Recurrent gingival fibrous lesions: comparison of 2 cases and potential need for additional classification

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Historically, the classification of localized gingival fibrous lesions has been inconsistent, leading to multiple naming schemes and confusion among pathologists. Currently, lesions are broadly grouped into localized hyperplastic lesions and true neoplasms. Although some cases are clearly defined histologically (i.e., peripheral ossifying fibroma, peripheral odontogenic fibroma), another set of "reactive" fibrous lesions exhibit overlapping histologic features including nondistinctive fibrosis and inflammation. This group can exhibit recurrence, classically related to a local stimulus. However, when local factors are absent, recurrence suggests inherent neoplastic potential. Herein, we describe 2 recurrent fibrous gingival masses, one of which reportedly recurred 3 times with no obvious inciting agent. The histologic appearance of both lesions was similarly distinctive but not well classifiable, while the immunohistochemical profile indicated divergent lesions. This highlights the need for further study of recurrent gingival fibrous lesions to better predict independent growth potential. (Oral Surg Oral Med Oral Pathol Oral Radiol 2013;116:e287-e296)

Localized gingival fibrous lesions are relatively common and for decades have elicited a variety of classification schemes. Although a variety of names exist, it is reasonable to group these lesions as either localized hyperplastic lesions (i.e., peripheral ossifying fibroma [POF], focal fibrous hyperplasia [FFH])[1] or as neoplasms containing a prominent fibrous component (i.e., aggressive fibromatosis, peripheral odontogenic fibroma [POdF]). Among the hyperplastic group, local irritants (e.g., plaque and calculus, poor crown margins, etc.) are the often reported cause,[2-4] although many cases do not present with an obvious etiology.[5] Reported recurrence rates are highly variable but generally appear to be low (2%-9%) for lesions likely corresponding to FFH[3,6-8] and somewhat higher for POF (8%-30%).[3,9-13] With each of these lesions, recurrence may be attributed to either continued irritation,[5,9,10] inadequate excision,[9,10] or a lack of correction of the periodontal defect.[8]

Histologically, POF and POdF have been clearly defined in the literature such that typical cases do not present a diagnostic challenge. The difficulty arises when trying to classify exophytic fibrous lesions that do not possess the characteristic features of these other well-defined entities and fall into a category of "fibrous hyperplasia," "fibrous epulis," or "peripheral fibroma". We prefer the term "focal fibrous hyperplasia" as suggested by Buchner[1] to define gingival lesions that show a poorly circumscribed collection of dense fibrous connective tissue arranged in haphazard strands of variably sized but poorly defined collagenous bundles lacking significant cellularity. We present 2 cases of recurrent gingival fibrous lesions which, despite having some distinctive features beckoning a more specific diagnosis, would by current classification likely be designated as FFH. Immunohistochemistry was performed to look for a myofibroblastic component in addition to the histology which suggested only fibroblastic differentiation. This description highlights the need for better characterization of recurrent lesions diagnosed as fibrous hyperplasia and other "fibromas" of the gingiva to establish if there is a unique subset of lesions that are better designated as neoplasms rather than hyperplasias.

MATERIALS AND METHODS

In order to better characterize the cellular differentiation, an immunohistochemical panel focusing primarily on myofibroblastic differentiation[14] was performed using available antibodies directed against vimentin (Leica, prediluted by manufacturer; Buffalo Grove, IL, USA), α-smooth muscle actin (SMA) (Leica, prediluted by manufacturer), fibronectin ED-A (clone IST-9 highlights the extra domain-A variant of fibronectin, 1:50; Abcam Cambridge, MA, USA), muscle-specific actin (HHF-35) (Leica, prediluted by manufacturer), smooth muscle myosin heavy chain (clone SMMS-1, 1:100; Dako, Carpinteria, CA, USA), H-caldesmon (H-CD,[1] 1:125; Dako), desmin (DE-R-11; Leica, pre-diluted by manufacturer), S-100 protein (polyclonal, 1:800; Dako), and CD34 (QBend/10, 1:100; Leica). Given the recurrent nature, Ki-67 (MM1, 1:100; Novocastra, Newcastle Upon Tyne, UK) was also performed to evaluate the proliferative index of these lesions. Percent of cells staining and staining intensity were each graded on a 4-point scale as depicted in Table I.

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Table I. Immunohistochemical studies

Markers	*Case 1 first recurrence (1993)*	*Case 1 third recurrence (2011)*	*Case 2*
Vimentin	4/+++	4/+++	4/+++
α-smooth muscle actin (SMA)	3/+++	4/+++	0/+
Fibronectin ED-A	1/+ to ++	3/+ to ++	4/++
Muscle specific actin (HHF-35)	2/+ to ++	3/++ to +++	0/−
Smooth muscle myosin (SMMS-1)	0/−	1/+	0/−
H-caldesmon	0/−	0/−	0/−
Desmin	0/−	0/−	0/−
S-100	0/−	0/−	0/−
CD34	0/−	0/−	0/−
Ki-67	0/+++	0/+++	0/+++

Percent positive staining: 0 = <5%, 1 = 5%-30%, 2 = 31%-60%, 3 = >60%, 4 = >95%. Staining intensity: −, +, ++, and +++.

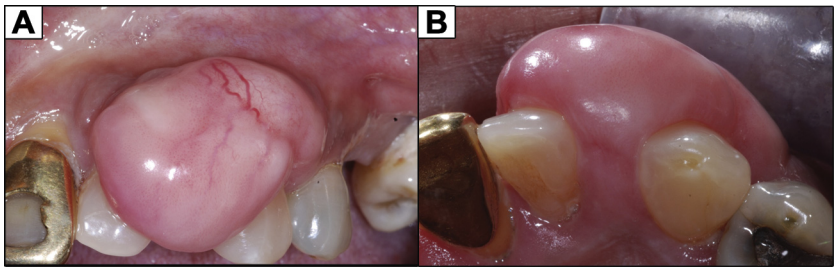

Fig. 1. Case 1, third recurrence: a 50-year-old female with gingival mass. **A,** Depicted are the facial aspect and **B,** an occlusal view of teeth #10 and #11 before treatment.

Case #1

A 50-year-old African American female presented to the Department of Periodontics, School of Dentistry, University of Louisville, with a localized gingival enlargement of the maxillary left lateral canine region in June 2011 (case 1, third recurrence). She reported that this lesion first appeared about 20 years prior and was removed. When the lesion recurred for the first time about 2 years later in 1993 (case 1, first recurrence), a 1-year history of a 1 cm firm, pink swelling was reported. No radiographic changes were described. The lesion was excised and diagnosed as "fibrosis." The patient indicated that it grew back for the third time and was removed in 2005 at another hospital for which records were not available. Over the ensuing 5 years, she reported that it had been slowly enlarging (third recurrence in 2011) with accelerated growth in the last month. No pain or paresthesia was reported. The patient was not taking any medications at the time of the biopsy, and there was no family history of gingival enlargement.

On examination, a 1.7 × 1.3 cm, pink, smooth, firm nodular mass of the maxillary facial gingiva of teeth #10 and #11 and mesial gingiva of #12 was noted (Figure 1). Interproximal extension of the lesion between #10 and #11 and separation of the crowns was observed. Teeth #9 and #10 were slightly mobile, there was no pain to palpation, and all teeth in the region were percussion negative. A periapical radiograph (Figure 2) showed

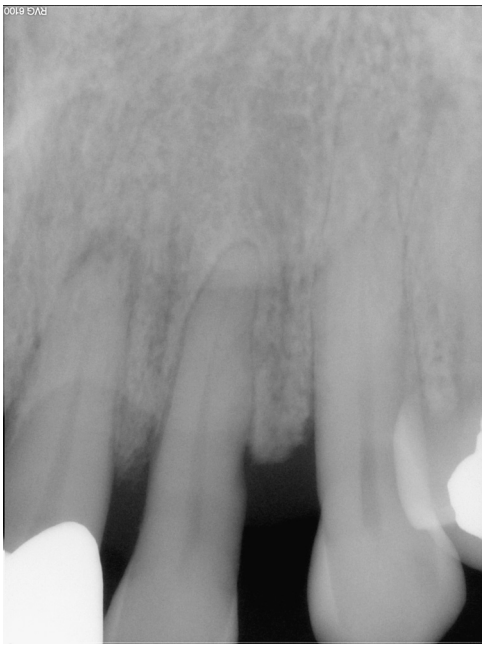

Fig. 2. Case 1, third recurrence: Preoperative periapical radiograph shows horizontal bone loss, slight widening of the periodontal ligament around the apical region of #9, and subtle sclerosis. No apparent bony alteration due to the gingival lesion is noted.

mild horizontal bone loss between teeth #9 and #11, slight widening of the periodontal ligament space at the apex of tooth #9, and mild peripheral bone sclerosis. The