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Clinicalmanagement of Idiopathic Gingival Fibromatosis: a Case Report

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ABSTRACT:Gingival fibromatosis is mainly due to an increase and thickening of the collagen bundles in the connective tissue stroma of the gingival tissues. The term idiopathic is for gingival fibrosis with unknown etiology. We present a clinical case report of a 24-year-old female patient with no pathologic or hereditary history of generalized gingival enlargement. The gingival tissue enlargement was diagnosed as idiopathic gingival fibromatosisby clinical and histopathological studies. the periodontal management consisted of periodontal surgery and maintenance treatment, with positive results at 9 months postoperatively. The purpose of this study was to report a case of gingival fibromatosis, which was generalized and involved both jaws.

KEYWORDS: Gingival fibromatosis, Gingival enlargement, Periodontitis, Collagen

I. INTRODUCTION

Gingivalfibromatosis(GF) is a condition characterized by a progressive increase in gingival tissue caused by a slow-growing fibrous proliferation of connective tissue, which in some cases covers the entirety of the involved teeth. It can present as a generalized enlargement of the gums bilaterally and symmetrically, being the less frequent localized form, this could be able to remain in a latent state and suddenly extend to one or both jaws. The prevalence is 1 in 175,000 and both genders are equally affected. GF is also called gingivomatosis, gingival enlargement, gingival hyperplasia, gingival overgrowth, gingival elephantiasis and congenital macrogingival. [1,3,21]

It usually begins at the time of eruption of the permanent teeth but may develop with the eruption of the primary dentition and is rarely present at birth. Hyperplasic gingival tissue has a color pink-red, a hard consistency, rough surface and abundant stippling. It can partially or totally cover dental crowns, present diastemas, delay or prevent tooth eruption and evolve to periodontitis. In severe cases, there may be alterations in chewing, phonation and lip incompetence, presenting difficulty in achieving adequate oral hygiene.[2]

The term idiopathic gingival fibromatosis (IGF) is for gingival fibrosis with no known etiology. The average age of presentation is 26 years. Gingival enlargement usually involves maxillary and mandibular gingival tissue. Recently, epithelial to mesenchymal transition has been proposed as another pathogenic pathway that promotes gingival fibrosis.[9]

Etiology

GF can be a congenital or acquired condition.In the congenital form the autosomal dominant hereditary component is the most frequent, although there are reported cases with a recessive component, and it can occur in isolation or as part of a syndrome. The autosomal dominant forms are not generally associated with syndromes but are genetically related to mutations of chromosome 2, in the 2p21-p22 and 5q13-q22 sector. Acquired forms may be related to inflammatory processes (oral biofilm, calculus) and systemic alterations such as uncontrolleddiabetes,leukemia, thrombocytopenia[7,9,10]Studies have suggested that an important pathogenic mechanism may enhance the production of transforming growth factor beta1 (TGF-B1), which reduces the proteolytic activities of GF fibroblasts, again favoring the accumulation of extracellular matrix. [23]

GF is mainly due to an increase and thickening of collagen bundles in the connective tissue stroma. The nodular appearance can be attributed to the thickened hyperparakeratinized epithelium.Keratinocytes present in GF appear to play an important role in pathogenesis by inducing the accumulation of extracellular matrix by fibroblasts. In addition, it has been observed that

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increased proliferation and elevated production of extracellular matrix molecules, fibronectin and type 1 collagen could lead to fibrous gingival enlargement.[3,4]

The connective tissue consists of excess collagen, but has relatively few fibroblasts and blood vessels. [5] The superficial epithelium shows thin, fine interpapillary ridges.[23] Enlarged fibroblasts appear to alternate with thin and thick collagen fibrils. Small calcified particles, connective tissue bone metaplasia, amyloid deposits or islets of odontogenic epithelium may be found as an unusual finding.[5] There is a significant number of mast cells, associated with fibroblastic proliferation, and an almost total absence of inflammatory cells.[23.] Elastic and oxytalan fibers are also present, increased gingival tissue may provide new niches for the growth of microorganisms, plaque accumulation and pseudo-pocket formation resulting in inflammatory infiltration of gingival connective tissue. [2,3]

Diagnosis

IGF is a diagnosis of exclusion, by definition, patients with IGF lack a family history of gingival hyperplasia and have not identifiable causative agents such as medication use or systemic conditions. After carefully ruling out all known causes of gingival enlargement, a diagnosis of IGF can be made. The inclusion criteria for diagnosing IGF are: no signs of genetic disorder, no evidence of medication use associated with GF, not associated with pregnancy or puberty, and no family history of gingival hyperplasia. [1] IGF may be associated with hypertrichosis (increased hairiness), mental retardation or epilepsy, and is also associated with syndromes such as Ruthenford, Cross, Ramon, Zimmerman-Laband, Prune-Belly, Jones, Klipper.[9,10]

Treatment

The patient's medical history (the patient's age and the presence of other diseases) and clinical examination findings (the type and severity of the enlargement) influence the patient's treatment. Nonsurgical treatment includes scaling and root planing, oral hygiene instructions, and administration of antibiotics, usually amoxicillin and metronidazole, along with anti-inflammatory medications (ibuprofen) and analgesics (acetaminophen)[14].Sahrmannconcludes that the adjuvant use of povidone iodine during scaling and root planing can increase the reduction in clinical pocket depth from small to moderate[16]. Povidone iodine is an antiseptic with a broad antibacterial spectrum that covers Gram-positive, Gram-negative and Candida albicans bacteria.Different studiesshowed additional short-term improvements with the use of povidone iodine during subgingival debridement.[17,18]

Management of patients diagnosed with GF includes surgical treatments such as regenerative or resective surgery and antimicrobial treatment.[2,15] An internal bevel gingivectomy is performed along with open flap debridement. This procedure eliminates the pocket, reduces the tissue mass and makes biofilm control much easier. Removal of the hypertrophic tissue can also be done with electrosurgery, which reduces the risk of bleeding and pain. This type of procedure significantly reduces the amount of local anesthetic used, leads to better visibility, which reduces the time of care and results in better patient acceptance.[2,12,14]

II. CASE REPORT

A 24-year-old female patient attended the dental clinic because of generalized gingival enlargement. She denies hereditary and personal pathological data. Reported brushing her teeth once a day, without the use of oral hygiene aids, she did not smoke or drink alcoholic beverages, denied drug use and reported not having undergone a previous prophylaxis.

Reports presenting gingival enlargement for8 years ago that have been increasing up to date, she reported generalized bleeding when brushing. Additionally, the patient reported pain provoked by brushing, accompanied by dental mobility in the posterior teeth. She does not present sensitivity or any other associated alteration. On interrogation with the accompanying family member, the complaints were not related to the use of medication or hereditary relationship.

Oral cavity examination

The oral examination showed pink cheeks with chewing lesions in both cheeks, a fissured tongue and a red vascular floor of the mouth, and a hypoplastic maxilla.Generalized gingival enlargement was observed in the maxilla and mandible both in the vestibular and palatal/lingual areas, irritated areas and generalized spontaneous bleeding. It was found red colored gingiva with rounded shape, smooth texture and soft consistency in the molar area and firm in the anterior area.

In the area of the upper first molar and lower left premolars there was an increase of sessile base volume very vascularized in the vestibular area. In the palatal area there were fibrotic nodules

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together with a thickening of the palatal mucosa. Generalized oral biofilm accumulation and oral calculus were observed. Furthermore, it was detected generalized enamel hypoplasia together with malocclusion and dental crowding.

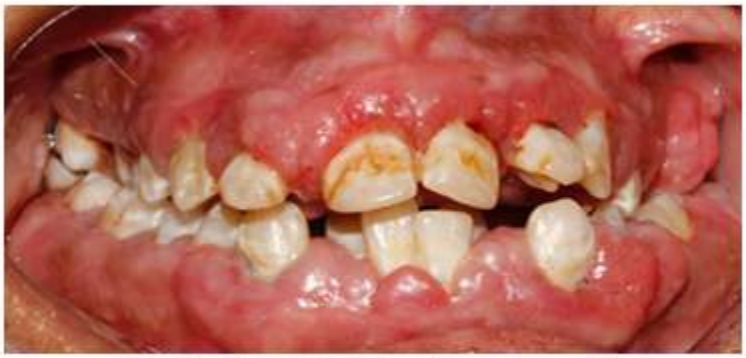

Fig. 2: Fibrotic gingival enlargement, vestibular area of both jaws.

Probe depht mean	7.5mm
Biofilm %	69%

Fig. 3

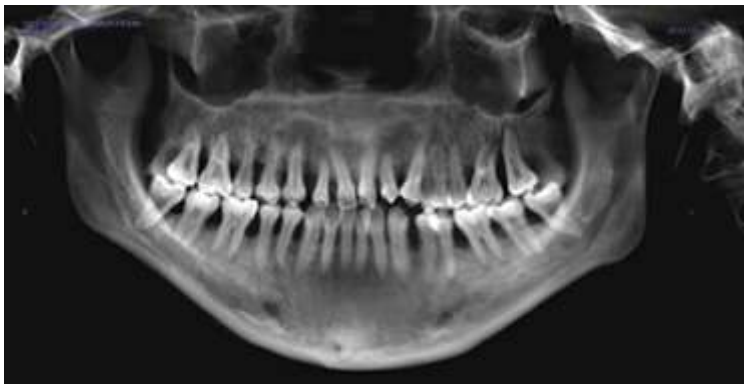

Fig.4. Panoramic X-ray, where we observe generalized moderate horizontal loss of bone crest height, and vertical loss in localized areas.

Diagnosis

Considering the results obtained from the clinical examination and radiographic data the periodontal diagnosis was concluded: Periodontitis stage II, Grade B Generalized, mucogingival deformity / gingival excess due to oral biofilm etiology.[26] A general prognosis is given depending on the treatment as reserved. Prophylactic and surgical treatment is performed.

Treatment

Scaling and root planning was performed in the 4 quadrants. Combined irrigation consisting of physiological solution (sodium chloride 0.9%) andpovidone-iodine (buccopharyngeal iodine 10%) was used. Excisional biopsy of the enlargement of the crown from the upper left second molar wasperformed, then gingivoplasty of the area was carried out

Histological examination(Fig.4) showed normal epithelium with elongated epithelial ridges, connective tissue with abundant intertwined fascicles of dense fibrous tissue with few fibroblasts and blood vessels and mild infiltration of

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inflammatory cells, giving a diagnosis of gingival fibromatosis.

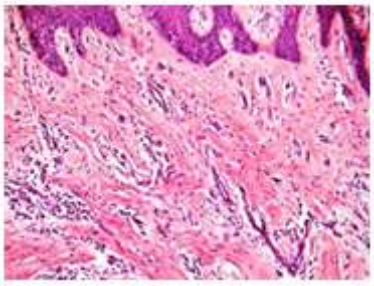

Fig. 4

In the second surgical intervention, a debridement flap (modified Widman), scaling and root planing is performed together with osteoplasty to return the proper bone architecture in the 4 quadrants (Fig. 5,6), combined irrigation consisting of physiological solution (sodium chloride 0.9%) and Povidone- iodine (buccopharyngeal Iodine 10%) is used. It is sutured with resorbable material 4-0 single mattress. Specific indications of care and oral hygiene are given, antibiotic (amoxicillin 500mg. Capsules, 1 c/8 hrs. For 7 days) and analgesic (ibuprofen 600mg tablets, 1c/8hrs. For 5 days) are prescribed. Indications are given for the use of mouthwash (iodine buccopharyngeal twice a day).

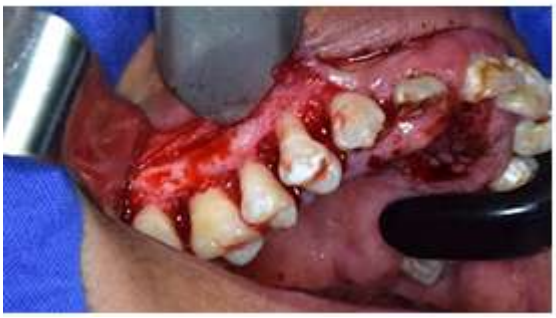

Fig. 5

Fig. 6

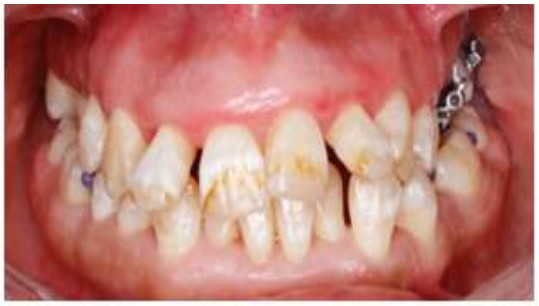

Fig.7

At 9 months postoperatively, generalized healthy gingival tissue is observed in the maxillary and mandibular arch, with an average probing depth of 3.7mm with the start of orthodontic treatment (Fig.7).

III. DISCUSSION

Gingival enlargement varies from mild growth of isolated interdental papillae to segmental or uniform and marked enlargement affecting 1 or both jaws. Gawron[3] reports that GF can be inherited, in most cases as an autosomal dominant disease or an autosomal recessive disease, or it can be caused by a new mutation.

The case presented in this work does not coincide with the results presented by Gawron[4] presenting a negative family history for GF, as no other family members were affected.

The modified Widman flap was described in 1974 by Ramfjord and Nissle and is known as open flap curettage. It includes the removal of the pocket epithelium, granulation tissue and scaling and root planing of the affected surfaces. Flap repositioning is performed aiming to close the pocket as coronally as possible.[5]Gawron K. In his study of resective surgery in patients with GF, four weeks after surgery, the condition recurred in 45% of the initial tissue volume present in the mandible and 25% in the maxilla. Two months later, no significant growth was observed in the mandible, whereas, in the maxilla, growth increased to 40% of the preoperative state[4].

Studies by Rani[9],Neville et al[22]report that gingival fibromatosis presents an association of inducing fibroblasts stimulating T-lymphocytes to undergo increased collagen production in the gingiva, so it is not necessary to use other diagnostic means in addition to histopathology to diagnose gingival fibromatosis. Another study by Gawron[4] reports a postoperative recurrence rate of gingival enlargement of 45%, although Kumar[10] reports that his studies on recurrence rates are contradictory so

the long-term postoperative benefit of periodontal surgery cannot be predicted.

Chitsazi in 2015[11]refers that the likelihood of IGF in adolescents is higher than in adults and is more rapid in regions with biofilm, proper control with maintenance visits for biofilm control and short term could be more effective in reducing the risk of recurrence of this condition. He also reports that GF is less likely to recur if gingivectomy is performed until the permanent dentition has erupted. [11]

Sahrmann[16] and Rosling[17] use povidone iodine irrigation to eliminate microorganisms in the treatment of periodontitis with minimal results, but Hoang[18], in his study of irrigation in the treatment of GF, obtained a reduction of pathogenic microorganisms with the use of povidone iodine during debridement, decreasing periodontal pocket depth and better healing results.

IV. CONCLUSION

Chemical and mechanical control of biofilm are important to avoid recurrence of gingival aggradation. An adequate clinical history is important because gingival enlargements are multifactorial, which represents a diagnostic challenge for the Periodontist.

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