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Association of Hereditary Gingival Fibromatosis with Aggressive Periodontitis: A Unique case report

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Abstract: Gingival overgrowth occurs due to increased number of cells specially fibroblasts. It occurs as a side effect to systemic drugs, such as nifedipine, phenytoin, cyclosporine or due to hereditary predisposition. Here is a special case report describing hereditary gingival overgrowth along with aggressive periodontitis.
Keywords: gingival overgrowth, collagen, pathogenesis.

INTRODUCTION

In the healthy state, the gingival tissues in mouth almost completely fill the interproximal spaces between teeth, beginning near the contact area and extending apically and laterally in a smooth curve. Gingival hyperplasia/ Gingival overgrowth (GO) can be defined as an abnormal growth of the periodontal tissue[1]. The term "gingival hyperplasia" is an inappropriate term because enlargement is not the result of an increase in the number of cells, but rather an increase in extracellular tissue volume[2]. Such enlargement can be caused by multiple stimuli and stands as a singular complaint in the dental office. Gingival enlargements may be either inflammatory, non-inflammatory or a combination of both[3]. The inheritance condition in which the gingival tissue spontaneously and progressively enlarges is identified as hereditary gingival fibromatosis (HGF)[4]. Gingival enlargement may cause discomfort, interfere with speech or chewing, resulting in halitosis and it may look unsightly. This case report describes an unique case of hereditary gingival fibromatosis with aggressive periodontitis.

CASE HISTORY

A 35 years old mentally retarded female patient, reported to department of periodontology with the chief complaint of swelling in gums and difficulty in closing mouth since two years. The patient gave a history of generalized bleeding from gums which was spontaneous in nature and subsided after 1 to 2 minutes. Swelling in gums increased in size over a period of two years (Fig. 1-3).

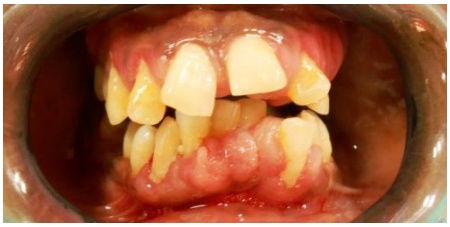

Fig-1

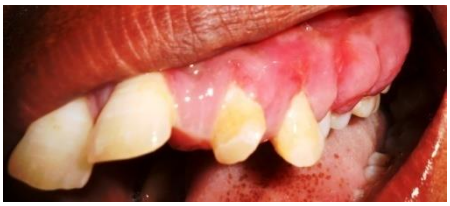

Fig-2

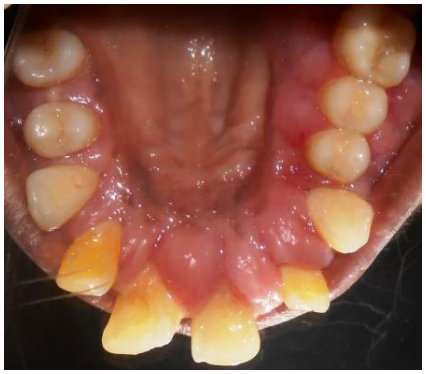

Fig-3

Patient neither had any history of systemic drug intake nor any gyaenecologic/hormonal problem. In her family history she told that her late mother, aged 73 suffered from the same gingival problem as she was. The general physical examination was non-

contributory. The extraoral findings revealed incompetent lips and absence of any lymphadenopathy.

The examination of gingiva revealed that the enlargement was painless, present in marginal and interdental gingival both in the maxilla and mandible. Pathologic migration with 11,12, 21, 22 and 32 was evident. Deep bite was also present. Calculus deposits were seen on exposed tooth surfaces. On palpation the surface of overgrowth was smooth, edematous and not attached to underlying bone. The overgrowth covered more than two thirds of crown. The degree of gingival overgrowth was scored as Grade III [5]. Periodontal examination showed that there was generalized probing depths ranging from 5 to 8 mm, Grade III mobility and Grade IV furcation involvement with 26 , 36 and 37 was evident.

Patient was made to undergo routine hemogram (RBC, WBC, platelet counts, ESR, bleeding time, clotting time, prothrombin time) and radiological examination. The haematologic parameters were within normal limits, while advanced bone destruction was clearly evident in Orthopantamogram (Fig. 4).

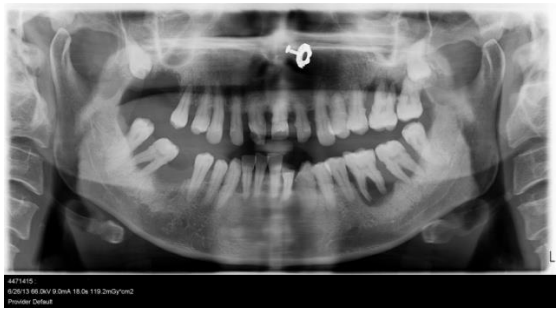

Fig-4

Based on the history and investigations a provisional diagnosis of hereditary gingival fibromatosis with generalized aggressive periodontitis was made. A small tissue was excised and sent for histopathological assessment, which revealed a mixture of dense and loose fibrous components with the chronic inflammatory cell infiltrate in the connective tissue and elongation of rete pegs in the epithelium (Fig.5).

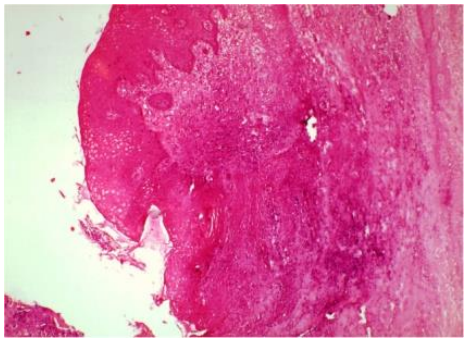

Fig-5

Thus, a final diagnosis of hereditary gingival Fibromatosis and aggressive periodontitis was arrived at.

Treatment plan included, extraction of teeth with hopeless prognosis i.e. 26, 36 and 37. A thorough supragingival and subgingival scaling was done followed by education and motivation to maintain the oral hygiene. After a period of four weeks of assessment, when the size of the enlargement didn't subside surgical intervention was decided upon.

Under local anesthesia, an external bevel incision was given and lesion was removed both buccally and palatally (Fig. 6&7).

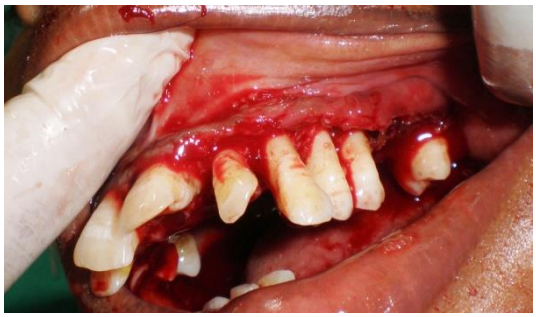

Fig-6

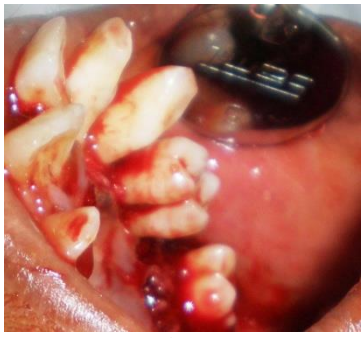

Fig-7

Even after the incision, a considerable amount of fibrotic tissue was left and the teeth surfaces were not visible completely. Hence flap technique was chosen. With a # 11 Bard Parkar blade, initial sclloped internal bevel incision was given including the creation of new interdental papilla. The same blade was used to thin the gingival tissues in buccolingual direction and a full-thickness flap was elevated till 2mm of alveolar bone was visible. The excised marginal and interdental tissue were removed with curettes. Tissue tabs were removed and the roots were thoroughly scaled and root planed, no osseous recontouring was done. The flap was sutured using 3-0 non resorbable suture and periodontal pack was placed (Fig.8 & 9).

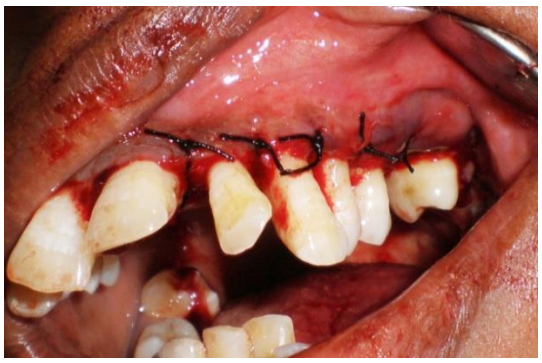

Fig-8

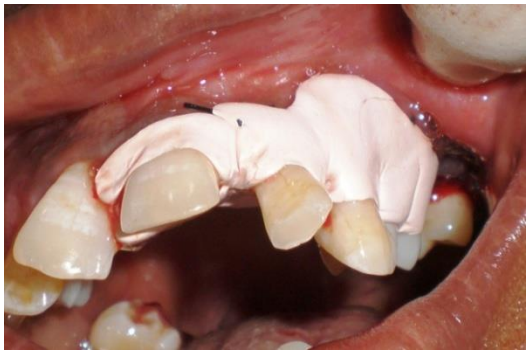

Fig-9

The patient was prescribed Cap. Amoxycillin 500mg thrice daily for seven days, Tab. Diclofenac sodium 50 mg thrice daily for seven days and chlorhexidine mouth wash (0.2%) twice daily for two weeks. Patient was recalled after one week for suture and pack removal, healing was found to be satisfactory (Fig.10 a & b).

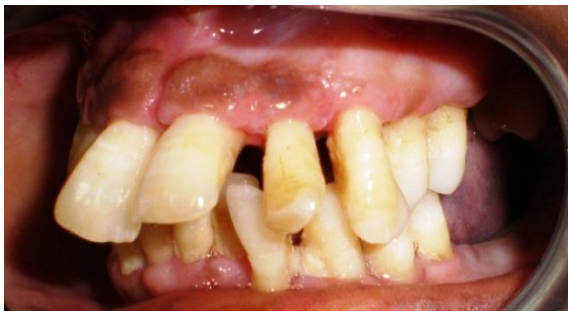

Fig-10 (a)

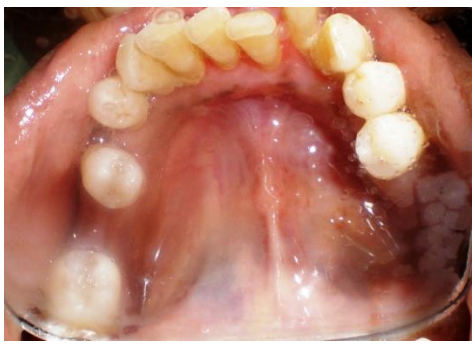

Fig-10 (b)

There was no discomfort or side effects were observed in the postoperative period. At 3 weeks after the treatment of gingival overgrowth, there was no recurrence and the patient expressed a high level of satisfaction. After 3 months, removable prosthesis was given to patient for better masticatory function. Regular follow up was done till 18 months and there was no reoccurrence of gingival overgrowth observed (Fig.11). Further follow up was lost as the patient's family moved to a different city.

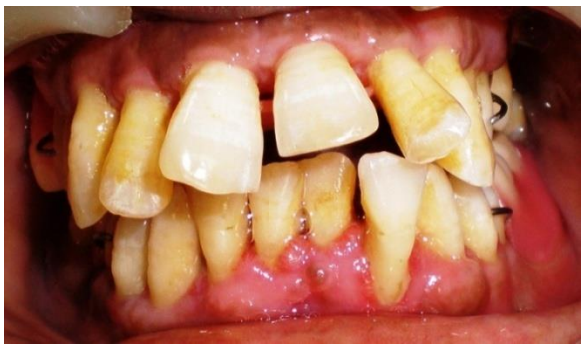

Fig-11

DISCUSSION

Gingival overgrowth varies from mild enlargement of isolated interdental papillae to segmental or uniform and marked enlargement affecting one or both of the jaws with a diverse etiopathogenesis[6]. It may be caused by medications, including antiepileptic drugs (AED)[7] or genetic abnormalities, such as hereditary gingival fibromatosis[4]. This case reports management of hereditary gingival fibromatosis with aggressive periodontitis. HGF is a rare disease impaired by the intense clinical, genetic, and biologic heterogeneity. Gingival enlargement may be generalized or localized to a specific area, typically the maxillary tuberosities and the labial gingiva around the lower molars[8]. The most common effects are diastemas, malpositioning of teeth, prolonged retention of primary dentition, delayed eruption, cross and open bites, prominent lips, and open lip posture[9]. HGF is traditionally considered an autosomal dominant disease[10-11]. Another study [12] reported autosomal dominance in a four-generation pedigree with 50 of 105 at risk family members developing gingival fibromatosis. In the present case, the gingival enlargement was a hereditary condition, probably autosomal-dominant, due to its existence in her parent (mother).

HGF is associated with several syndrome (ear, nose, bone and nail defects with hepatosplenomegaly), Rutherfurd syndrome (corneal dystrophy, mental retardation, impairment of dental eruption by radicular resorption), or Cross syndrome (microphthalmia, mental retardation, athetosis and hypopigmentation). The characteristics most often associated with HGF are hypertrichosis, mental retardation and epilepsy[13]. In this case, the association with mental retardation was present.

Possible biological mechanism of HGF could be due to somatic mutations resulting in gingival fibroblasts with elevated synthesis of transforming gowth factor beta (TGF-b), which in turn promotes cellular proliferation and abnormal collagen production and reduces the synthesis and activity of extracellular matrix-degrading metalloproteinases[14]. Additionally, TGF-b1 may induce fibroblast transdifferentiation into myofibroblasts, which are regarded as the predominant cells in matrix synthesis in interstitial fibrosis, such as HGF[15]. All of the actions of TGF-b result in a dysregulation of the connective tissue homeostasis, leading to the accumulation of extracellular matrix, which clinically ends up with gingival overgrowth[16].

A study has shown that aggressive periodontitis is also a genetically inherited disease and mixed model segregation analysis of 100 families confirmed with 104 probands with aggressive periodontitis, indicated that most families were consistent with dominant mode of transmission[17].

This is a unique case where two genetically modified clinical pathologies were coexisting in the same individual. The suggested treatments vary according to the degree of severity. As the excess tissue increases, appearance and functional impairment dictate the need for surgical intervention. Hence, in this case surgical intervention was chosen. Though there is a risk for reoccurrence, many studies have demonstrated that there was no reoccurrence as long as 14 years[13]. In this case there was no reoccurrence noted for 18 months.

CONCLUSION

HGF is a challenge both to dentist as well as patient. Not only it impairs the upkeep of good oral hygiene, it also causes functional impairment and psychological stress. Here a case of HGF and aggressive periodontitis was treated. The treatment not only established the functionality of the dentition but also helped the patient to live her life without any stigma. This report highlights the unique occurrence of aggressive periodontitis which is also a genetically modified disease. Genetic studies need to be carried out to establish any relationship between the two pathologies.

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REFERENCES

1. American Academy of Periodontology; Informational paper: drug associated gingival enlargement. J Periodontol, 2004; 75:1424-1431.
2. Desai P, Silver JG; Drug-induced gingival enlargements. J Can Dent Assoc, 1998; 64:263-268.
3. Rajendran R, Shivpathasundharam B; Shafer's Textbook of Oral Pathology. 5th ed. Elsevier, 2007; 543- 548.
4. Ricardo DC, Edgard G; Hereditary gingival fibromatosis: A review. J Periodontol, 2006;77:753-764.
5. Carranza FA, Hogan EL; Gingival enlargement. In: Newman MG, Takei HH, Carranza FA, editors. Clinical Periodontology. 9th ed. Philadelphia: Saunders; 2002; 279-296.
6. Tiwana PS, Kok IJ, Stoker DS, Cooper LF; Facial distortion secondary to idiopathic gingival hyperplasia: surgical management and oral re-construction with endosseous implants. Oral Surg Oral Med Oral Pathol Oral Radiol Endod, 2005;100(2):153–157.
7. Tan H, Gurbuz T, Dagsuyu IM; Gingival enlargement in children treated with antiepileptics. J Child Neurol, 2004; 19:958-63.
8. Bittencourt LP, Campos V, Moliterno LF, Ribeiro DP, Sampaio RK; Hereditary gingival fibromatosis: Review of the literature and a case report. Quintessence Int, 2000;31:415-418.
9. Baptista IP; Hereditary gingival fibromatosis: A case report. J Clin Periodontol, 2002;29:871-874
10. Raeste AM, Collan Y, Kilpinen E; Hereditary fibrous hyperplasia of the gingiva with varying penetrance and expressivity. Scand J Dent Res, 1978;86:357-365
11. Emerson TG; Hereditary gingival hyperplasia: A family pedigree of four generations. Oral Surg Oral Med Oral Pathol, 1965;19:1-9.
12. Bozzo L, de Almedia OP, Scully C, Aldred MJ; Hereditary gingival fibromatosis: Report of an extensive four-generation pedigree. Oral Surg Oral Med Oral Pathol, 1994;78:452-454.
13. Baptista IP; Hereditary gingival fibromatosis: a case report. J Clin Periodontol, 2002; 29: 871–874.
14. Cotrim P, de Andrade CR, Martelli-Junior H, Graner E, Sauk JJ, Coletta RD; Expression of matrix metalloproteinases in cyclosporin-treated gingival fibroblasts is regulated by transforming growth factor (TGF)-beta1 autocrine stimulation. J Periodontol, 2002;73:1313-1322.
15. Zhang C, Meng X, Zhu Z, Liu J, Deng A; Connective tissue growth factor regulates the key events in tubular epithelial to myofibroblast transition in vitro. Cell Biol Int, 2004;28:863-873.
16. Wright HJ, Chapple IL, Matthews JB; TGF-beta isoforms and TGF-beta receptors in drug-induced and hereditary gingival overgrowth. J Oral Pathol Med, 2001;30:281-289.
17. Piero C, Ilhan UM, Alpdogan K, Hatice H, Serge D, Thomas CH, et al; Hereditary gingival fibromatosis associated with generalized aggressive periodontitis: A case report. J Periodontol, 2004;75:770-778.