Case Report

Gingival Fibromatosis - An Uncommon Case Report

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Abstract

A peculiar and diverse collection of conditions known as gingival fibromatosis manifest as slowly developing, localized, or diffuse enlargements in the interdental papilla or the gingiva's margins and attachments. In extreme situations, the extra tissue may cover the tooth crowns, which could result in functional, cosmetic, and periodontal issues such bleeding and bone loss from pseudopockets and plaque buildup. It has an equal impact on men and women.A uncommon genetic disorder called hereditary gingival fibromatosis (HGF) is characterized by gradual gingival overgrowth. In addition to frequently delaying tooth eruption, gingival overgrowth can result in major functional and cosmetic issues. An odd disorder known as gingival hyperplasia affects the oral cavity's masticatory, functional, psychological, and cosmetic aspects. Gingival hypertrophy causes can in clude be due to plaque accumulation, due to poor oral hygiene, inadequate nutrition, or systemic hormonal stimulation.

Introduction

One aspect of idiopathic fibrous hyperplasia of the gingiva is a gradual fibrous expansion of the gingiva[1]. Other names for GF include elephantiasis gingivae, familial elephantiasis, gigantism of the gingiva, gingivomatosis, gingival enlargement, gingival hyperplasia, and gingival overgrowth (GO)[2]. Idiopathic fibrous gingival fibromatosis is an uncommon inherited disorder for which there is no known cause[3].

In clinical practice, gingival expansion is frequently observed and can be caused by interactions with the environment, the host, or a variety of stimuli. These lesions may be more widely distributed, affecting broader regions of the oral cavity, or they may be limited to a specific area. Possible causes of this illness include systemic-induced manifestation, hormone abnormalities, or plaque[4].

Idiopathic gingival fibromatosis, also known as hereditary gingival fibromatosis (HGF), is an uncommon, benign, asymptomatic, non-hemorrhagic, non-exudative, proliferative fibrous lesion of gingival tissue that affects both men and women equally and occurs in both arches with differing degrees of intensity in members of the same family[5].

There is also a recessive version of the disorder, however autosomal dominant is the more common. There appears to be a higher chance of autosomal dominant inheritance in consanguinity. Consanguinity seems to increase the risk of autosomal dominant inheritance. It affects the marginal gingival, attached gingival and interdental papilla presenting as pink, non-hemorrhagic and have a firm, fibrotic consistency[6].

This illness can present as a standalone disorder or as a component of a syndrome. It can also have an autosomal dominant or, less frequently, an autosomal recessive mode of inheritance[7-9]. Gingival fibromatosis autosomal-dominant variants, typically nonsyndromic, have been genetically associated with chromosomes 5q13-q22 and 2p21-p222[10].

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Although no conclusive connection has been made, a mutation in the son of sevenless-1 (SOS-1) gene has been proposed as a potential cause of isolated (nonsyndromic) gingival fibromatosis in the modern era [11].

Case Report

A 32 years old female reported to the Department of Periodontics, Career Postgraduate Institute of Dental Sciences and Hospital, Lucknow, Uttar Pradesh with a chief complain of gingival swelling in the left lower anterior region of jaw since 2 year.(Figure 1) The patient reported that the gingival enlargement was smaller in size earlier and increased gradually.The patient gave no medical history. There was no family history of GF.

Upon intraoral inspection, a pink, leathery-consistency, and hard-to-palpate region was found to have localized gingival overgrowth in the left mandibular lateral incisor, spreading into the vestibular and canine areas. The intra oral periapical radiograph showed that there was bone loss, grade II mobility, and tooth locations were compromised by swelling. (Figure 2)After routine blood work, results were found to be within the normal range.

Following the administration of local anesthetic (2% lignocaine hydrochloride mixed with 1:80,000 epinephrine), the growth in the mandibular arch was removed via an external bevel gingivectomy using a knife. The removed tissue measured 10 mm in diameter and 5 mm in length. To confirm the diagnosis, a biopsy of the tissue was sent for histological analysis. A periodontal dressing was used.

The patient was instructed to take B-complex vitamins, analgesics (ibuprofen 400 mg twice daily for five days) and antibiotics (amoxicillin 500 mg thrice daily for five days) in addition to rinsing their mouth with 0.2% chlorhexidine gluconate for two weeks. Postoperative instructions and advice on oral hygiene were provided.

Histological analysis showed irregularly organized collagen bundles inside dense collagenous connective tissue. There was little in the way of inflammatory cell infiltration and avascular connective tissue. The epithelium covering it had larger rete ridges and was hyperplastic. (Figure 4) shows that the histological picture was indicative of GF. The healing process after surgery went smoothly. After a week, the patient was brought back, the periodontal dressing was taken off, and the affected area was treated with regular saline and betadine. The patient was being monitored for a postoperative assessment. The patient was happy with the outcome.

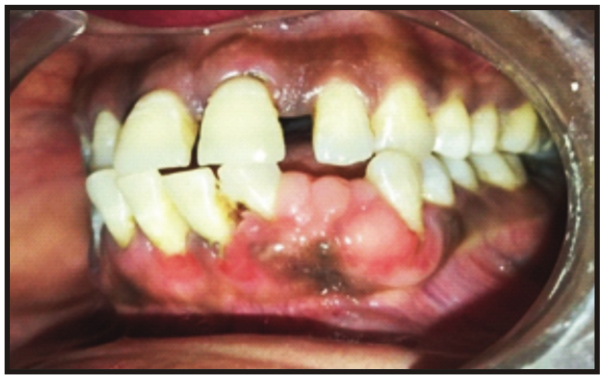

Figure 1. Gingival Overgrowth i.r.t.31,32,33

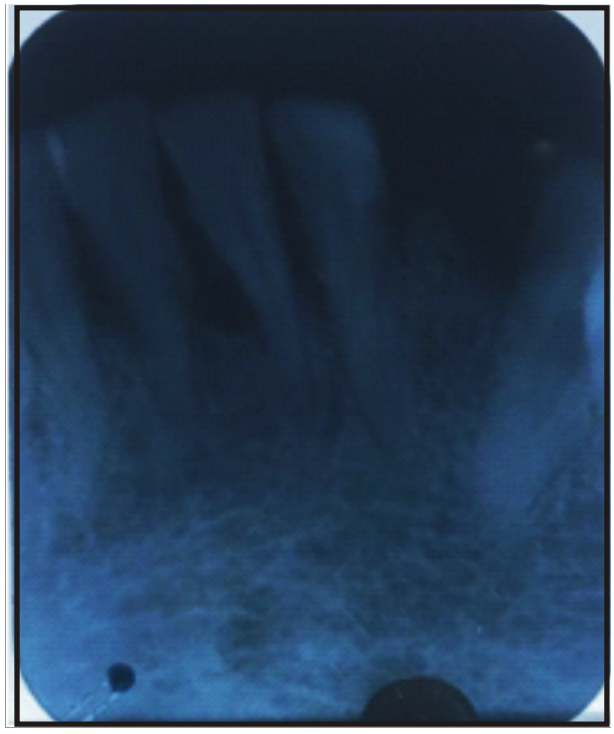

Figure 2. IOPAR i.r.t. 31,32,33

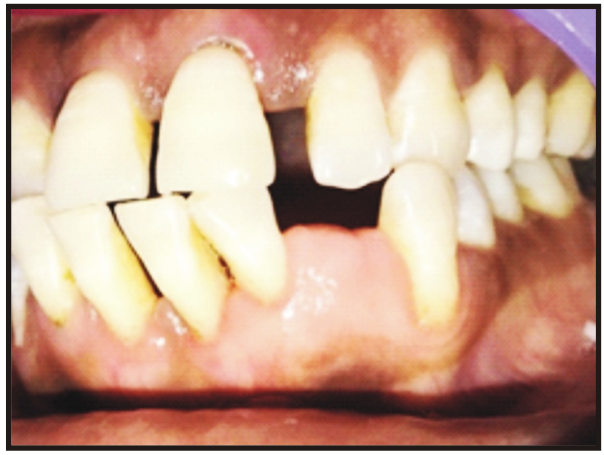

Figure 3. Post-op after 3 month

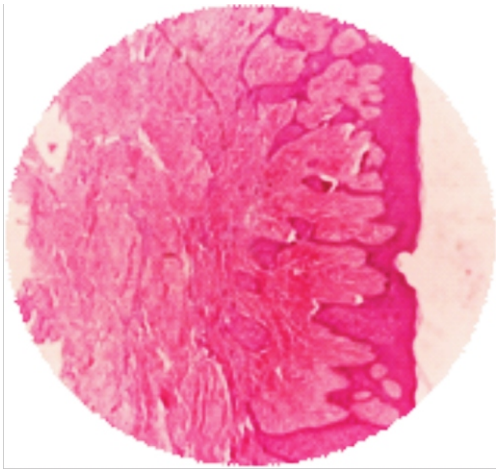

Figure 4. Histopathological Picture.

Discussion

The degree of gingival overgrowth varies, ranging from a small amount of expansion in one or both of the jaws to a segmental or uniform, noticeable enlargement[12] Hereditary Gingival Fibromatosis (HGF) is a non-dental plaque-induced gingival disease that progresses slowly.[13] Several writers have explained this clinical enlargement as the result of an excessive build-up of collagen and a considerable proliferation of fibroblasts in the gingival fibrous connective tissue, despite the fact that the exact mechanism of HGF remains uncertain.[14].

We diagnosed the patient with idiopathic gingival fibromatosis because the patient's medical, prenatal, and family histories did not support the diagnosis. Generalized gingival fibromatosis has several etiological factors. These include leukemia-induced gingivitis, drug-induced gingival hypergrowth, scurvy gingival hypergrowth during pregnancy, and mouth-breathing gingivitis, Canthosis nigricans, Wegener granulomatosis, hereditary gingival fibromatosis, idiopathic type. There are several disorders with different modes of inheritance that present with generalized gingival fibromatosis.which include the autosomal-recessive syndromes (Cross, Murray-Puretic-Dresher, and Ramon) and the autosomal-dominant syndromes (Laband and Rutherford).[15]

Idiopathic gingival fibromatosis's exact process is unclear, although it seems to be limited to the fibroblasts that are present in the gingivae. The hyperplastic reaction happens peripherally to the alveolar bone within connected gingival tissue and is not related to the periodontal ligament[16]. While severe, diffuse gingival enlargements necessitate surgical intervention, routine treatment for mild and local enlargements depends on maintaining proper dental hygiene and/or root scraping. Recurrences might happen months or years following surgery [17,18,19,20.]

Case reports by Dhadse et al.[21]Hereditary gingival fibromatosis affecting marginal and attached gingival to varying degrees of both arches, impeding esthetic, phonetic, and masticatory function,dictates the surgical intervention in the management of hereditary gingival fibromatosis. Quadrant-wise surgical excision was performed under local anesthesia, followed by oral hygiene reinforcement, and periodic follow-up. Recurrence is a common feature varying over a period and this dictates the importance of regular recall visits to evaluate the stability of periodontium.

Coletta RD[22],have also reported that the activated gingival fibroblasts in hereditary gingival fibromatosis produce more collagen, fibronectin than normal gingival fibroblasts under the autocrine control of TGF-β favoring the accumulation of extracellular matrix production.

Tipton DA[23],reported an increase in the proliferation of gingival fibroblasts in hereditary gingival fibromatosis than the normal gingival fibroblast. Elevated and prolonged expression of proto-oncogenes c-myc is implicated in increased proliferation of human gingival fibroblast cell line.

Bozzo and colleagues[24],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 siblings (sister), although her mother and father were phenotypically normal. Moreover it was unrelated to endocrine problems or use of medications.

Mastication issues, speech issues, tooth displacement, cosmetic consequences, and psychological issues for the patient are among the complications associated with GF; for this reason, proper therapy and postoperative care are essential.

Conclusion

The etiological heterogeneity of GF, an uncommon and slowly developing illness, is another characteristic. Furthermore, as recently revealed,this illness is a common sign of multiple genetic disorders and may also arise sporadically in various other syndromes and diseases. Diagnosis is made based on medical history,

clinical examination, blood tests and histopathological evaluation of affected gingival tissue. Since gingival overgrowth is disfiguring and can obstruct speaking and chewing, a complete understanding of the pathophysiology is crucial. It is plausible that the distinct characteristic of gingival cells stems from their anatomical position, or that moderate localized inflammation—even in clinically healthy tissue—activates the cells.

For an accurate diagnosis based on medical history, clinical examination, blood tests, and histological evaluation as well as for appropriate care, a thorough case history is necessary to ascertain the etiology and histopathological examination. Fibroma, peripheral giant cell granuloma, PG, peripheral odontogenic fibroma, and peripheral ossifying fibroma are among the clinical differential diagnoses for localized gingival overgrowths.Reducing the local microflora will aid in removing the primary site of infection in an oral environment that is quite healthy and is maintained by the patient and the dentist.

Patient compliance and motivation are essential components of a successful therapy outcome. The patient needs to be scheduled for routine dental checkups, during which time the entire state of their teeth should be described. This would enable the use of less intrusive therapeutic techniques than surgery in standard dental practice and enhance disease management. Creating awareness and educating patients regarding the influence of poor oral hygiene and behaviors to these gingival lesions are vital to avoid further recurrence.

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