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Unusual Gingival Enlargement with Aggressive Periodontitis: A Case Report

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Abstract

Aim: This article describes the surgical management of a young, female patient with severe gingival enlargement of unknown etiology.

Background: Gingival enlargement frequently occurs as gingival hyperplasia, representing a reaction to a known stimulus or agent and, histopathologically, implies an increase in both extracellular matrix and cell numbers. The enlargement may range from mild, in which gingival architecture is minimally affected, to severe, in which the gingiva becomes bulbous and covers the clinical crowns of teeth. A number of local and systemic factors, such as plaque, hormonal changes, drug ingestion, and heredity, can cause or influence gingival enlargement. Mild to moderate increase in gingival bulk is relatively common, but massive gingival enlargement with associated bone resorption is rare.

Case Description: This case involved a 19-year-old female patient who presented with generalized severe gingival enlargement with aggressive periodontitis, a condition of some five to six months in duration.

Results: Based on a thorough clinical and radiographic examination, laboratory tests, and oral hygiene instructions, an internal bevel gingivectomy was performed to remove excess gingival tissue and areas of bone loss were debrided properly. There was no recurrence eight months following the last surgery.

Summary: Although we were not able to identify the exact cause of the gingival enlargement, amelioration of the unusual very soft, friable, enlarged gingivae and the severe periodontal attachment loss was observed.

Clinical Significance: Before initiating any periodontal management of a case of severe gingival enlargement with aggressive periodontitis, it is recommended to perform a complete extraoral and intraoral examination with radiographs, take a family and medical history, and determine if any medications may be responsible for the gingival enlargement. Additional testing and analysis, as described in this case, may be necessary.



Keywords: Case report, gingival enlargement, idiopathic, heredity, amyloid/amyloid-like, bone resorption, aggressive periodontitis

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Introduction

Gingival enlargement is a common finding in inflammatory periodontal diseases. Several types of gingival enlargement that differ from common lesions have been classified according to etiologic factors and histopathological findings. Drug-induced gingival enlargement, such as that produced by phenytoin, cyclosporine,[1] and calcium channel blockers,[2] and conditioned gingival enlargement associated with systemic disorders produced by hormonal factors,[3] leukemia,[4] vitamin C deficiency,[5] and idiopathic gingival fibromatosis[6] have been reported.

Ligneous periodontitis is a unique destructive membranous periodontal disease characterized by gingival enlargement and periodontal tissue destruction due to accumulation of amyloid-like material.[7,8]

This report presents the clinical features and dental management of an unusual case of gingival enlargement. Clinically, this patient showed extensive gingival enlargement with very soft, butterlike consistency of this gingiva. Histopathologically, the tissue was edematous and exhibited severe infiltration of inflammatory cells. At the same time, severe alveolar bone loss was observed. Although we were not able to identify the cause of the gingival enlargement, amelioration of the gingival inflammation and the periodontal attachment loss was observed.

Case Description

In the middle of April 2008, a 19-year-old female reported to the Department of Periodontics with a chief complaint of massive swelling in her gums for five to six months. Her family history was unremarkable. There were no relevant findings in her medical history, and she was not taking any regular medication, such as phenytoin, cyclosporine, or nifedipine, that could have induced gingival enlargement. Eight months earlier she was seen at a private dental clinic and had an extraction of her badly carious and mobile tooth #19. Two months later, mobile and badly carious teeth #3 and #14 were extracted.

The enlarged gingiva partially covered all of her teeth. Because of her partially competent lips, she appeared to be a mouth breather. The gingiva was reddish pink, very soft, friable, and edematous and had a shiny surface. On both sides of the posterior region of the palate, the enlarged gingiva was overhanging toward the midline of the palate. Bleeding from the gingival pockets occurred easily on probing. The lesion was confined to the marginal, interdental, and attached gingiva; the alveolar mucosa was not involved. The patient's oral hygiene was poor, yet negligible calculus was noted around all the teeth. Some of the teeth were pushed out of the dental arch. This mal-arrangement was due to insufficient arch space to accommodate the teeth, resulting in the malalignment (Figures 1–3).



Figure 1. Pre-operative front view of gingival enlargement.



Figure 2. Preoperative mirror view of right maxillary tuberosity region.

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Intraoral periapical radiographs had been taken at a private dental clinic two months earlier that showed moderate to severe bone loss around all the molars and the mandibular incisors (Figure 4).



Figure 3. Pre-operative mirror view of left maxillary tuberosity region.

Laboratory tests, including complete hemogram, showed all blood cell counts within normal limit. Her chest x-ray also was normal.

An incisional biopsy was taken from the retromolar region of the maxillary arch. Histopathologically, parakeratinized stratified squamous epithelium with densely arranged collagen fibers in underlying connective tissue was seen. Marked inflammatory edema and the predominance of lymphocytes, in addition to other inflammatory cells, were observed.

Treatment

Oral hygiene instructions were given, but the enlarged gingiva restricted plaque removal. Therefore, internal bevel gingivectomies were performed for all quadrants in the maxillary and mandibular arches. The surgical intervention was



Figure 4. Intraoral periapical radiograph showing severe bone loss around all molars and mandibular incisors.

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Figure 5. Treatment procedure consists of internal bevel gingivectomy.



Figure 6. Example of the amount of excised gingival tissue.

carried out under local anesthesia using internal bevel and crevicular incisions to remove the large increments of hyperplastic tissues (Figure 5).

The patient was given a prescription for an antibiotic for 24 hours before the surgery and five days postoperatively (Amoxicillin 500 mg, three times a day) to prevent postoperative bacteremia and an analgesic for three days postoperatively (Aceclofenac 500 mg, twice a day) to relieve postoperative pain. Sutures (3-O, silk) and a periodontal dressing (Coe-Pak) were placed and removed after one week. The interval between surgical procedures was two weeks.

An excisional biopsy sample of the gingival tissues was again submitted for histopathologic examination. Oral pathologist examined the excised tissue by staining it with H/E stain and Masson's Trichrome; the specimen was negative for fibrin. Congo red staining for amyloid/amyloid-like deposition also was negative (Figures 6–9).

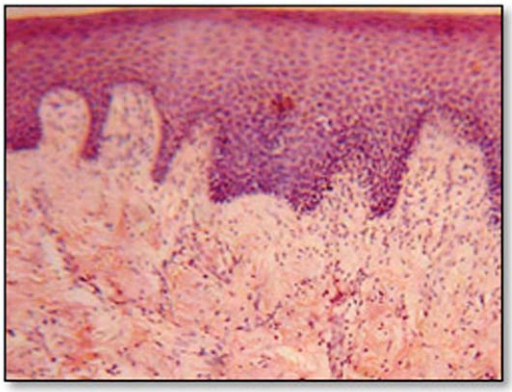


Figure 7. Histopathologic examination showing the parakeratinized stratified squamous epithelium with densely arranged collagen fibers in connective tissue.

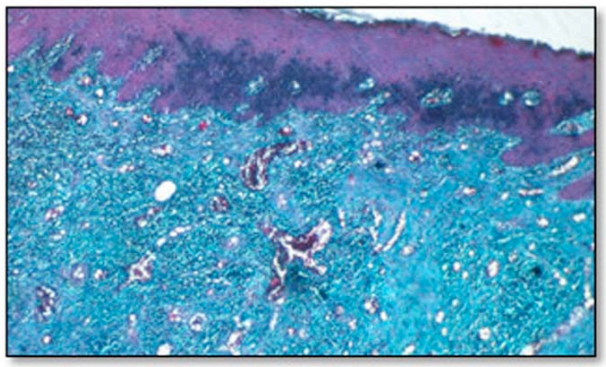
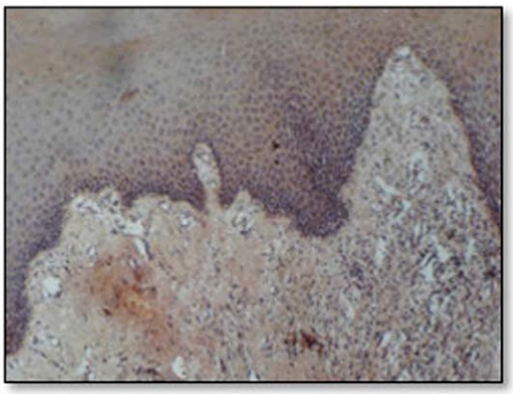


Figure 8. Masson's trichrome staining showing negative stain for fibrin deposition in underlying connective tissue.



Congo red staining showing negative stain for amyloid/amyloid-like material.

Periodontal management with hand and ultrasonic scaling was performed at monthly intervals, and there was no recurrence of gingival enlargement until seven months later. However, for personal reasons, the patient could not visit for the following four months (Figures 10-12).

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Figure 10. Postoperative front view showing stippled gingival margins.



Postoperative mirror view of the right maxillary tuberosity region.



Figure 12. Postoperative mirror view of left maxillary tuberosity region.

Discussion

This patient demonstrated extensive gingival enlargement with the clinical features of the enlarged gingiva being softness, friability, and edema. Generalized alveolar bone loss was present radiographically. Histopathologically, intense edema and inflammatory cell infiltration was seen.

Initially it was believed the gingival enlargement was due to an uncommon entity—ligneous periodontitis, which is a rare destructive membranous periodontal disease in which amyloid-like material and subepithelial fibrin deposition both play a part, resulting in characteristic gingival enlargement and periodontal breakdown.[7,8] Clinically it presents with soft, friable gingiva with a pseudomembrane. But, in the absence of a pseudomembrane and having a negative Masson's trichrome stain for fibrin and a negative congo red stain for amyloid/amyloid-like material, a diagnosis of ligneous periodontitis was ruled out. The absence of early recurrence of gingival enlargement confirmed the exclusion of ligneous periodontitis.

The clinical and histopathologic features and systemic examinations excluded the diagnosis of neoplastic enlargement. Gingival hyperplasia occurs in some patients taking certain drugs, such as phenytoin, cyclosporine, and nifedipine. This patient had not taken any of these medications.

The extensive gingival enlargement suggests that the patient may have been suffering from a syndrome associated with gingival fibromatosis, i.e., Murray-Puretic-Drescher syndrome, Rutherford's syndrome, Leband syndrome, or Cross syndrome.[9] Clinically, the enlarged gingiva in hereditary or idiopathic gingival fibromatosis is firm and leathery in consistency. The histologic features are densely arranged collagen bundles with numerous fibroblasts, both of which differ from the features in this patient. This patient had developed normally, without any symptoms of mental retardation. She did not suffer from epilepsy or hypertrichosis, nor did she have any tumors or corneal dystrophy. No skeletal deformities or defects of the skin or fingernails were observed. Thus, it is unlikely that the above-mentioned syndromes were related to this gingival enlargement.

Systemic factors affect host response and alter the tissue response to plaque.[3–5] The laboratory tests revealed no evidence of any systemic disorders such as leukemia, diabetes mellitus, or hormonal disorders, including disorders of the sex hormones. Gingival enlargement also is a symptom of scurvy;[5] however, the patient did not have any other signs of scurvy such as petechiae, ecchymoses, or spontaneous bruising of the extremities.

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Our case was more similar to one case reported by Nitta et al.[10] involving a 15-year-old female patient with unusual gingival enlargement accompanied by rapidly progressive periodontitis. In that particular case, the enlargement was recurrent and accompanied by severe bone loss.[10]

Schmidt et al.[11] presented two cases of generalized massive gingival enlargement and osteolysis of alveolar bone in a 30-year-old female and a 36-year-old male patient. Hereto, the etiology of the gingival enlargement could not be established in either case.[11]

In many types of gingival enlargement, the rapid loss of alveolar bone or attachment level is not observed. However, our 19-year-old patient exhibited rapidly progressive destruction of the periodontal tissues. Because we could not obtain any prior dental records from the patient, we were not able to determine when the periodontal destruction had begun. It would appear that local plaque irritation had produced the unusual gingival enlargement and then deepened the gingival pockets. Such a condition may have allowed subgingival periodontopathic bacteria, including *B. gingivalis*, to colonize and proliferate, and the alveolar bone loss might then have advanced very rapidly.[12,13] Periodontal treatment prevented further loss of the alveolar bone.

Summary

Management of a patient with generalized gingival enlargement should include a complete medical history and physical examination to rule out known causative agents and factors. A biopsy should be performed to confirm the diagnosis and to rule out a neoplasm. Surgical management may require gingivectomy or total extraction of teeth, if required. In the case presented, internal bevel gingivectomies for all quadrants were performed. A satisfactory patient response to this treatment was observed and no recurrence of gingival enlargement was seen in this patient at the one-year, follow-up visit.

As shown in this particular case, it is very important to correlate the extraoral and intraoral clinical findings, family and medical history, medications, and results of histopathologic and laboratory tests to rule out known causative agents/factors and different pathologic conditions that may manifest themselves similar to the conditions seen in this case.

Additionally, regularly scheduled maintenance visits and maintenance therapy will be necessary to prevent additional destruction of the periodontal tissues.

Clinical Significance

Although the etiologic factors responsible for the unusual enlargement may have differed from those responsible for the rapid alveolar bone loss, it is likely that such an unusual condition could have been caused by a number of interrelated factors and undiagnosed factors, as well as a periodontal infection. Additional testing and analysis, as described in this case, may be necessary.

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