Hereditary gingival fibromatosis about a Case

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Case Report

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Abstract: Hereditary gingival fibromatosis (FGH) is a gingival pathology of genetic origin characterized by a slow and progressive proliferation of the keratinized gingiva. The diagnosis is clinical, confirmed by histology. The treatment is surgical and involves the excision of excess tissue with gingivoplasty, but the recurrence rate remains high, especially in severe cases. We report a case of generalized FGH, whose evolution was favorable after surgical treatment. Observation: A 42-year-old patient, with no notable history, who presented in our consultation for a gingival hypertrophy evolving since childhood. Endobuccal examination showed diffuse gingival hypertrophy with poor oral status. Gingival biopsy revealed gingival fibromatosis. Treatment consisted of excision of excess tissue with gingivoplasty. No recidivism after a decline of three years. Discussion: FGH is a genetic gingival pathology whose clinical appearance is variable, it may be generalized or localized to a maxillary sector, isolated or associated. The positive diagnosis is mostly clinical confirmed by histology (hyperplastic epithelium and dense connective tissue), the treatment is surgical and consists of excision of excess gingival tissue with gingivoplasty. Our patient has a severe FGH that has evolved after surgical treatment.

Keywords: Fibromatosis, gingival, hereditary.

INTRODUCTION

Hereditary gingival fibromatosis (FGH) is a gingival pathology of genetic origin characterized by a slow and progressive proliferation of the keratinized gingiva [1]. The diagnosis is clinical, confirmed by histology.

The treatment is surgical and involves the excision of excess tissue with gingivoplasty, but the recurrence rate remains high, especially in severe cases.

We report a case of generalized FGH, whose evolution was favorable after surgical treatment. Hereditary gingival fibromatosis about a case.

OBSERVATIONS

A 42-year-old patient, with no notable pathological history, who presented in our consultation for a gingival hypertrophy evolving since childhood. Endobuccal examination showed diffuse gingival hypertrophy with poor oral status (Fig.-1).

Gingival biopsy revealed gingival fibromatosis. A family survey was conducted and showed the absence of other cases in the family.

Treatment consisted of excision of excess tissue with gingivoplasty and extraction of all teeth. Currently, no recurrence after a decline of three years.

Fig-1: Photo showing diffuse gingival hypertrophy covering teeth with multiple dental caries.
DISCUSSION

Hereditary gingival fibromatosis (FGH), also known as elephantiasis gingiva or hereditary gingival hyperplasia [1], is a gingival pathology of genetic origin characterized by a slow and progressive proliferation of the keratinized gingiva [2]. It affects both sexes with a frequency of 1 per 750,000 [3].

Clinically, the gingiva maintains a normal color and a firm consistency and is neither bleeding nor painful. The increase in gingival volume can be generalized or localized to a segment of the maxillary or mandible and the degree of gingival hyperplasia is also variable [4]. In severe cases, the gum almost completely covers the tooth surfaces and deforms the palate, causing not only an aesthetic and functional problem (disturbed phonation and chewing), but also a difficulty in maintaining adequate oral hygiene [2], as in the case of our patient (Fig-1).

FGH is either isolated or associated with other symptoms such as hypertrichosis (the most common symptom), mental retardation, epilepsy and / or deafness indicating the pleomorphic effect of one or more genes mutants [5]. It may also be one of the features of rare syndromes such as Murray-Puretic Dresher syndrome [2].

The positive diagnosis of FGH is both clinical and familial and confirmed by pathological examination of eliminated gingival tissue (hyperplastic epithelium and dense connective tissue). The differential diagnosis is mainly with idiopathic gingival hyperplasia and gingival medicinal hyperplasia, in which the phenomenon is induced by specific drugs, particularly phenytoin, cyclosporine and nifedipine.

Genetically, FGH is generally transmitted in the autosomal dominant mode with incomplete penetrance, but autosomal recessive inheritance has also been reported. Nevertheless, the genes responsible remain unknown although some studies have identified the presence of locus in relation to the 2p chromosome condition at region 21 or chromosome 5q at regions 13 and 22 without determining the type genetic alteration. [1, 6, 7].

The treatment is most often surgical and consists of the excision of excess tissue by gingivectomy and / or gingivoplasty to restore the gingival contours. The recidivism rate is unfortunately important especially in severe cases. The gingivectomy can be performed conventionally to the blade, by electrocautery or laser. Nevertheless, the prognosis is reserved since recurrence is frequent. To avoid this recurrence, some authors have recommended not only the excision of the excess gingival tissue but also the extraction of all the teeth [1, 8] as the case of our patient.

CONCLUSION

FGH is a rare disease. Its clinical repercussions on aesthetic, functional and dental aspects require early diagnosis and treatment. The positive diagnosis is mostly clinical confirmed by histology, the treatment is surgical and consists of excision of excess gingival tissue with gingivoplasty. Our patient has a severe FGH that has evolved after surgical treatment.

REFERENCES