Case Report

Management of Idiopathic Gingival Fibromatosis: Report of a Case and Literature Review

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Abstract: *Gingival hyperplasia is a rare condition and is of importance for cosmetic and mechanical reasons. Idiopathic gingival fibromatosis, a benign, slow-growing proliferation of the gingival tissues, is genetically heterogeneous. The enlargement is most intense during the eruption of the primary and permanent teeth, and minimal or nondetectable growth is observed in adults. The genetic aspect, clinical feature, histopathology, immunohistochemistry, and treatment aspects are reviewed. The purpose of this paper was to report a case of idiopathic gingival fibromatosis in a 13-year-old female who had a negative family history for a similar type of gingival enlargement. The diagnosis was established through history, clinical examination, and histopathology using both hematoxylin and eosin and Van Giesen stain (a special stain for collagen). Surgical treatment, which included both gingivectomy and gingivoplasty, was carried out. The case showed remarkable esthetic and functional improvement. The patient returned after a year and showed no recurrence.* *(Pediatr Dent 2011;33:431-6) Received February 18, 2010 / Last Revision June 1, 2010 / Accepted June 20, 2010*

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Gingival fibromatosis (**GF**) is a heterogeneous group of disorders characterized by progressive enlargement of gingiva caused by increase in submucosal connective tissue elements.[1] Idiopathic gingival fibromatosis (**IGF**) is a rare, benign, asymptomatic, nonhemorrhagic, and nonexudative proliferative fibrous lesion of the gingival tissue.[2] A slowly progressive fibrous enlargement devoid of inflammatory erythema of the maxillary and mandibular gingiva without a family history is a feature of idiopathic fibromatosis of the gingiva. Characteristically, this massive gingival enlargement appears to cover the tooth surfaces and displace the teeth. While the cause of the disease is unknown, there appears to be a genetic predisposition.[3,4]

The gingiva in IGF is usually pink in color. Enlargement may be generalized or localized to specific areas such as the maxillary tuberosity and labial gingiva around lower molars. Severity may vary from mild involvement of 1 quadrant to severe involvement of all the quadrants.[1] Effects of the lesion include diastema, malposition of teeth, and prolonged retention of primary teeth resulting in esthetic and functional problems. In extreme cases, the lips are everted by the excess gingival bulk and are incompetent.[5] The enlargement is most intense during the eruption of the primary and permanent teeth, and minimal or nondetectable growth is observed in adults.[6] It is rarely present at birth or arises in adulthood.[7]

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Takagi et al.,[8] divided the age of onset into the: preeruptive period (< 6 months); primary dentition period (6 months to 6 years); mixed dentition period (6-12 years); permanent dentition period before adolescence (12-20 years); and permanent dentition period after adolescence (≥20-years-old).[9] Maximal enlargement occurs either during loss of primary teeth or in the early stages of eruption of permanent teeth. Enlargement progresses rapidly during "active" eruption and decreases with the end of this stage.[9]

GF can occur as an isolated finding or as a part of a genetic syndrome,[10,11] and the involvement may be generalized or localized. Accordingly Takagi et al.,[8] have classified GF as: isolated; hereditary (localized/generalized); isolated idiopathic GF (generalized/localized); GF with hypertrichosis; GF with hypertrichosis and mental retardation and/or epilepsy; GF with mental retardation and/or epilepsy; and GF with other diseases and/or with formation of syndromes (Table 1).[12] Isolated GF, also referred to as IGF,[11] may result from a single gene mutation, whereas syndromic forms may result from alteration in multiple genes or a single gene dosage effect.[12] GF syndromic forms show dominant or recessive Mendelian transmission patterns.

The HGF gene has been localized to chromosome 2p21-p22 (HGF1), and the gene locus has been localized to a 37 cM genetic interval on chromosome 2[13] and chromosome 5q13-q22 (HGF2).[14] Recently, a mutation in the son of sevenless-1 (SOS-1) gene has been suggested as a possible cause of isolated (nonsyndromic) GF, but no definite linkage has been established.[15] The clinical presentation of HGF is variable, both in the distribution (number of teeth involved) and severity of expression. This variability may be due to the

Table 1. SYNDROMES ASSOCIATED WITH GINGIVAL FIBROMATOSIS[11]

Syndrome	Characteristics
Rutherford syndrome	Congenitally enlarged gingivae, delayed tooth eruption, curtain-like superior corneal opacities, mental retardation, aggressive behavior, and dentigerous cysts
Zimmerman-Laband syndrome	Gingival fibromatosis, large nose, ear abnormalities, syndactily (frog-like fingers and toes), hyperplasia of nails and terminal phalanges, hyper-extensible joints, hepatomegaly, and splenomegaly
Cowden syndrome	Gingival papillomas as part of widespread oral, facial, and pharyngeal papillomas, multiple hamartomas, and neoplasms
Jones syndrome	Gingival fibromatosis with progressive deafness
Tuberous sclerosis	Single or multiple fibromas of gingivae, oral mucosa, and skin; epilepsy; mental retardation (MR); and hamartomas of brain, heart, and kidney
Goltz-Gorlin syndrome	Gingival and other mucosal papillomatosis, lip and tooth defects, piokiloderma, dermal fat herniation, adactyly, and syndactily
Mereay-Pruretic-Drescher syndrome	Gingival fibromatosis with multiple juvenile PAS-positive hyaline fibromas of the head (turban tumor), trunk, and extremities; suppurative lesions of skin and mucosa; MR elevated urinary hyaluronic acid, and dermatin sulfate
Cross syndrome	Gingival and alveolar enlargement, hypopigmentation, microphthalmia, cloudy corneas, athetosis, and MR
Ramon syndrome	Gingival fibromatosis, hypertrichosis, cherubism, MR epilepsy, juvenile rheumatoid arthritis, and perivascular fibromas in gingival biopsies

variable expression of a common gene mutation, allelic mutations, or nonallelic mutations.

Gingival enlargement occurs because of increased deposition of extracellular matrix (**ECM**), particularly interstitial collagen type I, which is one of the major components of the ECM of gingival connective tissue. Its content is determined by the balance between synthesis and degradation of matrix metalloproteinases.[16] Increased production and reduced degradation of ECM is thought to be brought about by transforming growth factor β1 (TGF-β1), a cytokine, which is an important mediator of wound healing and tissue regeneration.[17] TGF-β1 is the major mediator influencing collagen turnover[18] and is an autocrine stimulator of HGF fibroblast proliferation.[17] (Fibroblasts from hereditary GF tissues produce increased amounts of tgf-β1 and β2.[19] Wright et al.,[20] documented the expression of TGF-β isoforms and TGF-β receptors in hereditary and drug-induced gingival overgrowth (**DGIO**). They indicated differences in TGF-β isoform expression between DIGO and HGF and noted that alterations in TGF-β isoform expression by fibroblasts are implicated in the pathogenesis of both types of gingival overgrowth.

Microscopic sections show connective tissue alterations and an increased amount of collagen fibers arranged in different directions associated with few fibroblasts. Marked interwoven fascicles of dense fibrous tissue with slender fibroblastic nuclei scattered throughout the stroma and mild chronic inflammatory cell infiltrate are also noted.[21] Unusual findings, including small calcified particles, amyloid deposits, islands of odontogenic epithelium, and osseous metaplasia, were observed in familial GF.[22]

The proliferative potential of mesenchymal fibroblasts has been assessed using immunohistochemical expression of the markers, proliferating cellular nuclear antigen (**PCNA**), and pKi-67. In vivo studies by Saygun et al.[23] and Marteli Jr. et al.[24] did not reveal any proliferating fibroblasts in the lesional connective tissue, but indicated an increase in collagen and glycasaminoglycans. Recently, studies of tissue specimens obtained from individuals with IGF examined histochemically and by light and electron microscopy suggest that this condition may be due to an increase in collagen synthesis.[25] Histomorphometric evaluation suggests that epidermal growth factor (**EGF**) and its receptor (**EGFr**) in epithelial cells of such patients have demonstrated a higher proliferation rate than in normal gingiva. EGF and EGFr may stimulate epithelial cell proliferation, with the resultant apical migration of oral epithelium and formation of slender deep epithelial papillae without hyperplastic alterations.[26]

Cosmetic concerns aside, a compromised oral cavity may cause difficulty in eating, speech, hygiene, and oral competence. In addition, social consequences can be dramatic, forcing patients to lead an isolated, reclusive life. Treatment depends on the severity of the enlargement. Minimal enlargement is treated through scaling of teeth and home care to maintain good appearance.[6] Overgrowth of tissue needs to be surgically removed. Various treatment modalities have been suggested, but controversy still remains regarding the exact period in which it should be accomplished.

Case report

A 13-year-old female patient of Asian origin and Dravidian descent presented with her father and complained of nonvisibility of her teeth due to overgrowth of her gums, which had gradually covered her teeth. This condition developed when she was 6-years-old. She had visited a dentist who performed gingivectomy to expose her teeth. The condition recurred, however, at 11 years of age. This had led to impairment of esthetics, and the patient was not able to close her lips completely and had difficulty chewing food. All developmental milestones were normal. The patient attained puberty at approximately the age of 10½ years. There was no record of gingival bleeding, consanguineous marriage between parents, or family history of a similar complaint.

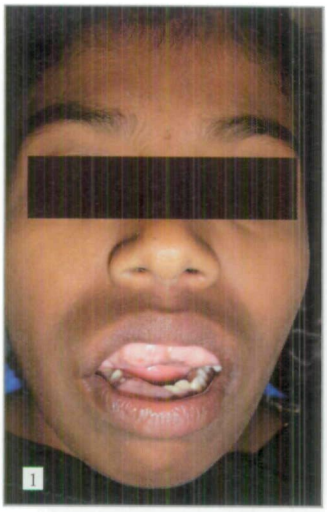
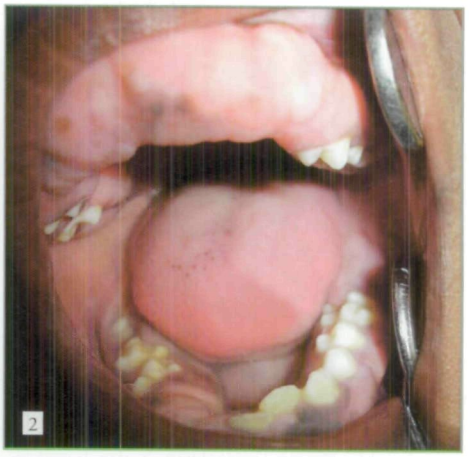
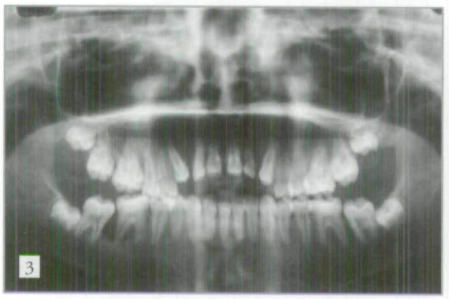
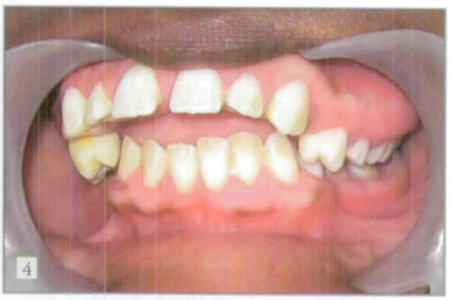
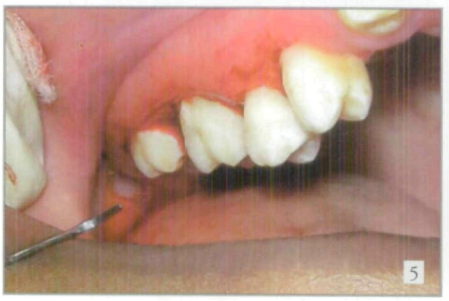
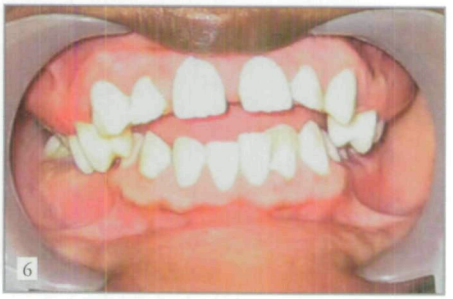
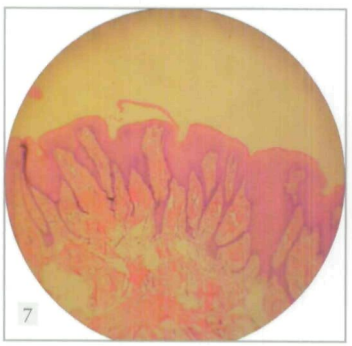
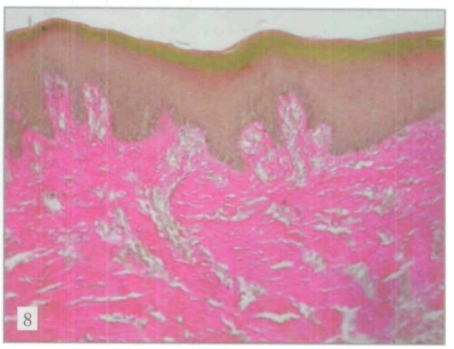

Figure 1. Extraoral photograph showing incompentent lips.

Figure 2. Intraoral photograph showing dense firm gingival enlargement involving both the maxillary and mandibular arches.

Figure 3. Panoramic radiograph showing severe localized periodontitis.

Figure 4. Post gingivectomy intraoral photograph.

Figure 5. Intraoral photograph showing gingivoplasty in the maxillary right quadrant.

Figure 6. Intraoral photograph taken after gingivoplasty.

Figure 7. Photomicrograph of hematoxylin and eosin section (10x light microscope) showing epithelium with thin, long rete pegs and dense collagen in all direction in connective tissue.

Figure 8. Photomicrograph of Van Giesen stain (10x) showing collagen fibers.

On examination, the lips were incompetent with a convex profile (Figure 1). There was generalized enlargement of the gingiva involving both the maxillary and mandibular arches, which was more pronounced in the maxillary arch, and gingiva partially or completely covered the crowns of the teeth (Figure 2). The gingiva was pink in color, firm, and resilient with areas of brown pigmentation and mild bleeding. Malposition of teeth was also noted. A periodontal pocket measuring approximately 7 mm was noted distal to the permanent mandibular right first molar. The case was provisionally diagnosed as IGF. To evaluate the presence of any bone pathology, maxillary and mandibular occlusal radiographs and panoramic radiographs were taken. Panoramic radiography showed a full complement of teeth and severe periodontitis with respect to the mandibular right first molar (Figure 3). Maxillary and mandibular cross-sectional occlusal radiographs did not show any bone pathology.

The peripheral blood results were normal and correlated with an absence of any history of systemic disease. Gingivectomy of both the arches was done under general anesthesia. Incisions were made from the distal aspect of the first molar extending till the midline, on both the facial and lingual surfaces using Kirkland knives, bilaterally. Interdental incisions were done with Orban's knife, and the gingival overgrowth was removed (external bevel gingivectomy). A pressure pack was used to control postsurgical bleeding. Once the bleeding was under control, a periodontal dressing (Coe-Pak periodontal dressing, GCAmerica Inc, Alsip, Ill) was placed to promote healing. Anterior open bite, spacing and proclination of anterior maxillary teeth and bilateral posterior telescopic bite were observed. The patient was recalled after 1 week, and the periodontal pack was removed.

The patient was prescribed a 0.12% chlorhexidine mouthrinse for 2 weeks to maintain oral hygiene and was recalled after 1 month for a checkup (Figure 4). Three weeks later, a gingivoplasty was performed in quadrants under local anesthesia during 4 sittings over 4 consecutive weeks (Figure 5). The patient was asked to continue using the chlorhexidine mouthrinse and to maintain good oral hygiene habits. The patient was recalled after 20 days for a checkup (Figure 6). Histopathology of the excised specimen, stained with hematoxylin and eosin (Figure 7) and Van Giesen stain (Figure 8), was consistent with fibrous hyperplasia of the gingiva. The patient was advised to maintain good oral hygiene habits. Reconstructive periodontal surgery for the mandibular right first molar was planned. A combination of surgical and fixed orthodontics to correct the telescopic bite was also planned. However the patient did not come back for the treatment. She reported back to us after 1 year, at which time her gingival condition was satisfactory with no evidence of recurrence and oral hygiene maintenance was adequate. The planned treatment was reemphasized to the patient, but she did not return for it.

Discussion

Gingival overgrowth varies from mild enlargement of isolated interdental papillae to segmental or uniform and marked enlargement affecting 1 or both of the jaws. The purpose of this paper was to report a case of gingival fibromatosis, which was generalized and involved both the jaws. GF may be inherited, in most cases as an autosomal dominant disease or an autosomal recessive disease, or may be caused by a new mutation. In cases where a new mutation may have occurred, with family history showing no evidence of genetic transmission, the diagnosis may be IGF.[6] Our case had a negative family history for GF, as no other member of the family was affected. Hence, the case was diagnosed as IGF, which was probably caused by variable gene expression or a gene mutation.

Enlargement of gingiva is most intense during eruption of the primary and permanent teeth, and minimal or nondetectable growth is observed in adults.[6] Our case presented with gingival enlargement first at 6-years-old, which coincides with the eruption of the permanent teeth, and then recurred at 11-years-old. The condition causes delayed eruption of teeth, diastema, malposition of the teeth, and prolonged retention of primary teeth resulting in esthetic and functional problems. The present case reported to us because she was not able to close her lips, the malposition of her teeth led to impaired esthetics, and she had functional problems in that she was not able to eat because of overgrowth of gingiva. Enlargement seen in IGF appears to be confined to the fibroblasts which harbor in the gingiva. The hyperplastic response does not involve the periodontal ligament and occurs peripheral to the alveolar bone within attached gingiva.[27] The alveolar bone is not affected directly, but gingival enlargement may result in plaque accumulation, which promotes periodontitis and bone resorption. The present case showed severe localized alveolar bone loss distal to the mandibular right first molar, probably due to food accumulation and impaired oral hygiene maintenance as a result of gingival overgrowth. Chaturvedi[28] has also reported a case of nonsyndromic IGF with generalized aggressive periodontitis.

According to many authors, the best time for surgical intervention is when all the permanent dentition have erupted, because the risk of recurrence is higher preceding that.[2,29,30,31] Appropriate time for removal of the enlarged gingiva is 3, 6, or 12-years-old so as to ensure effective plaque control and to maintain oral hygiene after the gingivectomy procedures.[30] Delay in treatment may lead to significant problems such as retention of primary teeth, delay in the eruption of the permanent teeth, difficulties in mastication and phonetics, malposition of teeth, and esthetic and psychological problems. In this case, the treatment was first conducted at 6-years-old and later at 13-years-old, as esthetics was a major concern for the patient due to nonvisibility of teeth. Coletta and Graner[6] have suggested that treatment should be performed once the patient is cooperative and shows good oral hygiene. The local and psychological benefits, even if temporary, must not be underestimated and may outweigh the probability of recurrence.

In the past, extraction of all teeth and reduction of the alveolar bone have been advocated.[31,33-35] Various techniques used for the excision of enlarged gingiva include external or internal bevel gingivectomy in association with gingivoplasty, an apically positioned flap, electrocautery, and carbon dioxide laser.[21,34,35,36,37,38] If carbon dioxide laser is not available, the

most effective method for removing large quantities of gingival tissue, especially when there is no attachment loss and all the pocketing is false, is the conventional, external bevel gingivectomy. Ramer et al.,[21] advocated quadrant-wise gingivectomy with periodontal pack placement for 1 week, followed by 0.2% chlorhexidine oral rinse twice a day for 2 weeks after each surgery. In our case, we followed a conservative treatment that consisted of external bevel gingivectomy of both arches; 1 month later, quadrant-wise gingivoplasty was done in 4 sittings. A 0.12% chlorhexidine oral rinse was prescribed twice a day 2 for weeks following surgery.

Following surgery, recurrence is expected within a few months after surgery. Though this recurrence is unpredictable, it is most commonly seen in children and teenagers rather than adults.[8,36] The patient may have to undergo repeated gingivectomy procedures. In our case, recurrence was seen after approximately 4 years of performing gingivectomy, which was initially performed at 6-years-old. This repeated recurrence often causes a further increase in the patient's and parents' psychological and emotional stress. Hence, psychological counseling is a must for both the patients and parents.[39] Studies have demonstrated that recurrence is faster in areas with plaque accumulation.[37] Emerson,[28] however, found that the degree of enlargement did not appear to be related to the oral hygiene or to the amount of calculus present and that correct physiologic contouring of marginal gingiva is more important. Normally, recurrence is minimal or delayed if good oral hygiene is achieved by a combination of monthly examinations with professional cleansing and oral hygiene instructions. Our patient was given oral hygiene instructions for home maintenance and had been advised to pursue regular follow-up. She failed to return for the periodic recall, however, and returned after 1 year.

Further reconstructive periodontal therapy was planned for the mandibular right first molar, and the procedure was explained to the patient. Camargo and Carranza[40] have advocated an internal bevel gingivectomy with open-flap debridement in cases where gingival enlargement is associated with deep pockets and severe loss of underlying alveolar bone. The same treatment was also followed by Chaturvedi,[38] who reported a case of IGF with generalized aggressive periodontitis. Orthodontic therapy to correct the malocclusion resulting from the gingival overgrowth was also planned, as was a combination of surgical and fixed orthodontics to correct the telescopic bite. The patient, however, did not return for the treatment. She reported back to us after 1 year, at which time her gingival condition was satisfactory with no evidence of recurrence and oral hygiene maintenance was adequate. The planned treatment was reemphasized to the patient, but she did not return for it.

This case report highlights a nonsyndromic incidence of gingival fibromatosis and the need for a systematic approach in its diagnosis and management. We followed the conventional surgical procedure of gingivectomy and gingivoplasty, which resulted in remarkable improvement in esthetics and function. Localized bone loss, an unusual feature, was seen in our case. Maintenance of oral hygiene is of paramount importance not only to prevent recurrence but also to minimize the periodontal involvement that could be associated with food accumulation. Management of such cases is based on a multidisciplinary approach, and psychological counseling plays a very important role in patient management. Though the appropriate time for treatment is typically found to be after the eruption of permanent teeth, when recurrence is found to be minimal, surgical intervention should not be delayed when the patient's developmental, esthetic, and psychological needs outweigh the probability of recurrence.

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