REVISTA CIÊNCIAS e ODONTOLOGIA

STUDY ABOUT HEREDITARY GINGIVAL FIBROMATOSIS AND ITS IMPACT ON DENTAL TREATMENT

Yago Moreira Marques[1], Lais Natália de Carvalho Santos[2], Ricardo Fabris Paulin[3], Erica Carine Campos Caldas Rosa[4]

[1] Student of Scientific Initiation and Graduation of the Dentistry Course of the University Center ICESP

[2] Graduation Student of the Dentistry Course of the University Center ICESP.

[3] Doctor in Orthodontics. Senior Professor at Icesp University Center and Coordinator of the Dentistry Course from ICESP University Center

[4] Doctor in Health Sciences / UNB. Senior Professor of the Undergraduate Dentistry Course and Advisor of Scientific Initiation at the University Center ICESP

Summary

Introduction: Gingival Fibromatosis or Gingival Hyperplasia is a rare disorder with slow and progressive growth and is characterized by being a neoplasm in the gingival tissue, where it can be found in a localized manner. This study aims to elucidate the characteristics and possible manifestations, aiming to facilitate its identification, treatment and control. ***Methodology:*** This is a literature review. Forty-two articles and theses with languages in English, Spanish, French, and Portuguese were researched, whose sources are Scielo and Pubmed, and 25 articles were included for their relevance to the topic. ***Literature review:*** Gingival fibromatosis is a clinical manifestation present in several genetic syndromes that can lead to a craniofacial involvement that is characterized by the expansion and accumulation of connective tissue with increased number of cells and collagen fibers. Fibromatosis has a proliferation of well-differentiated fibroblasts with varying amounts of collagen between proliferating cells and can be classified as: drug-induced and hereditary. Through histopathology, the stratified squamous epithelium is obese with prominent and elongated ridges, dense connective tissue and disorganized cells with slow but progressive growth. It is also a rare disorder of non-neoplastic gingival tissue (1 case/750,000 people) that presents normal coloration, firm consistency and absence of inflammatory signs. ***Conclusion:*** The expression of Gingival Fibromatosis has several origins, making it necessary to know the causative factor in the patient to prepare the treatment plan according to their needs, so the conversation with the patient through the anamnesis is so important at the beginning of treatment. Oral manifestations are important clinical findings to assist in the diagnosis of genetic syndromes, allowing the dentist to guide their patients and clarify doubts about dental impairment.

Keywords: Fibromatosis; Gingival fibromatosis; Hereditary gingival fibromatosis.

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INTRODUCTION

Gingival Fibromatosis or Gingival Hyperplasia is a rare disorder with slow and progressive growth that can lead to a considerable change in the patient's physiognomy. It is characterized by being a neoplasm in the gingival tissue, where it can be found in a localized manner, affecting less than 30% of the dental arch or in a generalized manner, affecting more than 30% of the arch, whether in the lower or upper jaw [1, 2]. Its expression is due to the accumulation of collagen in the hypocellular and hypovascular connective tissue, forming several longitudinal bundles in a disorganized manner that are arranged in different directions. The growth may be diffuse, affecting other tissues and appears in a benign form. It can generate pathological periodontal pockets due to the difficulty in brushing, in which case Gingival Fibromatosis occurs in a more aggressive form, disposing of chronic inflammatory periodontitis. The difficulty of brushing also generates an increased risk of caries due to the difficulty of removing plaque, thus making the main causative agent of caries proliferate freely on the dental surface[3,4]. Their origins are variable, they may have hereditary, medicinal, inflammatory, syndromic, and even idiopathic origins. Some syndromes contain clear characteristics about the presence of Gingival Fibromatosis, such as hypertrichosis and cherubism; hereditary syndromes such as Zimmermann-Laband, Murray-Puretic-Drescher and Rutherfurd also have the phenotype of Gingival Fibromatosis. Some pathologies also go hand in hand with Gingival Fibromatosis, such as generalised aggressive periodontitis, hypothyroidism and chondrodystrophy [4-8]. In some patients there is a genetic predisposition, with a higher prevalence in the inbreeding family. Isolated cases where there are no relatives with a history of Gingival Fibromatosis are of autosomal dominant origin; in rare cases they are found in an autosomal recessive form [5].

2.0 LITERATURE REVIEW

2. 1 CAUSING FACTORS

2.1.1 HEREDITY

The first case was described by Gross (1856). Its characteristics are the exacerbation of the inserted gingiva, the marginal gingiva and the interdental papillae. In most cases an autosomal dominant pattern is observed; some reports of autosomal recessive origin also exist, but are rarer. It can also be expressed in cases in which the parents are inbreeding, but there are no data in the literature explaining the relationship between inbreeding and the expression of fibromatosis [9].

2.2.2 MEDICATIONS

Medicamentous Gingival Fibromatosis is established by generating hyperplasia in patients who did not have the expression of Gingival Fibromatosis, but after the use of the drug express the abnormal growth of the gingiva. In this specific case, neither the epithelium nor the connective tissue cells show hypertrophy. Gingival size is due to the growth of the extracellular matrix in which there is a predominance of collagen [10]. There are several medications linked to Medicated Gingival Fibromatosis, but there are two main groups: anticonvulsants and calcium channel blockers, cyclosporine, erythromycin and oral contraceptives, which can also cause gingival hyperplasia [9].

2.2.3 INFLAMMATION

Gingival fibromatosis generated by inflammation is a consequence of untreated periodontitis. Periodontitis is generated through the bacterial plaque accumulated on the surface of the teeth, in cases where no treatment and no removal of plaque are then formed periodontal pockets. Periodontal pockets are consequences of a deepening of the gingival margin due to the chronification of periodontal disease, this periodontal pocket leads to a tendency to generate collagen fibers, leading to hyperplasia and consequently to Gingival fibromatosis.

2.2.4 SYNDROMES

Some syndromes express in their nature the Gingival Fibromatosis in the oral environment of the patient are examples of Hyper-

trichosis and cherubism, appearing in most cases. Generally, the expression occurs before the age of 20 and its progression is slow and painless[10]. Hypertrichosis is a congenital or acquired syndrome. Its main characteristic is the growth of hair in abnormal quantities. In patients with Hypertrichosis, there is the presence of Gingival Fibromatosis. Gingival Fibromatosis is a consequence of one of the several expressions of the patient's phenotype[11]. Kerubism is a rare hereditary condition, with rare exceptions of isolated cases, in which the patient has the face in a cherubic form, its expression occurs in the bone tissue in a non-neoplastic form in which there is a bilateral enlargement of the mandible and the maxilla. The phenotype of this syndrome also generates in several cases the expression of Gingival Fibromatosis in the patient [12]. The idiopathic origin relates to the group of patients who express Gingival Fibromatosis, but does not have a cause.

3.0 CLINICAL EXPRESSION

It has a firm, pink, non-haemorrhagic appearance, with an apparent consistency similar to that of leather and can be covered by a smooth or dotted surface. It may cause the covering of the dental arch, in some cases covering the dental crown of all the teeth of the arch in its totality. Radiographically in general, with few exceptions, they present bone resorption and well-defined carious lesions [3].

The maxilla is affected more frequently than the palate, for as yet unknown causes. Most commonly, it is available for implementation in hard tissues, most of which develop up to 20 years of age and are frequently related to the growth of deciduous and permanent teeth[1,16].

3.1 HISTOPATHOLOGICAL EXPRESSION

Histologically, it resembles fibrous hyperplasia, characterized by stratified and keratinized epithelial tissue disorder, presents crest of the epithelium-conjunctive junction of irregular and disorganized forms, also presents bundles of collagen fibers arranged in irregular shapes and in all directions, with accumulation of bundles per region disseminated along the connective tissue [3, 17, 18].

3.2 DIAGNOSTIC EXAMINATIONS

Gingival fibromatosis has characteristics similar to those of gingival hyperplasia, so it is important to confirm the difference between these two diseases by means of complementary tests[13]. Radiographic examinations are extremely important in confirming the diagnosis of Gingival Fibromatosis; the bone structure affected by Fibromatosis has high bone resorption, which is presented in a generalized manner along the entire affected extension; in addition to bone resorption, radiography may also show other problems caused by Gingival Fibromatosis, such as retention of eruption of dental components[19, 20].Due to the growth of the tissue and the fact that it covers the faces of the dental surface, its cleaning is difficult to perform and may generate accumulation of bacterial plaque, causing the presence of tartar, extensive carious lesions, and periodontal problems such as gingivitis and periodontitis[16, 21, 22].

To assist in the diagnosis there is the option of sending a biopsy for histopathological analysis. In the microscopic realm, gingival fibromatosis has hypocellular and hypovascular collagenous tissue, which are arranged in clustered bundles in an irregular arrangement and in various directions[22,23].

3.3 DATA SYNTHESIS

The prevalence varies according to the etiological factor of the patients. Hereditary Gingival Fibromatosis has a genotype frequency of 1:175,000 and a gene frequency of 1:135,000, if the parents are inbred, this frequency may increase from 60% to 90%[4, 6, 16, 24].

Medicamentous Gingival Fibromatosis has a frequency of 50% of onset in case of use of antiepileptic drugs, and 20% of onset in case of use of calcium channel blocking drugs[3, 25]. Syndromic Gingival Fibromatosis appears in 95% of cases of syndromes such as cherubism and hypertrichosis; the prevalence is too high because it is a phenotypic expression of the reported syndromes.

FINAL CONSIDERATIONS

The diagnosis of Fibromatosis is made essentially based on the medical history and clinical examination of the patient. There are studies being carried out to establish the phenotypic binding of this disorder and a region on chromosome[2] (2p21-p220 between genetic markers D2S1788 and D2S441.There is no consensus among the various authors on the activity of fibroblasts in the affected gingival tissues, but there is a consensus on treatment, which can be performed by simple gingivectomy in milder cases until the extraction of dental elements and mass removal of gingival tissue in more complex cases, but there will always be the possibility of recurrence of expression if the main causative factor for Gingival Fibromatosis is not removed.

Therefore, the expression of Gingival Fibromatosis and FGH has several origins, making it necessary to know the causative factor in the patient to prepare the treatment plan according to his or her needs, which is why the conversation with the patient through the anamnesis is so important at the beginning of treatment, and to make effective the removal of tissue growth caused by Gingival Fibromatosis, it is first necessary to remove the etiological factor of its cause, either by inflammation or the accumulation of tartar on the tooth surface. There are causes in which these factors cannot be removed, such as Idiopathic Gingival Fibromatosis, hereditary Fibromatosis and spontaneous Fibromatosis; in such cases, what can be done is only to follow the gingival growth and remove the excess from time to time[2, 16, 22, 26-30].