of Langerhans cells within the epithelium and adjacent to inflamed sites. Gingivectomy is recommended for patients who have moderate-to-severe gingival enlargement. No diet restrictions are recommended for patients with gingival enlargement other than minimizing the consumption of sweets, starch, soft drinks, and simple carbohydrates.

Empiric antimicrobial therapy must be comprehensive and should cover all likely pathogens in the context of the clinical setting.

To monitor the oral complications associated with gingival hyperplasia such as bleeding gums, poor oral hygiene, gingivitis, oral candidiasis, periodontists should provide follow-up care at least a year.

Dental hygiene is recommended every month, initially for 3 months followed by 3 month intervals with a view to control dental plaque for 2 years. Patients should practice thorough oral hygiene twice a day after breakfast and before going to bed and rinse the mouth with plain water after each meal.

Chlorhexidine 0.12% once before going to bed and after meals is recommended for those patients known to be at risk for gingivitis. Chlorhexidine 0.12% mouthwash might cause staining of teeth, however, brushing the teeth prior to rinsing out with Chlorhexidine can prevent such an eventuality. The stains can be removed by routine oral prophylaxis.

Severe gingival enlargement in patients with poor oral hygiene can lead to early loss of teeth, however,

the prognosis is better if patients practice regular oral hygiene and plaque control.

CONCLUSION

Apprising the patients regarding recurrent gingival enlargement and the role of oral health in minimizing post operative complications is integral to successful management of gingival hyperplasia. Furthermore, significance of baseline clinical/radiographic evaluation and extraction of teeth with questionable prognosis cannot be underscored. Nevertheless, effort focused on implementing optimum oral hygiene and routine dental care need to be reinforced.

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