

Hereditary Gingival Fibromatosis: A Case Report

¹Seyed Reza Arabi, ¹Zahra Ebrahimzade, ²Kadkhodazadeh Mahdi and ¹Soulmaz Amani Jourdehi

¹Department of Periodontics, School of Dentistry,
Hamedan University of Medical Science, Hamedan, Iran

²Department of Periodontics, School of Dentistry,
Shahid Beheshti University of Medical Science, Tehran, Iran

Abstract: Hereditary gingival fibromatosis is slowly progressive enlargement of gingival characterized by a generalized enlargement of the buccal and lingual aspects of the attached and marginal gingiva. A case of 14 years old female is reported who has generalized moderate to severe gingival overgrowth of the maxillary and mandibular arches. The diagnose was based on clinical, radiographic and familial history. The treatment included gingivectomy combined with open flap debridment.

Key words: Gingival enlargement, gingivectomy, hereditary gingival fibromatosis, flap debridment, primary dentition, Iran

INTRODUCTION

Hereditary gingival fibromatosis is rare, benign, non-inflammatory fibrotic enlargement of maxillary or mandible arches associated by familial aggregation also known as gingivomatosis elephantiasis, familial elephantiasis, Juvenile hyaline fibromatosis, congenital familial fibromatosis, idiopathic fibromatosis, idiopathic gingival fibromatosis and hereditary gingival hyperplasia (Casavecchia *et al.*, 2004; Tavargeri *et al.*, 2004; Lindhe *et al.*, 2008). The etiology and pathogenesis of this rare condition has not been established but it could be because of these three factors; individual susceptibility, local factors (dental plaque, caries, iatrogenic factors) and the action of chemical substances and their metabolites (Rajesh *et al.*, 2006). It seems to be a slowly progressive keratinized gingival overgrowth with various degree (Rajesh *et al.*, 2006; Baptista, 2002) and can be localized or generalized (Lindhe *et al.*, 2008). On histopathological feature of fibromatosis include increase amount of mature collagenous connective tissue and mild epithelial hyperplasia (Clochert *et al.*, 2003). Gingival fibromatosis can happen as an inherited condition (Rajesh *et al.*, 2006). Most recently, mutation in the son of sevenless-1 gene has been concentrate as a genetic factor responsible for hereditary gingival fibromatosis (Hart *et al.*, 2002). Also can inherited as mainly autosomal dominant (Tavargeri *et al.*, 2004). Where the medical and drug histories did not cause gingival enlargement and present of familial history. It was diagnoses as hereditary gingival fibromatosis. It tends to happen more often as a

generalized type (Anderson *et al.*, 1969). Enlargement commonly grows with eruption of the permanent dentition but can also begins with the eruption of the deciduous dentition; seldom it may occurred at birth or arise in adulthood. The age of beginning is divided into the pre-eruptive period (<6 months), deciduous dentition period (6 months and 6 years), mixed dentition period (6-12 years), permanent dentition period after adolescence (20 years). Maximal enlargement happen either during the loss of deciduous teeth or in the early stages of eruption of permanent teeth if progresses fast while there is active eruption and decrease with the end of this stage (Jorgenson, 1971). The hyperplastic tissue reveals usually a normal pink color. Enlargement can be generalized or localized to specific areas of the mouth for example the maxillary tuberositis and the labial gingiva around the molars of lower arch. Severity can alter from involvement of one quadrant to severe involvement of all four quadrants (Rajesh *et al.*, 2006). A pink color and generalized enlargement including all four quadrants was diagnosed in the patient. There are different procedure for treatment of Gingival Fibromatosis (GF) and it involves removed of GF by surgery, electrocautery and use of carbon dioxide laser. If carbon dioxide laser is not valid, the conventional external bevel gingivectomy is the most effective method for removing large quantities of gingival tissue, especially when there is no attachment loss and all the pocketing is false (Takagi *et al.*, 1991). Presence of teeth looks important in this condition as because it is common recurrence of enlargement after surgery and permanent remodeling of tissue happen after extraction of

the teeth. This disease can have inheritance pattern such as simple Mendelian trait in some chromosomal disorders and as malformation syndrome (Lindhe *et al.*, 2008). In this study, researchers present a case of hereditary gingival fibromatosis with clinical and radiographic features. The treatment included gingivectomy with open flap debridement.

CASE REPORT

A 14 years old girl accompanied by her parent to the Department of Periodontics in Faculty of Dentistry University of Hamedan Medical Sciences with the chief complain of gingival enlargement, poor esthetic and difficulty in chewing function. She did not have any history of fever, prolonged medication, anorexia, weight

loss, seizures or hearing loss and any history of physical or mental disorder and syndrome sign. It was noticed by patient when she was 10 years old. Her father also was gummy smile and had short clinical crown in his anterior upper teeth because of gingival enlargement. On intraoral examination patient shows generalized, obese, nodular, diffuse enlargement of gingiva on both maxillary and mandibular arches which were pink in color, firm and fibrous consistency. The crowns of the teeth at the upper right sextant were barely visible because they were interred deep within the enlarged gingiva (Fig. 1). Full mouth periapical radiographs display no abnormality in number, size and structure of the teeth and there was no alveolar bone loss and missing tooth and her four wisdom teeth was not erupted and tooth number 20 and 29 was extracted (Fig. 2). Hematological examination reveals all



Fig. 1: Photographs before surgery



Fig. 2: Full mouth intraoral x ray



Fig. 3: About 6 months after surgery

parameter were within normal limits on the basis of medical, familial and drug histories and the clinical finding. It was diagnosed as hereditary gingival fibromatosis. Gingivectomy with open flap debridement was performed by sextant except the mandibular lower incisors under local anesthesia to restore esthetic, functional and masticatory needs of the patient.

The post operative course was not eventful and post surgical follow up at 6 month shows no recurrence (Fig. 3).

CONCLUSION

The gingival enlargement in GF is one of the factors that cause food excursion, collects food debris and irritating plaque be composed resulting in bone loss and root resorption.

The suitable time for removal of GF varies. Emerson (1965) suggested the appropriate time is at the ages of 3-6 and 12 years. Oral hygiene and super imposition of plaque accumulation have critical affect on progression of GF.

The treatment is surgical removal often in a series of gingivectomies but relapse are not unusual if the volume of overgrowth is extensive, a repositioned flap to prevent exposure of connective tissue by gingivectomy can better achieve elimination of pseudo-pocket (Lindhe *et al.*, 2003). Also if surgery was done after eruption of permanent teeth, the tendency to reappear is minimal (Lee and Donnell, 2003). May be these reasons are responsible to prevent relapse in the case after 6 months.

REFERENCES

- Anderson, J., W.J. Cunliffe, D.F. Roberts and H. Close, 1969. Hereditary gingival fibromatosis. *Br. Med. J.*, 3: 218-219.
- Baptista, I.P., 2002. Hereditary gingival fibromatosis: A case report. *J. Clin. Periodontol.*, 29: 871-874.
- Casavecchia, P., M.I. Uzel, A. Kantarci, H. Hasturk, S. Dibart, T.C. Hart, P.C. Trackman and T.E. Van Dyke, 2004. Hereditary gingival fibromatosis associated with generalized aggressive periodontitis: A case report. *J. Periodontol.*, 75: 770-778.
- Clochert, K., C. Dekeyser, C. Carels and G. Willems, 2003. Idiopathic gingival hyperplasia and orthodontic treatment: A case report. *J. Orthod.*, 30: 13-19.
- Emerson, T.G., 1965. Hereditary gingival hyperplasia. A family pedigree of our generation. *Oral. Med. Oral. Pathol.*, 19: 1-9.
- Hart, T.C., Y. Zhang, M.C. Gorry, P.S. Hart and M. Cooper *et al.*, 2002. A mutation in the *SOS1* gene causes hereditary gingival fibromatosis type 1. *Am. J. Hum. Gen.*, 70: 943-954.
- Jorgenson, R.J., 1971. Gingival fibromatosis. *Birth Defects Orig. Artic Ser.*, 7: 728-780.
- Lee, W. and D. O'Donnell, 2003. Severe gingival hyperplasia in a child with I-cell disease. *Int. J. Paediatr. Dent.*, 13: 41-45.
- Lindhe, J. and N.P. Lang and T. Karring, 2008. *Clinical Periodontology and Implant Dentistry: Clinical Concepts*. 5th Edn., Blackwell Munksgaard, New York, ISBN: 9781405160995, Pages: 413.

- Lindhe, J., N.P. Lang and T. Karring, 2003. *Clinical Periodontology and Implant Dentistry*. 4th Edn., Blackwell Publishing Ltd., Oxford, UK., ISBN: 9781405102360, pp: 276.
- Rajesh, T.A., A.N. Ullal and P. Joby, 2006. Idiopathic gingival fibromatosis: A case report. *Hong Kong Dent. J.*, 3: 53-57.
- Takagi, M., H. Yamamoto, H. Mega, K.J. Hsieh, S. Shioda and S. Enomoto, 1991. Heterogeneity in the gingival fibromatosis. *Cancer*, 68: 220-22.
- Tavargeri, A.K., S.S. Kulkarni and P. Sudha, 2004. Idiopathic gingival fibromatosis. A case report. *J. Indian Soc. Pedo. Prev. Dent.* December., 2004: 180-182.