

## Idiopathic Gingival Fibromatosis

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### Abstract

Gingival hyperplasia is a heterogenous division of disorder characterized by increasing enlargement of the gingiva caused by a raise in submucosal connective tissue elements. Idiopathic gingival fibromatosis is a circumstance of undecided origin which can expand as an lonely disorder but frequently it is associated with a number of syndrome. It usually begins at the time of eruption of everlasting teeth but can build up with the upsurge of deciduous dentition and hardly ever present at birth.

**Keywords:** idiopathic, gingival, fibromatosis, gingivectomy, nitrendipine

### 1. Introduction

Gingival enlargements are quite common and may be either inflammatory, non-inflammatory or a combination of both. Idiopathic gingival hyperplasia is a rare condition of undetermined etiology described variously as fibromatosis gingivae, gingivaematis [1]. Hereditary gingival fibromatosis [2], idiopathic fibromatosis [3]. Familial elephantiasis [4] and diffuse fibroma [5]. Diffuse gingival enlargement is also found to be associated with syndromes like Cross syndrome, Rutherford syndrome, Ramen syndrome, Zimmerman Laband syndrome and Juvenile hyaline syndrome.

### 2. Discussion

Idiopathic gingival fibromatosis may be congenital or hereditary. Though the genetic mechanism is not well understood, the majority of the authors of reported cases attributed the condition to hereditary factors. The mode of transmission is mainly autosomal dominant [6, 7]. The first polymorphic marker for hereditary gingival fibromatosis (HGF) phenotype in chromosome 2p21. Many cases are sporadic with no familial background.

Gingival hyperplasia can occur after therapy with drugs like phenytoin, [8] cyclosporine, nifedipine and nitrendipine. Long term use of these drugs has to be ruled out. The incidence of gingival enlargement caused by phenytoin, an anticonvulsant used in the treatment of epilepsy varies from 3 to 84.5%. whereas, cyclosporine a fairly potent immunosuppressive agent used to prevent organ transplant rejection and to treat several disease of autoimmune origin induced gingival enlargement in 30% of the cases [9] Nifedipine, which is a calcium channel blocker used in the treatment of acute and chronic coronary insufficiency, including angina pectoris and refractory hypertension and nitrendipine an analogue of nifedipine have also been reported to induced gingival enlargement [10, 12]

The condition may be associated with physical developmental retardation and hypertrichosis [12]. Although gingival tissue may appear normal at birth, hyperplastic gingival fibromatosis may become evident with the

eruption of primary or permanent dentition, suggesting a trauma induced tissue reaction during the eruption [13].

Sometimes gingival enlargement does not occur until the eruption of the permanent dentition. Further enlargement does not occur once the growth of jaw is completed [14]. It has been suggested that gingival enlargement may be due to nutritional and hormonal factors, but these have not been completely substantiated. The constant increase in the tissue mass can result in delayed eruption and displacement of teeth, arch deformity, spacing and migration of teeth [15].

The condition is not painful until the tissue enlarges to partially cover the occlusal surface of the molars and become traumatized during mastication, which was observed in the present case. Due to massive gingival enlargement, an affected child usually develops abnormal swallowing pattern and experiences difficulty in speech and mastication. Along with these features, there may be some interference with the oral hygiene measures and normal mastication. All these will favor accumulation of materia alba and plaque, which further complicates the existing hyperplastic tissue. Maintenance of good oral hygiene is very important. It is not known if plaque control measures are effective in this condition, but it is good practice to maintain the plaque control following gingivectomy procedure.

Histologically, the gingival hyperplasia is mainly due to an increase and thickening of mature collagen bundles in the connective tissue stroma [16]. The nodular appearance can be attributed to the thickened para hyperkeratinized epithelium [17]. Various modalities of treatment had been proposed including radical treatment with extraction of the involved teeth, which was reported not to favor a recurrence of the growth. The only treatment of choice in this condition was gingivectomy to satisfy the patient's esthetics. Though the tissue appeared to be pale and firm, the surgical procedure was complicated with excessive hemorrhage. Some have reported a case where apically positioned flap surgery and CO laser evaporation were used to reduce the gingival tissue. Since recurrence could be expected within a few months after surgery and may return to the original condition within few years, the

patient may have to undergo repeated gingivectomy procedures. This often causes further increase in the patients and parents' psychological and emotional stress. Hence psychological counselling is a must for patients and parents.

### 3. References

1. Ball EI. Case of gingivomatosis or elephantiasis of the gingiva. *J Periodontol.* 1941; 12:26.
2. Laskin J, Weisberger D. Hereditary gingival fibromatosis. *J Oral Surg* 1961; 14:828.
3. Thukral PP. Idiopathic hyperplasia. *J Ind Dent Assoc.* 1972; 44:109.
4. Raman Y, Berman, Bubbis JJ. Gingival fibromatosis combined with cherubism. *Oral Surg.* 1967; 24:455.
5. Buckner HJ. Diffuse fibroma of the gums. *J Am Dent Assoc.* 1937; 27:2003.
6. Emerson TG. Hereditary gingival hyperplasia. A family pedigree of four generations. *Oral Surg.* 1965; 19:1.
7. Jorgenson RJ, Cocker ME. Variations in the inheritance and expression of the gingival fibromatosis. *J Periodontol.* 1974; 45:472-477.
8. Angelopoulos AP, Goaz PW. Incidence of diphenylhydantoin gingival hyperplasia. *Oral Surg.* 1972; 34:898.
9. Seymour RA, Smith DG, Rogers SR. Comparative effect of azathioprine and cyclosporine on some gingival health parameters of renal transplant patients. *J Clin Periodontol.* 1987; 14:610.
10. Barclay S, Thomason JM, Idle JR, Seymour RA. Incidence and severity of nifedipine induced gingival overgrowth. *J Clin Periodontol.* 1992; 19:311.
11. Brown RS, Sein P, Corio R, Bottomley WK. Nitrendipine - induced gingival hyperplasia. *Oral Surg.* 1990; 70:593.
12. William G Shafer, Maynard K Hine, Bernat M Levy. Developmental disturbances of the perioral structures. In: *Text book of Oral Pathology.* 4 ed. A Prisma Indian. 1993, 23-24.
13. Gupta N, Maheshwari S. Advanced gingival fibromatosis. *J Ind Dent Assoc.* 1996; 167:46-47.
14. Stewart RE. Periodontal diseases in children. In: *Pediatric Dentistry and Clinical Practice.* Mosby Company, 1982, 623-639.
15. McDonald RE, Avery DR. Gingival and periodontal diseases. In *Dentistry for the Child and Adolescent.* 7th ed. Mosby Company, 2000, 452-453.
16. Zachin SJ, Weisberger D. Hereditary gingival fibromatosis - report of a family. *Oral Surg Oral Med Oral Pathol.* 1961; 14:825-835.
17. Brightman VJ. Benign tumors of the oral cavity including gingival enlargement. In *Burkett's of Oral Medicine, Diagnosis and Treatment Plan.* 8 ed. 1984, 367-371.