

C. Ercoli, M. Bartolino, L. Montesani, R. Docimo*

Paediatric Dentistry, University of Rome "Tor Vergata", Rome, Italy
*Professor of Pediatric Dentistry

e-mail: cristina.ercoli@live.it

Gingival fibromatosis: a case report

ABSTRACT

Aim Gingival Fibromatosis is characterised by a large increase in the gingival dimension which extends above the dental crowns, covering them partially or completely. The causes of the disease may have a genetic origin, in which case gingival hyperplasia may occur in isolation or be part of a syndrome, or acquired origin, which comes from specific drugs administered systemically. A form of gingival fibromatosis of idiopathic origin has been described. The therapy involves mainly the surgical removal of the hyperplastic gingival tissue, although in these cases recurrences are frequent.

Case report A 9 years old male patient came to observation at the Clinic of Pediatric Dentistry of the Tor Vergata Polyclinic of Rome. After Primary Gingival Fibromatosis was diagnosed, the therapeutic choice was to wait and postpone gingivectomy at the end of the development phase.

Keywords Gingival fibromatosis; Fibroblastic proliferation.

Introduction

Prevalence and aetiology

Gingival Fibromatosis is a rare disease with a prevalence of 1 in 175,000 individuals and an equal distribution between males and females, characterised by a generalised or localised thickening of the gingival tissue, caused by expansion and accumulation of connective tissue mostly constituted by type I collagen with the occasional presence of an increased number of cells due to fibroblastic proliferation [Araiche and Brode, 1959].

The causes of the disease are classified into genetic,

acquired or idiopathic.

When gingival hyperplasia has a genetic origin, it is defined Hereditary Gingival Fibromatosis and can occur in isolation or associated with other clinical manifestations which are grouped within of a specific syndrome. Most cases have an autosomal dominant inheritance with a good level of penetrance, although in the literature autosomal recessive transmissions have been described [Bhowmick et al., 2001; Hart et al., 2000; Lindhe, 2006; Singer et al., 1993]. The most common conditions associated with gingival hyperplasia of hereditary origin includes hypertrichosis, epilepsy and mental retardation. Multiple hyaline fibromatosis, corneal dystrophy, defects of the nasal bones and of the ears, hepatosplenomegaly, microphthalmia, IQ below normal, athetosis (involuntary movements of the limbs caused by a disorder of the extrapyramidal system), hypopigmentation and hypertrichosis may also occur together with the fibromatosis [Araiche and Brode, 1959; Lindhe, 2006]. Tooth absence or delay in eruption can affect the deciduous or permanent dentition. Partial expressions of the syndrome have been described, and in these cases the increase of gingival tissue involves only the teeth of the upper front area.

Another cause of Gingival Fibromatosis is linked to the side effect resulting from the use of systemic drugs such as anticonvulsants (phenytoin), calcium channel blockers (nifedipine), and immunosuppressants (cyclosporine). A prolonged treatment with hydantoin (sodium-5,5-diphenylhydantoin, a drug used to treat epilepsy and certain heart rhythm disorders) may cause cases of fibrous gingival hyperplasia, which occurs in 50% of the treated patients (reactive subjects) 3-9 months after the start of treatment. Teenagers and subjects under 30 years show worst reactions than older patients. Even children and teenagers undergoing post-transplant therapy with cyclosporine A can show some form of fibrous gingival hyperplasia. Cyclosporin A (CsA) is a drug used to prevent rejection reactions or "Graft-Versus-Host Reaction" in transplantations of kidney, liver, bone marrow, heart and heart-lungs. Gingival hyperplasia appears in reactive subjects within 3 months of treatment. The percentage of reactive subjects under treatments with CsA vary between 25-81%. This variability is due to the dosage or plasma concentration of CsA, length of treatment, periodontal situation, possible presence of associated systemic disease [Brown et al., 1991; Lindhe, 2006; Seymour et al., 1996; Soames et al., 1985]

In the literature a form of Gingival Fibromatosis of idiopathic origin is also described.

Clinical-histological aspects and diagnosis

clinically Gingival Fibromatosis entails a localised or more often generalised fibrous hyperplasia of the gingival tissue, which grows above the dental crowns, covering them partially or totally, affecting the front sector first and then the lateral and posterior sectors [Araiche and Brode,



FIG. 1 OPG of the patient.



FIG. 4 Teleradiography.

1959; Soames et al., 1985].

The hyperplasia affects especially the interdental tissues and the labial surfaces of the front teeth.

Gingival fibromatosis is more common on the maxillary arch. The colour of the gingiva may be normal or erythematous in case of inflammation.

Histologically the fibrous tissue is composed of bundles of collagen fibers of type I, while the covering epithelium is often hyperplastic [Lindhe, 2006; Soames et al., 1985].

The diagnosis is mainly clinical, following the detection of a solid, fibrous, compact, pink body, frequently lobular, covering the dental crowns and causing difficulty in chewing, speech, swallowing and closing of the mouth, delays in teeth eruption, periodontal pockets without loss of attachment, and aesthetic problems of. Sometimes the patient's lips are incompetent.

Occasionally these patients show traits suggestive of acromegaly [Araiche and Brode, 1959; Lindhe, 2006; Soames et al., 1985].

Treatment

The therapeutic options for gingival fibromatosis consider the surgical removal of hyperplastic gingival tissue, usually with multiple gingivectomies, although in these cases the risk of relapse is high. As an alternative, an apically repositioned flap can be raised, which prevents the exposure of the connective tissue [Lindhe, 2006].

The gingivectomy may be problematic because of the intense bleeding due to tissue inflammation.

The initial treatment consists in the accurate removal of dental plaque, an important cofactor in the induction of the fibrous gingival hyperplasia; the gingivectomy can be postponed at the end of the growth phase of the patient, in order to decrease the risks of relapse.



FIG. 2 Frontal view of the dental arches.



FIG. 3 Intraoral view of the maxillary arch.



FIG. 5 Frontal view of the right side.



FIG. 6 Intraoral view of the mandibular arch.



FIG. 7 Frontal view of the left side



FIG. 8 Dental arches in protrusion

If gingival fibromatosis is caused by the use of antiepileptic, calcium channel blockers, immunosuppressants drugs, the initial treatment, which has to be planned together with the pediatrician or with the internist, should be aimed at the reduction of the dosage of the drug. In addition, the careful management of dental plaque through professional and at-home oral hygiene can reduce the degree of inflammation of the hyperplastic tissue. If dose reduction is not possible or does not yield the desired effect, it is advisable to perform gingivectomy.

Case report

The patient LS, a boy of nine years, presented at the Paediatric Dentistry Department of "Tor Vergata" Polyclinic of Rome (Italy) for examination. Later the patient was referred to the Department of Genetic Medicine, where he was diagnosed with Primary Gingival Fibromatosis of genetic origin due to a new gene mutation with autosomal dominant transmission, without other important clinical expressions and not associated with other syndromes.

The intraoral examination showed a generalised fibrous

hyperplasia of the gingival tissue both the maxillary and the mandibular arch, where the typical hyperplastic gingiva covered the crowns of the teeth (Fig. 1-8).

The therapeutic choice was to postpone removal of the gingival tissue until the end of the growth phase. This decision was imposed by the high rate of relapse when the surgery is performed during the growth phase.

Furthermore a scoliosis and a premature pubertal development confirmed the need of a multidisciplinary approach.

The patient was enrolled in a prevention program that requires periodic checkups and professional oral hygiene sessions in order to control the dental plaque, the most important cofactor in the onset of gingival hypertrophy.

Discussion

This is a case of Primary Gingival Fibromatosis observed in a child of nine years caused by a new gene mutation with autosomal dominant transmission.

The therapeutic choice was a wait-and-see approach, postponing the removal of excess tissue to a later time, because this condition recurs when treated in childhood.

Conclusion

Even though gingival fibromatosis is an uncommon condition, its knowledge and its aetiological, pathological and clinical aspects is very important.

This condition may be isolated or part of a syndrome.

When the generalised fibrous hyperplasia of the gingival tissue is the only evident sign of the disease, the diagnosis is clinical and can be formulated after the intraoral examination.

If the condition is part of a syndrome the clinical aspects most commonly associated with it are hypertrichosis, epilepsy, and mental retardation.

An early diagnosis is crucial in order to prevent complications, such as malocclusions, labial incompetence, periodontal defects, problems in chewing, speech, swallowing and psychological problems due to the disfigurement that gingival fibromatosis involves.

Proper oral hygiene and an accurate dental plaque control are key prognostic factors.

The careful evaluation of the most appropriate timing for removal of the hyperplastic tissue is imperative in order to avoid post-gingivectomy relapses.

References

- › Araiche M, Brode H. A case of fibromatosis gingivae. *Oral Surgery* 1959; 12, 1307-1310.
- › Bhowmick SK, Gidvani VK, Rettig KR. Hereditary gingival fibromatosis and growth retardation. *Endocrine Practice* 2001; 7, 383-387.
- › Brown RS, Beaver WT, Bottomley. On the mechanism of drug-induced gingival hyperplasia. *J Oral Pathol Medicine* 1991; 20, 201-9.
- › Hart TC, Pallos D, Bozzo L. Evidence of genetic heterogeneity for hereditary gingival fibromatosis. *J Dental Research* 2000; 79, 1758-1764.
- › Lindhe J. *Clinical periodontology and implant dentistry*. 2006; 286-287.
- › Seymour RA, Thomason JM, Ellis JE. The pathogenesis of drug-induced gingival overgrowth. *J Clinical Periodontology* 1996; 23, 165-75.
- › Singer SL, Goldblatt J, Hallam LA. Hereditary gingival fibromatosis with a recessive mode of inheritance. Case reports. *Austrian Dental J* 1993; 38, 427-432.
- › Soames JV, Southam JC. *Oral pathology*. 1985. Italian Edition; 2005; 116-117.