Case Report

Inflammatory Gingival Enlargement-A Case Report

Nitin Tomar, Munjal Vidhi, Kaushik Mayur, Department of Periodontology, Subharti Dental College and Hospital, Meerut-250001, Uttar Pradesh, India.

Corresponding Author

Dr. NitinTomar,
Department of Periodontology,
Subharti Dental College and Hospital,
Swami VivekanandSubharti University,
Delhi-Haridwar bypass road,
Meerut-250001, Uttar Pradesh, India.
E- Mail:drnitintomar@gmail.com

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Abstract
Gingival enlargement is a common clinical problem, usually associated with specific conditions. This condition finds a unique place in literature, because it has been associated with a variety of local and systemic factors. The aim of publishing this case report is to present the clinical, histopathological features and treatment of inflammatory gingival enlargement which disturbed the aesthetics and masticatory function of the patient.

Key Words: Inflammatory gingival enlargement, Aesthetics, Masticatory function, Internal Bevel Gingivectomy

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Introduction

Gingival enlargement is a common feature of gingival disease and may be caused by fibrous overgrowth or gingival inflammation or a combination of two.[1] The types of gingival enlargement can be classified according to etiologic factors and pathologic changes as follow:[2]

1. Inflammatory enlargement
2. Drug induced enlargement
3. Enlargement associated with systemic disease
4. Neoplastic enlargement

Gingival enlargement is a common finding in clinical practice and the appropriate treatment depends on correctly diagnosing the cause of the enlargement. The most common form of enlargement is due to plaque induced inflammation of the adjacent gingival tissues (inflammatory hyperplasia) and this tends to be associated most commonly with the interdental papillae andmay be localized or generalized. Such gingival enlargement can be exaggerated by hormonal effects, as found in puberty and pregnancy, and may also be complicated by certain systemic medications.[3] Plaque-induced inflammatory hyperplasia should resolve with debridement of plaque and calculus and improved oral hygiene, especially when the gingival tissue is edematous. Where the gingival tissue is fibrotic, resolution of enlargement may not occur, resulting in the persistence of periodontal pocket such that effective oral hygiene is impeded. This scenario requires a

more detailed assessment and a longer term management plan designed to map the level of gingival and possibly periodontal involvement. Surgical management to remove enlarged tissue and provide improved access for the patient's oral hygiene may be required.[4]

In the inflammatory type of enlargement, the gingivae are soft, edematous, hyperemic or cyanotic and usually painful or at least sensitive. These gingivae are quick to bleed when prodded and appear smooth and distended; the normal stippling has usually been lost, clinically as well. "Pitting" can be observed after application of a point source of pressure. In contrast, in non-inflammatory enlargement the gingivae appear normal in color or even somewhat pale, and stippling may be normal or exaggerated.[5] In puberty and pregnancy, gingival hyperplasia can be due to poor oral hygiene, inadequate nutrition, or systemic variation in hormonal stimulation.[6]

Figure 1: Pre-operative extraoral photograph

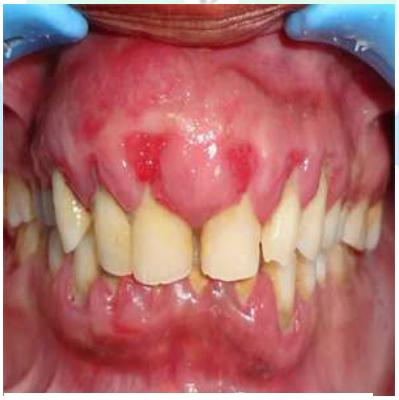

Figure 2: Pre-operative intraoral photograph

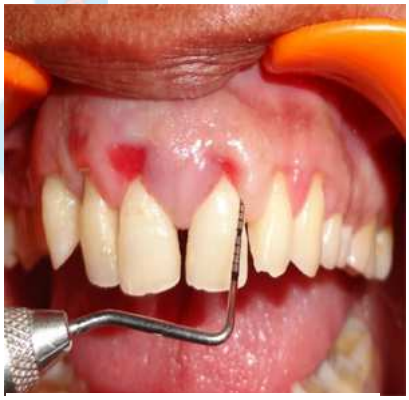

Figure 3: Photograph showing the probing depth

Gingival enlargements are also seen in several blood dyscrasias e.g. leukaemia, thrombocytopenia, or thrombocytopathy.[7]

Case Report

A 40 years old woman reported to the Department of Periodontology, Subharti Dental College and Hospital, Meerut with the complain of gingival enlargement in relation to the maxillary and mandibular anterior teeth region since 8 months.(Figure 1) Patient also complained of difficulty in chewing and concern for the aesthetics was reported by the patient. The patient had not previously used any medication known to provoke gingival enlargement. An intra-oral examination revealed the presence of Grade III enlargement in relation to the maxillary and mandibular anterior teeth region, with generalized gingival bleeding on probing, and probing depth of more than 5 mm was present in the anterior region. (Figure 2 and 3)

Treatment

Periodontal therapy including Phase I therapy followed by internal bevel gingivectomy for esthetic benefit was performed. (Figure 4) Pockets were measured with pocket marker and bleeding points were produced on the outer surface of the gingiva. After that an internal bevel incision was made to a point apical to the alveolar crest depending on the thickness of the tissue. The thicker the flap the more apical is the ending point of the incision. Then the thinning of the flap was done with the initial incision. Flap was reflected with a periosteal elevator. Instrumentation was done with complete debridement. An intrabony defect was seen distal to right

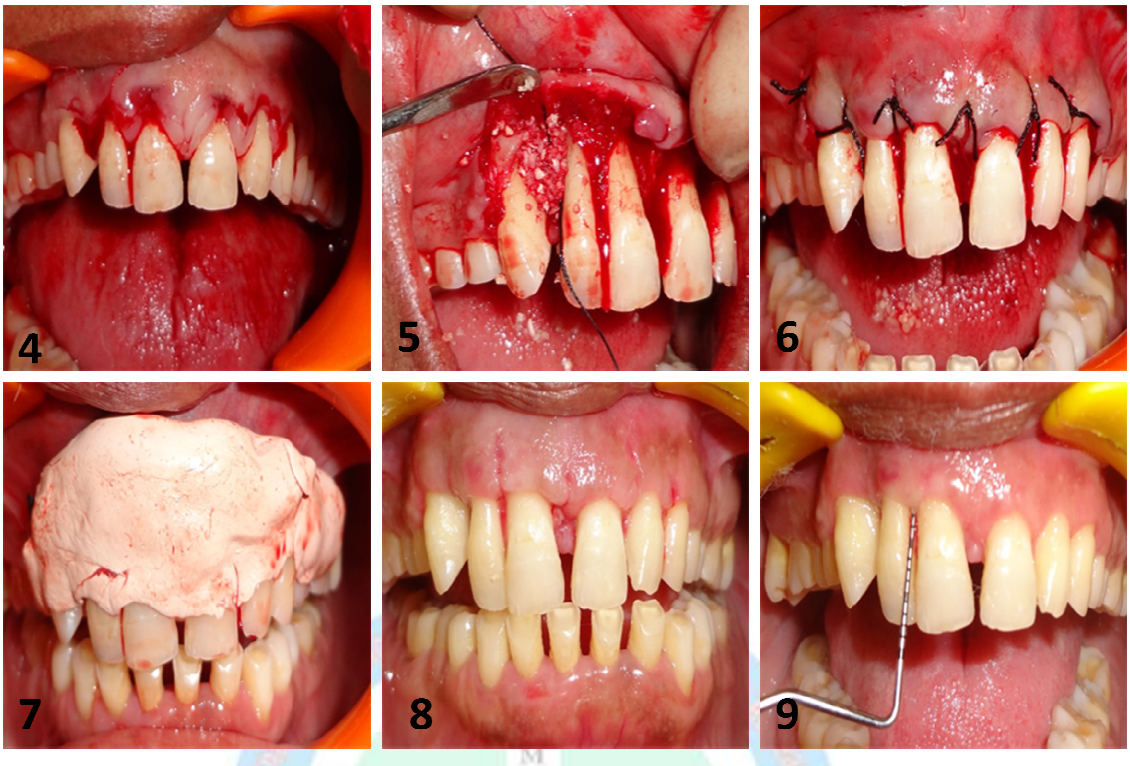

Figure: 4) Internal bevel incision; **5)** Mucoperiosteal flap raised and DFDBA graft placed; **6)** Sutures given; **7)** Coe pack placed; **8)** 7 days post-operative intraoral picture; **9)** 3 months post-operative picture.

Figure 10: 3 months post-operative extraoral picture

lateral incisor. Demineralised freeze dried bone allograft (DFDBA) was filled in the defect after pre-suturing was done and then the flap was sutured backto secure the facial and the palatal flaps. (Figure 5, 6) The area was covered with a periodontal pack. (Figure 7) An excisional biopsy of the tissue was done. H and E staining was done for the specimen and it showed hyperplastic stratified squamous epithelium with underlying connective tissue showing numerous blood vessels with increased inflammatory cells predominantly plasma cells arranged in a diffuse pattern. Features were suggestive of inflammatory hyperplasia. Antibiotics and analgesics were prescribed for 5 days. One month recall checkup showed uneventful healing and patient was followed for next 6 months at a

regular interval of 1 month. (Figure 8, 9 and10)

Discussion

With regard to the enlargement of the gingiva, the terms "hypertrophy" and "hyperplasia" have been used for decades, unfortunately without much definition or specificity. In fact, "gingival hypertrophy" as a pathological entity probably does not exist.[8] Gingival enlargement may be caused by a multitude of causes. Enlargements are a common clinical finding and most represent a reactive hyperplasia as a direct result of plaque-related inflammatory gingival disease.[9] The most common is chronic inflammatory gingival enlargement, when the gingiva presents clinically as soft and discolored. This is caused by tissue edema and infective cellular infiltration caused by prolonged exposure to bacterial plaque, and is treated with conventional periodontal treatment, such as scaling and root planing. Situations in which the chronic inflammatory gingival enlargement include significant fibrotic components that do not respond to and undergo shrinkage when exposed to scaling and root planing are treated with surgical removal of the excess tissue, most often with a procedure known as gingivectomy.[10]The usual clinical presentation of plaque-induced gingival hyperplasia includes enlarged gingival contours due to edema or fibrosiscolor transition to a red and ⁄ or bluish red hue, bleeding upon probing and increased gingival exudates.[9] Inflammatory enlargement caused by local factors is self-perpetuating since it is often impossible to properly clean the "pseudopockets" which are formed by bulging tissue. The situation is exacerbated as bacterial colonization proceeds within the dental plaque and the host response to microbial products intensifies. A foul breath results as food debris is degraded by the accumulating microorganisms. While local etiologic factors are almost always present in cases of inflammatory type gingival enlargement, there are also several important systemic factors which may contribute to the problem and compromise the success of therapy directed at elimination of focal irritants.[5] In the present case report inflammatory enlargement was present in relation to both maxillary and mandibular anterior teeth region causing esthetics and phonetics problem in the patient. After non-surgical therapy there was resolution of the inflammation in relation to the lower anterior teeth whereas maxillary anterior teeth showed no resolution and fibrotic component was present which was then corrected by surgical therapy.

Conclusion

The local factors i.e. plaque and calculus are known to be responsible for gingival enlargement. Therefore, the importance of regular check-up and oral prophylaxis cannot be overlooked. In the present case, size of the hyperplastic tissue interfering with the patient's ability to chew, speak and was causing serious esthetic problems so it was excised completely. Future studies conducted on this aspect of the case report would provide us with the better information regarding the etiology of gingival enlargement.

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