International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: 6.614
Available Online at www.journalijcar.org
Volume 7; Issue 7(I); July 2018; Page No. 14447-14448
DOI: http://dx.doi.org/10.24327/ijcar.2018.14448.2620

Case Report

A CASE REPORT AND REVIEW OF LITERATURE OF HEREDITARY GINGIVAL FIBROMATOSIS

Mansi Tiwari, Sen[1]., Anshul Sharma[2]., Abhijeet Deoghare[3]., Pramod Krishna B[4]., Rajdeep Singh[5] and Neha Agarwal[6]

[1,3]Department of Oral Medicine, C.D.C.R.I. Rajnandgaon, Chhattisgarh
[2,4,5]Department of Oral Surgery, C.D.C.R.I. Rajnandgaon, Chhattisgarh
[6]Departemnt of Endodontics, N.H.C.D.R.I Bilaspur, Chhattisgarh

A R T I C L E I N F O

Article History:

Received 7th April, 2018
Received in revised form 16th May, 2018 Accepted 3rd June, 2018
Published online 28th July, 2018

Key words:

Hereditary gingival fibromatosis, gingival enlargement, idiopathic gingival fibromatosis.

A B S T R A C T

Hereditary Gingival Fibromatosis (HGF) also referred to as idiopathic gingival hyperplasia is a rare type of slow growing gingival enlargement. HGF can present as an isolated entity or as part of a syndrome, the affected gingiva is characterized by firm, asymptomatic, nonhemorrhagic enlarged (hyperplastic) tissue with characteristic pale pink colour, covering most of the anatomic crown, involving usually all the quadrants. Pathogenesis of this disease is unclear. Gingivectomy and maintaining good oral hygiene is the treatment of choice in these cases.

INTRODUCTION

Hereditary gingival fibromatosis is a rare disease which affects 1 in 750,000 population. It is thought to be benign condition which is characterized by firm, enlarged gingival tissues that cover most of the anatomic crowns. It was recognized probably more than a century ago, the first case was reported by Gross in 1856.[1]

The onset of disease is concurrent with the eruption of permanent teeth. The excessive growth of gingival tissue may cause displacement of teeth, overretention of primary teeth, and spacing. Both autosomal dominant and autosomal recessive modes of inheritance have been reported.[2] Recently, Son-of- sevenless (SOS-1) has been identified as the prime etiology for non-syndromic HGF. SOS-1 is a guanine nucleotide-exchange factor that functions in the transduction of signals that control cell growth and differentiation[3]. Hereditary gingival fibromatosis can occur as a solely manifestation affecting the gingiva only, with no other local or systemic involvement or is associated with certain syndromes.

Corresponding author:* **Mansi Tiwari, Sen
Department of Oral Medicine, C.D.C.R.I. Rajnandgaon, Chhattisgarh

Examples of syndromic GF are Zimmerman-Laband syndrome (GF, hypoplastic distal phalanges, hepatosplenomegaly, epilepsy, hypertrichosis, and mental retardation), Jones syndrome (GF and progressive neural deafness), Klippel-Trenaunay syndrome (GF, hemihypertrophy, Nevus flammeus, hemangioma, hypertlorism, and macrocephaly), Ramon syndrome (GF, hypertrichosis, mental retardation, epilepsy, rheumatoid arthritis, and diabetes mellitus), Rutherfurd syndrome (GF, unerupted teeth, corneal dystrophy, and mental retardation), and Cross syndrome (GF, nanophthalmos, microcornea, and severe mental retardation).[4]

Hereditary gingival fibromatosis should also be differentially diagnosed from a number of diseases which are listed below in the table.

Generalized symmetric enlargements	Generalized nodular enlargements
• Plasma cell gingivitis	• Neurofibromatosis (type 1 and 2)
• Plaque induced gingival hyperplasia	• Cowden's syndrome
• Drugs (phenytoin, cyclosporine, and calcium channel blockers) induced gingival enlargement	• Gardner's syndrome
	• Tuberous sclerosis
• Scurvy	
• Leukemia (Acute myeloid leukemia)	
• Oro-facial granulomatosis	
• Crohn's disease	
• Sarcoidosis	
• Wegener's granulomatosis	
• Amyloidosis	
• Sturge Weber syndrome	

Hereditary gingival fibromatosis can be diagnosed by its peculiar gingival features (with or without involvement of associated syndromes) and positive family history.

The classical histopathological features of hereditary gingival fibromatosis are highly fibrous connective tissue, with haphazardly arranged dense collagen bundles, numerous spindle shaped fibroblasts, and connective tissue that is relatively avascular. Thickened, acanthotic and hyperkeratotic stratified squamous epithelium is also present with elongated rete ridges. Superficial layers of epithelium may occasionally show features of inflammation (edema).

Case Report

A 43 year old male patient reported in the Department of oral medicine and radiology with the chief complaint of swollen gums since 2 years. Swelling was not associated with pain. The swelling began 2 years earlier and caused difficulties in speaking and eating and he also had obvious implications for his aesthetic appearance. Patient had no history of using drugs i.e. phenytoin, nifedipine, or cyclosporine which are notorious in producing gingival enlargement. Patient gave positive family history that his mother was also having similar problem when she was alive.

Intra-oral examination of the patient revealed generalized enlargement of the gingival on both arches with pale pink colour and firm fibrous consistency with moderate stains and calculus, enlargement was having nodular pattern of and it was covering most of the teeth to the incisal and occlusal surfaces with a firm to fibrotic consistency. Periodontal assessment revealed extensive mobility with 13, presence of deep pockets and moderate calculus deposits with bleeding on probingin certain areas. Pathological migration was seen in maxillary and mandibular anteriors. The patient's medical history did not reveal any pathological condition. The radiographic findings, which corroborated those of the clinical examination, revealed severe generalized alveolar bone loss.

Histopathological examination revealed bulbous increase in the connective tissue, which was relatively avascular and had densely arranged collagen-fiber bundles, numerous fibroblasts, and mild chronic inflammatory cells. The overlying epithelium exhibited hyperplasia and had elongated rete ridges suggesting histological diagnosis of fibroepithelial hyperplasia.

A treatment plan was devised for the patient, which included complete scaling and root planning followed by full-mouth gingivectomy.

DISCUSSION

Gingival enlargement, either localized or generalized might be attributed to a number of reasons, ranging from inflammation, leukemic infiltration, and association with use of medicines like phenytoin, cyclosporine, and nifedipine etc.[5]

HGF is transmitted as either autosomal dominant or recessive. We justify our diagnosis of the gingival fibromatosis as hereditary and as an autosomal dominant, solely relying on the positive family history given by the patient and further corroborated by histopathological findings.

HGF can be present along with other abnormalities in the form of certain syndromic associations. Most of the associated syndromes may include epilepsy, deafness, hypertrichosis, mental retardation along with hereditary gingival fibromatosis. Also, some isolated generalized gingival lesions such as Plasma cell gingivitis, Plaque induced gingival hyperplasia, Scurvy; may show generalized symmetric gingival enlargements. These conditions can be differentiated from hereditary gingival fibromatosis on the basis of medical history, clinical examination and histopathalogical findings.

The side effects of gingival enlargement in HGF include speech problems, painful mastication, spacing and diastema, malocclusion, and overretention of primary teeth. In our patient, gingival enlargement had led to spacing especially between upper anterior teeth. In order to address patients' esthetic and functional needs usually surgical excision of the enlarged tissue is necessary.[6]

Surgical excision of enlarged gingival tissues is the treatment of choice in this condition. Numerous treatment modalities have been employed for the excision of the enlarged gingival tissues, including of conventional surgery, electrosurgery, an apically positioned flap and lasers.[7] Since the enlargement recurs to a various extent, repeated gingivectomies are needed for restoring the gingival contours.

References

1. Fletcher J. Gingival abnormalities of genetic origin: a preliminary communication with special reference to hereditary generalized gingival fibromatosis. *J Dent Res.* 1966;45:597-612
2. G. H. Breen, R. Addante, and C. C. Black, "Early onset of hereditary gingival fibromatosis in a 28-month-old," *Pediatric Dentistry*, vol. 31, no. 4, pp. 286–288, 2009
3. Lee EJ, Jang SI, Pallos D, Kather J, Hart TC. Characterization of fibroblasts with Son of Sevenless-1 mutation. *J Dent Res*. 2006; 85:1050-5.
4. L. H¨akkinen and A. Csiszar, "Hereditary gingival fibromatosis: characteristics and novel putative pathogenic mechanisms," *Journal of Dental Research*, vol. 86, no. 1, pp. 25–34, 2007.
5. Seymour RA, Heasman PA. Drugs and the periodontium. *J Clin Periodontol*. 1988:15:1-16.
6. L.P.Bittencourt, V.Campos, L. F.M. Moliterno, D. P. B. Ribeiro, and R. K. Sampaio, "Hereditary gingival fibromatosis: review of the literature and a case report," *Quintessence International*, vol. 31, no. 6, pp. 415–418, 2000
7. Coletta RD, Graner E. Hereditary gingival fibromatosis: a systematic review. *J Periodontol*. 2006;77:753-64

****