Hereditary gingival fibromatosis is rare, affecting only one in 750,000 people. It usually develops as an isolated disorder but can be one feature of a syndrome. In their review of various syndromes, Gorlin and colleagues identified hypertrichosis as the characteristic most often seen with syndromic HGF and noted that HGF is occasionally associated with mental retardation and epilepsy. HGF is usually identified as an autosomal dominant condition though recessive forms are described in the literature.

The hyperplastic gingiva usually is of normal color and has a firm consistency, with abundant stippling on the adjacent gingiva. Buccal and lingual tissue may be involved in both the mandible and maxilla, and the degree of hyperplasia may vary between individuals within the same family.

**CASE REPORT**

A 28-year-old black woman with a chief complaint of gingival pain and swelling on the right side sought evaluation at the Long Island College Hospital dental department. Clinical examination revealed generalized, severe gingival hyperplasia involving both the mandibular and maxillary arches (Figures 1-3). The gingiva was pink, and its firm, dense, fibrous consistency caused considerable difficulty with plaque removal. In addition to the symptomatic gingiva, other areas also were inflamed. The patient noted that she was unhappy with the appearance of her gingiva. Her medical history was unremarkable, although she stated that she had had a gingivectomy at 7 years of age and that her gingiva had progressively enlarged to its present state by 18 years of age. She denied taking any medications. Her weight and height were considered to be within normal limits. She did not appear to have any mental impairment.

Her family history was of significance, since her deceased mother had had a similar gingival condition. She recalled that her mother had had several gingival excisional surgeries because of unpredictable, slowly progressing hyperplasia. She also stated that her mother had not had epilepsy or any type of seizure disorder, nor had she taken any medications associated with gingival hyperplasia. Her mother’s medical and dental records, however, were not available to confirm this.

A diagnosis was made of HGF. The patient’s family history combined with the absence of a relevant medication history

**ABSTRACT**

Hereditary gingival fibromatosis, or HGF, is characterized by varying degrees of attached gingival hyperplasia. The authors describe a case of generalized severe hereditary gingival fibromatosis involving the maxillary and mandibular arches. Removal of excess gingival tissue by conventional gingivectomy dramatically improved the patient’s appearance.
Figure 1. Severe gingival hyperplasia in a 28-year-old woman. Based on the patient's history, we diagnosed the condition as hereditary gingival fibromatosis.

Figure 2. Note the generalized involvement of the maxillary and mandibular tissue as seen on the right side of the patient's mouth.

Figure 3. Because of the dense, fibrous consistency of the tissue, the patient had difficulty maintaining adequate oral hygiene.

Figure 4. Grafting and an oral hygiene regimen that included 0.2 percent chlorhexidine rinses helped resolve the overgrowth of tissue.

distinguished this condition from the more common, drug-related gingival overgrowths associated with phenytoin, cyclosporine and nifedipine.46

TREATMENT

With exception of carbon dioxide laser, which has been used in a number of studies,7-10 the most efficacious method of removing large quantities of gingival tissue—particularly when there has been no attachment loss and all the pocketing is false—is the conventional, external bevel gingivectomy.11

In this case, treatment consisted of quadrant-by-quadrant gingivectomy with periodontal pack placement for one week, followed by 0.2 percent chlorhexidine oral rinses twice a day for two weeks after each surgery.

An oral pathologist examined the tissue removed during gingivectomy. The microscopy showed mild epithelial hyperplasia and marked interwoven fascicles of dense fibrous tissue. Slender fibroblastic nuclei were scattered throughout the stroma. A mild chronic inflammatory cell infiltrate also was noted. These microscopic findings supported the diagnosis of familial or hereditary gingival fibromatosis.

After the last gingivectomy and post-surgical follow-up visit, the patient returned periodically during a two-year span for observation. Scaling and prophylaxis were performed every six months. In spite of the patient's less-than-adequate oral hygiene and the presence of marginal gingivitis, professional dental care helped prevent a recurrence of the hyperplasia (Figure 4). Should the overgrowth recur, excision should be repeated, as the psychological benefits resulting from cosmetic improvement outweigh the risk of recurrence.12
DISCUSSION

Generalized gingival fibromatosis can be caused by a number of factors, including inflammation, leukemic infiltration and medication use such as phenytoin, cyclosporine or nifedipine. About 50 percent of patients taking phenytoin will manifest gingival hyperplasia. As noted earlier, gingival fibromatosis can occur as part of a syndrome. It has been reported as a feature of Murray-Puretić Drescher syndrome (multiple hyaline fibromas), Rutherford's syndrome (conveal dystrophy), Laband syndrome (ear, nose, bone and nail defects with hepatosplenomegaly), Jones syndrome (progressive deafness) and Cross syndrome (microphthalmia, mental retardation, athetosis and hypopigmentation). Last year, Wynne and colleagues reported a new syndrome of HGF occurring with hearing deficiencies, hypertelorism and supernumerary teeth.

HGF can be inherited as an autosomal dominant or recessive condition. Bozzo and colleagues reported autosomal dominance in a four-generation pedigree with 50 of 105 at-risk family members developing gingival fibromatosis. As in this case, the hyperplastic tissue usually is of normal color. The degree of severity varies from mild involvement of one quadrant to severe involvement of all four quadrants. Gingival tissue enlargement usually begins with the eruption of the permanent dentition but can develop with the eruption of the deciduous dentition; it rarely is present at birth. A few cases have arisen in adulthood. In a study involving 17 family members with gingival fibromatosis, Fletcher reported the most extensive enlargement appeared to occur either during loss of the deciduous teeth or in early stages of eruption of the permanent dentition. He noted that the enlargement seems to progress rapidly during "active" eruption and decrease with the end of this stage.

CONCLUSION

Hereditary gingival fibromatosis cannot be cured but can be controlled with varying degrees of success. When the enlargement is minimal, good scaling of teeth and home care may be all that is required to maintain good oral health. As the excess tissue increases, appearance and function indicate need for surgical intervention. Several authors have reported the recurrence of hyperplastic tissue following gingivectomy, necessitating a repeat of the procedure.

While CO₂ laser excision appears promising, further experience and study are needed to determine if it is the optimal alternative therapy. The psychological benefits resulting from cosmetic improvement far outweigh the risks of recurrence.

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