ELEPHANTIASIS GINGIVA IN 13 YEAR OLD BOY- A CASE REPORT

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Abstract

Hereditary Gingival Fibromatosis (HGF) is the most common genetic form of gingival fibromatosis. HGF is characterized by a slowly progressive, non-hemorrhagic, fibrous enlargement of maxillary and mandibular keratinized gingiva. The enlarged gingiva is normal in color, firm in consistency, and histologically benign, the main feature being accretion of mature collagenous connective tissues. The overlying epithelium is typically normal, with occasional areas of hyperplasia, and with rete pegs extending deep into underlying connective tissues. Gingival enlargement may be generalized or localized, either unilateral or bilateral.

A 13-year-old boy reported to the with massive gingival overgrowth. On clinical examination, a generalized gingival overgrowth covering almost entire clinical crowns of the teeth was found. Radiographic examination revealed evidence of anterior teeth bone loss. A provisional diagnosis of hereditary gingival fibromatosis was done after history, clinical & radiographical examination. Full mouth gingivectomy was carried out and tissue was sent for histopathological examination and final diagnosis was made. Patient was followed up for a period of 2 year and no recurrence was detected.

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Introduction

Hereditary gingival fibromatosis (HGF) is a benign, idiopathic condition affecting both arches. It effects males and females equally and is usually autosomal-dominant[1]. The gingiva is markedly enlarged, asymptomatic, nonhemorrhagic, nonexudative. It may be an isolated finding or associated with other syndromes[2].

HGF develops as a slowly progressive, benign, localized or generalized enlargement of keratinized gingiva that, in severe cases, may cover the crowns of the teeth. Localized forms of HGF usually affect the maxillary tuberosities and the labial gingiva around the mandibular molars. Since HGF has not been reported in edentulous patients, it appears that the presence of dentition is necessary for overgrowth to develop[3].

A relationship with growth hormone deficiency has been suggested. The condition is either of a nodular form or, more commonly, a symmetric form. Onset of the condition usually begins with eruption of the permanent teeth.

This condition predisposes one to malpositioning of the teeth, retention of deciduous teeth, esthetic and functional problems[4].

It is very rare cases affects 1 in750, 000 people with varying intensity and expressivity even in individuals within the same family [5,6]

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Case Report

A 13 year old boy patient reported to Department of Periodontology, Rungta College of Dental sciences & Research Bhilai, with a chief complaint of slowly growing gingival growth present in the upper and lower teeth region, since many years.

History revealed that the overgrowth began slowly, and covering entire surface of crown. The patient was in good systemic health, medical and dental history was non-contributory.

Intra oral examination revealed generalized severe enlargement of gingiva involving both the mandibular and maxillary arches (Figure 1 to Figure 6).

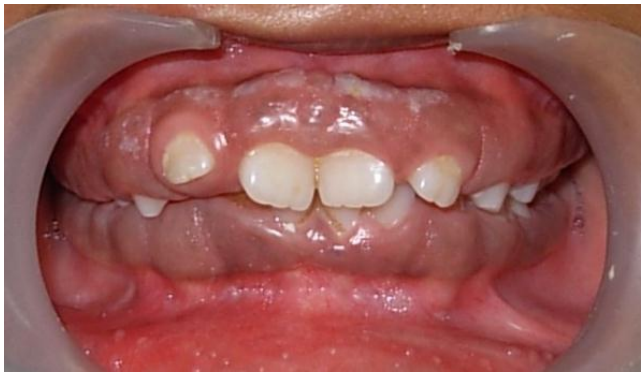

Figure 1. Preoperative frontal view.

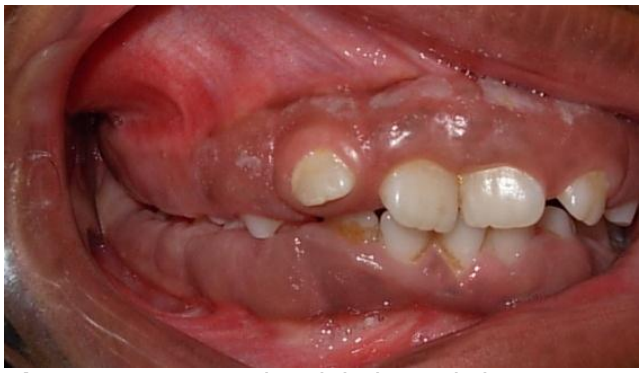

Figure 2. Preoperative right buccal view.

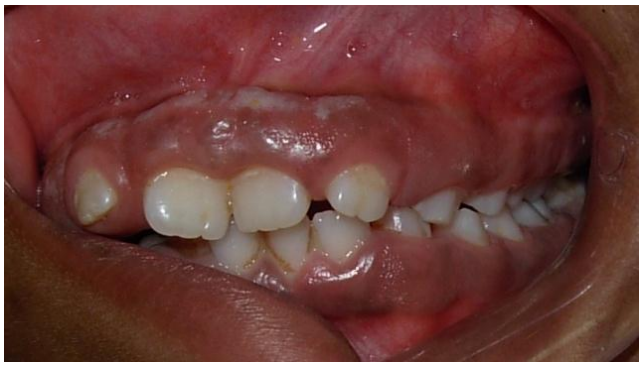

Figure 3. Preoperative left buccal view.

The gingiva was pink, and its firm, dense, fibrous consistency caused considerable difficulty with plaque removal. The presence of an overgrowth covering entire surface of exposed teeth reaching occlusal surface interfering occlusion. The patient was unhappy with the appearance of her gingiva. His medical history is noncontributory but family history was of significance, since his sister had a similar gingival condition. He reveled that his mother had had several gingival surgeries because of slowly progressing hyperplasia. He also stated that his sister not had any type disorder, nor had she taken any medications associated with gingival hyperplasia. On the basis of history and clinical presentation, a provisional diagnosis of 'hereditary gingival overgrowth' was made and a gingivectomy was planned.

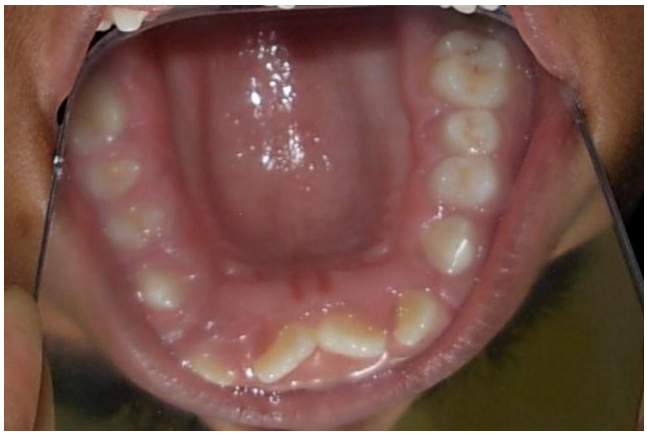

Figure 4. Preoperative upper occlusal view.

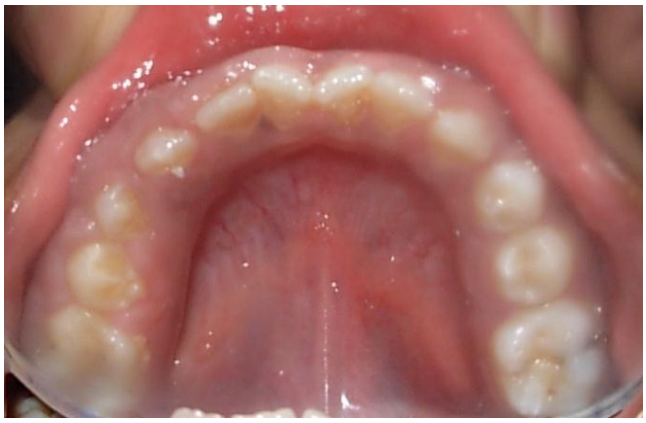

Figure 5. Preoperative lower occlusal view.

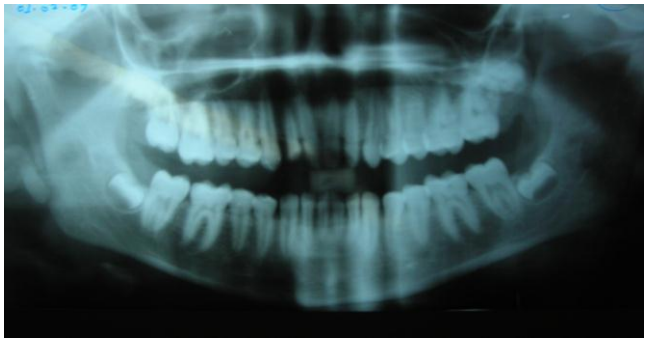

Figure 6. Pretreatment orthopantamogram.

In this case, treatment was carried quadrant-by-quadrant gingivectomy. The patient was operated under 2% Xylocaine HCl with 1:80,000 Adrenaline, after securing profound anesthesia the bleeding points were created by using crane Kaplan pocket marker(Fig.7), then excessive gingival tissue was removed by using gingivectomy knives(Kirkland knife, Orbans interdental knife) (Figure 8, Figure 9), the area was thoroughly irrigated with normal saline and the surgical wound was covered with the periodontal dressing for one week, followed by 0.2% chlorhexidine oral mouthwash twice a day for a week after each surgery. Antibiotics and analgesics were prescribed and the patient was discharged after giving him post operative instructions.

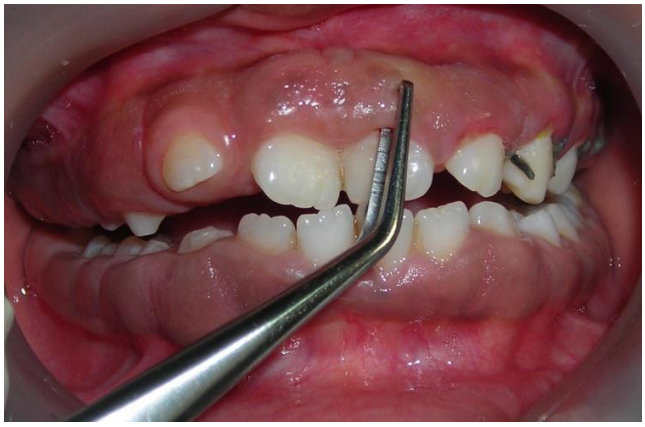

Figure 7. Bleeding points marked with pocket marker.

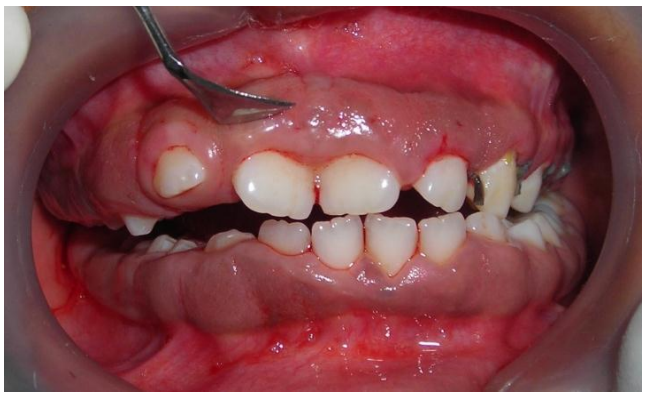

Figure 8. Incision being placed with Kirkland knife.

The histopathological report read as “The H & E stained section shows acanthotic stratified squamous epithelium with elongated rete pegs. There is a bulbous increase the amount of a vascular connective tissue densely arranged collagen bundles and plump fibroblasts were seen focally (Figure 10). Considering the history, clinical radiographic features and microscopic picture, a final diagnosis of ‘Hereditary Gingival Fibromatosis’ was made.

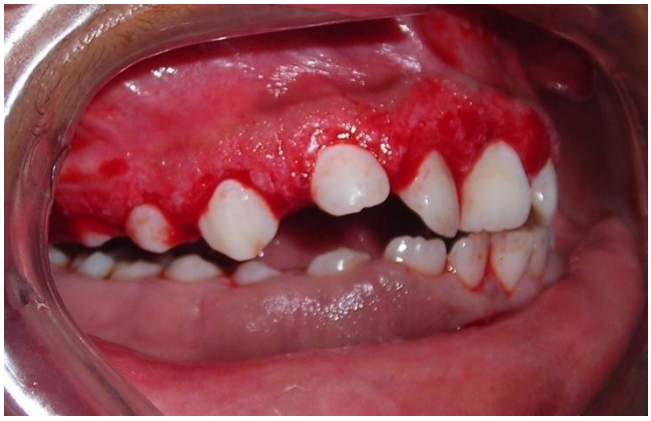

Figure 9. After gingvectomy.

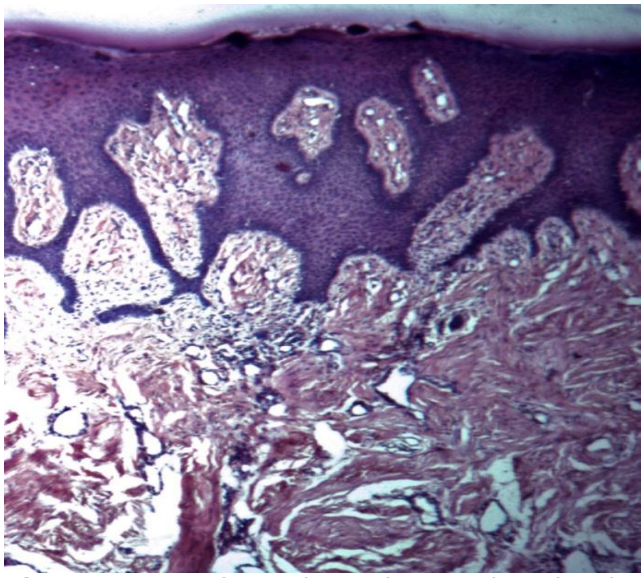

Figure 10. The photomicrograph showing hyperkeratotic stratified squamous of variable thickness with irregular rete ridges, highly fibrous connective tissue with dense collagen bundles arranged in haphazard manner with numerous fibroblasts. (HE, X100).

Patient reported after one week when the pack was removed and the area was thoroughly irrigated with saline and betadine. The oral hygiene was reinforced and patient was called for checkups at regular intervals. After eighteen months observation, Scaling and prophylaxis were performed every six months. So far there was no evidence of recurrence (Figure 11 to Figure15).

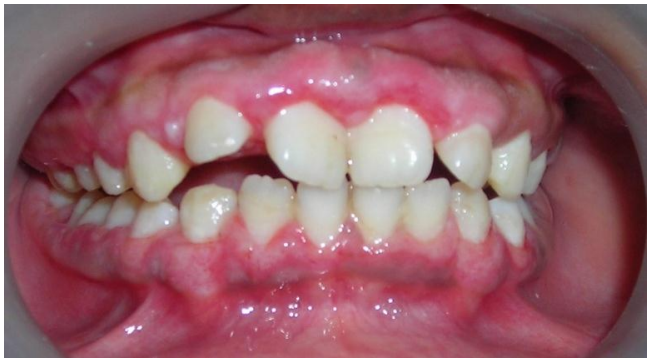

Figure 11. Post operative frontal view.

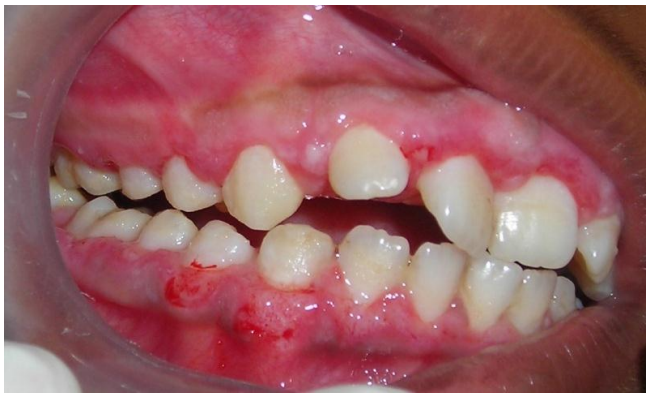

Figure 12. Post operative right buccal view.

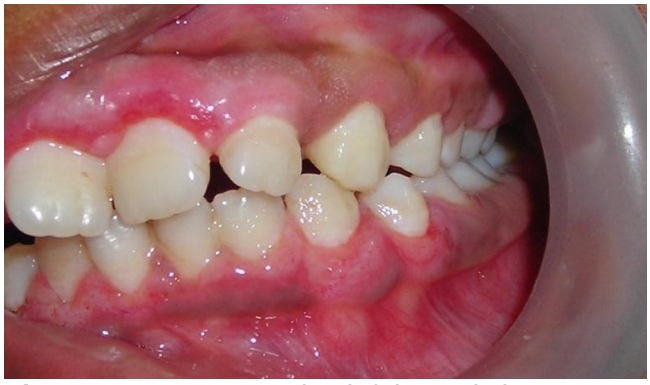

Figure 13. Post operative left buccal view.

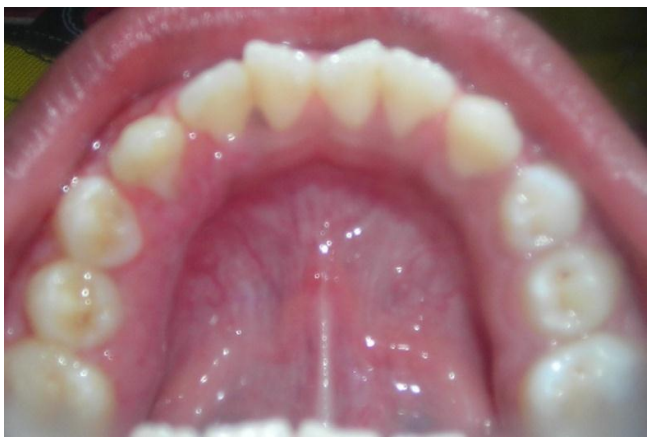

Figure 14. Post operative upper occlusal view.

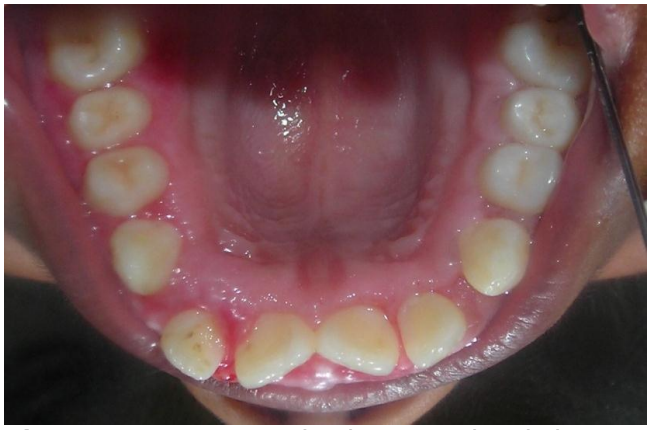

Figure 15. Post operative lower occlusal view.

Discussion

HGF is a rare condition, and, therefore, most information about its characteristics is based on case reports. Generalized gingival fibromatosis can be caused by a number of factors, including inflammation, Leukemic infiltration and medication use such as phenytoin, cyclosporine or nifedipine[7,8].

Gingival tissue enlargement usually begins with the eruption of the permanent dentition but can develop with the eruption of the deciduous dentition; it rarely is present at birth. The most extensive enlargement appeared to occur either during loss of the deciduous teeth or in early stages of eruption of the permanent dentition. He noted that the enlargement seems to progress rapidly during "active" eruption and decrease with the end of this stage[5].

Gingival fibromatosis may exist as an isolated finding or as part of a more general syndrome. It is possible that isolated gingival fibromatosis may result from a single gene mutation, while syndromic forms may result from alterations of multiple genes[9].

Gingival fibromatosis can occur as part of a syndrome. It has been reported as a feature of Murray- Puretic Drescher syndrome (multiple hyaline fibromas), Rutherfurd's syndrome, Laband syndrome Cross syndrom. Recently Wynne and colleagues reported a new syndrome of HGF occurring with hearing deficiencies, hypertelorism and supernumerary teeth.[10]

Hereditary gingival fibromatosis associated with generalized aggressive periodontitis.[11]

In addition to heritable forms, several classes of pharmacological agents (calcium

channel blockers, cyclosporin, and phenytoin) have been associated with the development of gingival hypertrophy. Yet, not everyone exposed to these medications develops gingival overgrowth, suggesting that genetic factors may underlie a differential propensity to develop drug-induced gingival overgrowth. Although genetic factors appear to be important in several types of gingival fibromatosis, few of the responsible genes have been identified, and none has been identified in isolated HGF[12].

Recent studies about the genetic characteristics of HGF have provided novel clues about the potential pathogenic mechanisms. Genetic loci for autosomal dominant forms of HGF have been localized to chromosome 2p21-p22 (HGF1) and chromosome 5q13-q22 (HGF2)[13, 14]. AND mutation in the Son of sevenless–1 (SOS1) gene has been identified in affected individuals.[15]

The best time to initiate treatment to HGF is when all of the permanent dentition has erupted because the risk of recurrence is higher before it. Treatments vary according to the degree of severity of gingival enlargement. When the enlargement is minimal, thorough scaling of teeth and home care may be sufficient. However, excessive gingival tissue and esthetic and functional impairment dictate the need for surgical intervention.[16,17]

In this case, HGF showed moderate hyperplasia of a dense, hyperkeratotic epithelium with elongated rete ridges these findings are similar to other reports.[18,19]

Conclusions

Hereditary gingival fibromatosis is a relatively rare entity, cannot be cured but can be controlled with varying degrees of success. When the enlargement is minimal, good scaling of teeth and home care may be all that is required to maintain good oral health. As the excess tissue increases, appearance and function indicate need for surgical intervention.

Declaration of Interest

The authors report no conflict of interest and the article is not funded or supported by any research grant.

References

1. Jorgenson RJ, Cocker ME. Variation in the inheritance and expression of gingival fibromatosis. J Periodontol 1974; 45: 472-477.
2. Hart TC, Pallos DL, Bozzo OP, et.al. Evidence of genetic heterogeneity for hereditary gingival fibromatosis. J Dent Res 2000; 79; 1758.
3. Flaitz CM, Coleman GC. Differential diagnosis of oral enlargement in children. Pediatr Dent 1995; 17: 294-300.
4. Raeste AM, Collan Y, Kilpinen E. Hereditary fibrous hyperplasia of the gingiva with varying penetrance and expressivity. Scand J Dent Res1978; 86:357–365.
5. Fletcher JP. Gingival abnormalities of genetic origin: preliminary communication with special reference to hereditary gingival fibromatosis. J Dent Res 1966; 45:597-612.
6. Avelar RL, De Luna Campos GJ, Falcao PG, et al. Hereditary gingival fibromatosis: a report of four cases in the same family. Quintessence Int 2010; 41(2):99-102.
7. Seymour RA, Heasman PA. Drugs and the periodontium. J Clin Periodontol 1988; 15:1-16.
8. Hakkinen L, Csiszar A. Hereditary Gingival Fibromatosis: Characteristics and Novel Putative Pathogenic Mechanisms. J Dent Res2007; 86(1):25-34.
9. Fryns JP. Gingival fibromatosis and partial duplication of the short arm of chromosome 2 (dup (2) (p13>p21)). Ann Genet1996; 39:54-55.
10. Wynne SE, Aldred MJ, Bartold PM, et al. Hereditary gingival fibromatosis with hearing loss and supernumerary teeth-a new syndrome. J Periodontol; 66(1):75-79.
11. Casavecchia P, Uzel MI, Hart TC, et al. Hereditary gingival fibromatosis associated with generalized aggressive periodontitis: a case report. J Periodontol 2004; 75:770-778.
12. Shashi V, Pallos D, Pettenati MJ, et al. Genetic heterogeneity of gingival fibromatosis on chromosome 2p. J Med Genet 1999; 36:683-686.
13. Xiao S, Bu L, Zhu L, Zheng G, et al. A new locus for hereditary gingival fibromatosis (GINGF2) maps to 5q13-q22.Genomics 2001; 74:180-185.
14. Ye X, Shi L, Yin W, et al. Further evidence of genetic heterogeneity segregating with hereditary gingival fibromatosis. J Clin Periodontol.2009; 36(8):627-33.
15. Thomas CH, Yingze Z, Michael CG, et al. A Mutation in the SOS1 Gene Causes Hereditary Gingival Fibromatosis Type -I. Am J Hum Genet 2002; 70(4): 943–954.
16. Ramakrishnan T, Kaur M. Multispeciality Approach in the Management of Patient with Hereditary Gingival Fibromatosis: 1-Year Followup: A Case Report. Int Journal Dentistry 2010; 1-5.
17. Lobao DS, Silva LC, Soares RV. Idiopathic gingival fibromatosis: a case report. Quintessence Int. 2007; 38(8):699-704.
18. Doufexi A, Mina M, Ioannidou E. Gingival overgrowth in children: epidemiology, pathogenesis, and complications. A literature review. J Periodontol 2005; 76:3-10.
19. Araujo CS, Graner E, Almeida OP, et.al. Histomorphometric characteristics and expression of epidermal growth factor and its receptor by epithelial cells of normal gingiva and hereditary gingival fibromatosis. J Periodontal Res 2003; 38:237-241.