Fibroepithelial hyperplasia: Rare, self-limiting condition - Two case reports

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Abstract:
Objectives: Fibro-epithelial hyperplasia is a histological variant of fibroma and a proliferative fibrous lesion of the gingival tissue that causes esthetic and functional problems. Methods: This article addresses the diagnosis and treatment of two cases of fibro-epithelial hyperplasia. Results: These lesions are a result of trauma/chronic irritation, or arise from cells of periodontium, periodontal ligament, or periosteum. Conclusion: The cases demonstrate the need for awareness, and role of biopsy and histologic evaluation in management of these lesions KEYWORDS: Fibroepithelial hyperplasia, ossifying fibroma, traumatic, pyogenic granuloma, inflammatory hyperplasia, neoplasia.
Key words: Oral mucosal lesions, Oral pathology, Periodontal diseases

Introduction:
Oral mucosa is constantly subjected to external and internal stimuli and therefore manifests a spectrum of disease that range from developmental, reactive, and inflammatory to neoplastic. ¹ These lesions present as either generalized or localized.¹,² Reactive lesions are clinically and histologically non-neoplastic nodular swellings that develop in response to chronic and recurrent tissue injury which stimulates an exuberant or excessive tissue response.³ They may present as pyogenic granuloma, fibrous epulis, peripheral giant cell granuloma, fibroepithelial polyp, peripheral ossifying fibroma, giant cell fibroma, pregnancy epulis; and commonly manifest in the gingiva.²,³,4,5,6,7,8,9,10 Such reactive lesions are less commonly present in other intraoral sites such as cheek, tongue, palate and floor of the mouth.¹¹,¹² Clinically, these reactive lesions often present diagnostic challenges because they mimic various groups of pathologic processes. They are clinically similar but possess distinct histopathological features. They may be termed 'epulides' when the connective tissue proliferation which occurs is confined to the gingiva.¹³,¹⁴ According to Romer,¹³ the term epulis was first employed by Galen to designate a tumor on the gums. The term as used by him applied generally to any kind of abnormal gingival growth. In more recent times its use has been restricted, as a rule, to certain types of growth found in this region of the oral cavity, although some writers still use the word in its more general meaning. Reactive lesions of the gingiva have been classified on the basis of their histology.¹⁵,¹⁶ Kfir et al.⁸ have specifically classified reactive gingival lesions into pyogenic granuloma, peripheral giant cell granuloma, fibrous hyperplasia and peripheral fibroma with calcification. A report of more than 30,000 oral biopsies submitted for diagnosis observed that nearly 13% were taken from the gingiva.¹⁷,¹⁸,¹⁹,²⁰ Several authors¹⁰,²¹,²²,²³ believed that many of these lesions are true fibromas, whereas Cooke 1956 ²⁴ believed that, they are reactive in nature as the cause being local irritation. Barker and Lucas 1967²⁵ examined 650 fibrous overgrowths of the oral cavity and felt that only 2 could be considered neoplasms. Daley et al.1990¹³ suggested the term, focal fibrous hyperplasia” which implies a reactive tissue response, is preferable to the term, fibroma” which implies incorrectly, a benign neoplastic proliferative fibrous connective tissue.²⁶,²⁷

Fibroma/fibro epithelial hyperplasia:
Almost all lesions in the oral cavity that are called fibromas are not true neoplasms, but merely fibrous overgrowths caused by chronic irritation. Many authors therefore prefer the term fibroepithelial polyp or fibrous hyperplasia for these type of lesions.²⁶ Axell (1976)²³ encountered prevalence of
3.25% for fibromas in the adult Swedish population. They rarely occur before fourth decade and show no preference for either sex.23

Fibroma is the most common oral fibrous growth. It represents focal fibrous hyperplasia due to trauma or local irritation. Found in 1.2% of adults, it is the most common oral mucosal mass submitted for biopsy and is composed of Types I and III collagen.28, 29. Gingival lesions are also common, although they probably result from chronic infection rather than trauma. The lesion presents as painless, sessile, round or ovoid, broad-based swelling, lighter in colour than surrounding tissue due to a reduced vascularity. The surface may be ulcerated and diameter varies from 1 millimeter to several centimeters. Treatment is surgical excision and a low recurrence rate is expected.30, 31. Differential diagnosis of fibroma includes: giant cell fibroma, neurofibroma, peripheral giant cell granuloma, mucocele, lipoma, or salivary gland tumor. Histologically, fibroblasts scattered in a dense, collagenous matrix, mild chronic, inflammatory infiltrate may be present, but is not a consistent finding.30, 31

Irritation fibroma, or traumatic fibroma, is a common submucosal response to trauma from teeth or dental prostheses and was first reported in 1846 as fibrous polyp and polypus. Although at that location they probably resulted from chronic infection rather than trauma. A number of variations on the theme of inflammatory fibrous hyperplasia are mentioned in the following discussion.

Here we present two cases of fibroma manifesting as inflammatory hyperplasia rather than a true neoplastic lesion of connective tissue origin. They exhibit a proliferative stratified squamous epithelium different from a classic fibroma where a stretched and flattened epithelium is observed. Thus, this indicates a need to differentiate between hyperplasia and neoplasia for correct therapeutic management.

Case report 1:

A 42 year old male patient (Figure 1) reported with the chief complaint of swelling in the right front region of the lower jaw since 5 years which was unaesthetic and hindered the chewing from lower front teeth. The swelling started as a small painless growth 5 years back gradually increasing to the present size. Her medical history was non contributory. No abnormality was detected on extra-oral examination. Intra-oral examination revealed fair oral hygiene. 12 had a probing depth of 5 mm with Grade III gingival enlargement. Enlargement was sessile, ovoid and pinkish with smooth surface extending mesio distally (Figure 2). All the inspectory findings were confirmed with palpation. It was firm, non-tender, non-reducible and non-compressible with no bleeding on probing.

Case report 2:

A 37 year old female patient (Figure3) reported with the chief complaint of swelling in the right front tooth region of the upper jaw (Figure 4) since 4 years, hampering her aesthetics. It started as a small painless growth 4-5 years back gradually increasing to the present size. Her medical history was non contributory. No abnormality was detected on extra-oral examination. Intra-oral examination revealed fair oral hygiene. 12 had a probing depth of 4mm with Grade III gingival enlargement. Lesion was sessile and pinkish with smooth surface extending 1.1cm buccolingually and 2.4cm mesiodistally. All inspectory findings were confirmed with palpation. It was firm, non-tender, non-reducible and non-compressible with no bleeding on probing.

In both the cases cross bite was present in relation to 12 and 42 with Angle’s bilateral class I molar relation. Based upon clinical findings, the lesions were provisionally diagnosed as traumatic fibroma. Differential diagnosis included inflammatory hyperplasia and peripheral ossifying fibroma.

Intra-oral periapical radiograph (Figure 5), and mandibular occlusal view (Figure 6) were taken which showed moderate amount of bone loss in relation to 42 and 43 with drifting of these teeth in case 1 and no bone loss around 12 in case 2 (Figure 7).

Phase I therapy with supragingival scaling was done. Patients were put on chlorhexidine mouthwash twice daily for 1 week and were advised complete blood investigations. All biochemical investigations Complete Blood Picture (CBP), Random Blood Sugar (RBS), Human Immuno deficiency Virus (HIV) I&II-tridot method were normal. Coronoplasty for lower anteriors with excisional biopsy (Figure 8) of the lesions was performed. Amoxycillin 500mg three times a day for 5 days was prescribed to the patients post-operatively. (Figure 9, 10)

Histopathological examination revealed hyperplastic, acanthotic parakeratinized, stratified squamous epithelium (Figure 11) with elongated and interconnected rete ridges. Underlying connective tissue consisted of dense collagen bundles, fibroblasts, blood vessels and inflammatory cells
Discussion:

Fibrous growths of the oral soft tissues are fairly common and include a diverse group of reactive and neoplastic conditions. Tissue enlargement of the oral cavity often presents a diagnostic challenge because a
A diverse group of pathologic processes can produce such lesions. Within these lesions a group of reactive hyperplasias which develop in response to a chronic, recurring tissue injury stimulates an exuberant or excessive tissue repair response. The term “epulis” is used clinically to describe any localized overgrowth on the gingiva. Histological examination of epulides indicates the vast majority are: Focal Fibrous Hyperplasia (FFH), Peripheral Ossifying Fibroma.
(POF), Pyogenic Granuloma (PG) or Peripheral Giant Cell Carcinoma (PGCG). Multiple polyps of the oral mucosa were described as early as 1881 by Marché. Cooke called all the pedunculated swelling from a mucosal surface as “polyp” (fibro epithelial polyp), where maximum number of lesions occurred on the mucosa in the line of occlusion, and the entire pedunculated and sessile lesion in the gingiva as “epulides” (fibrous epulides), which commonly occurred in the maxillary anterior region. In certain cases the histology may reveal the presence of spindle or stellate cells and multinucleated giant cells both of which appear to be of fibroblastic origin. These lesions have been termed by several authors as “giant cell fibroma”, . They appear in the interdental papilla as a result of local irritation from calculus, caries or restorations with irregular margins. Histologically they are characterized by a focal sub-epithelial mass of fibrous connective tissue composed of interlacing or parallel bundles of collagen, containing occasional vascular channel and variable inflammatory infiltrate. The fibroblast are apically narrow and elongated and relatively few in number. Recurrences of this lesion are uncommon or rare. However Cooke in his review reported recurrence in 3 cases out of 78 biopsy specimens.

Fibro epithelial hyperplasias are reactive/inflammatory conditions and they give rise to variety of lesions named according to their clinical presentation. For example, pyogenic granuloma or fibrous epulis is named so due to the vascularity and presence of ulceration. Most of these lesions arise on gingiva, reflecting universal presence of inflammation in the interdental papillae. Lesions are associated with local predisposing factor like mal-aligned teeth, ill-fitting restorations or calculus which prevent removal of bacterial plaque and indirectly induce inflammation. In both the present cases mal-aligned teeth and sub-gingival deposits were present explaining the possible etiologies.

Histologically, these lesions vary from rapidly growing granulation tissue to mature scar–like tissue, depending on age and vascularity. Lesions are collagenous, composed of mature fibrous tissue with prominent vascular pattern. Epithelial changes also correlate with the lesion’s age and degree of inflammation. Fibro epithelial hyperplasias when inflamed are covered by uniformly hyperplastic epithelium, with arcing rete pattern when ulcerated. Thin spiky or elongated bilaminar rete processes are seen which may penetrate deeply into the connective tissue. This rete hyperplasia is prominent when marked inflammation is present, but in less inflamed lesions epithelium becomes regular with flat basement membrane, or may get atrophied. These hyperplastic conditions are considered self-limiting. But since they interfere with form and function, they need to be excised. Also long standing hyperplastic lesions in the presence of chronic irritation can get converted to neoplasia.

A substantial overlap exists between the various histological types of reactive focal gingival hyperplastic lesions. The frequent gingival site occurrence supports an assertion that these hyperplastic lesions are the same lesions at different developmental stages. Daley et al suggested that the vascular component of pyogenic granuloma is gradually replaced by fibrous tissue with time and, hence, diagnosed as a fibrous hyperplasia or fibroma. In addition, Natheer Al- Rawi observed that fibrous hyperplasia on the gingiva not only have the same female gender preponderance but occur in the same age group and site as gingival pyogenic granuloma. An inference that fibrous hyperplasia represents a fibrous maturation of pyogenic granuloma especially in lesions with long duration was therefore made to support the assertion.

Identification of any reactive hyperplastic gingival lesion requires the formulation of a differential diagnosis to enable accurate patient evaluation and management. These lesions must be separated clinically and histologically from precancerous, developmental and neoplastic lesions. Differential diagnoses include metastatic tumours in the oral cavity, angiosarcomas, gingival non-Hodgkin’s lymphoma, Kaposi’s sarcoma and haemangioma. Metastatic lesions in the oral cavity may be the first indication of an undiscovered malignancy at a distant site . The clinical appearance of these lesions in most cases has a striking resemblance to reactive gingival lesions most especially pyogenic granuloma. In addition, the clinical appearance of non-Hodgkin’s lymphoma and small haemangiomas may also be clinically indistinguishable. Hodgkin’s lymphoma in the gingiva may present as an asymptomatic reactive gingival lesion.

Some authors reported that apart from irritation some drugs can also induce fibro-hyperplastic gingival overgrowth. George P.et al reported six patients with gingival fibrous hyperplasia associated with cyclosporin A (CyA) therapy. Arvio P.et al reported gingival overgrowth in patients with Aspartylglucosaminuria (AGU). It is a lysosomal storage disorder with main symptom as progressive mental retardation. Of 16 oral mucosal lesions studied histologically by them, 15 represented fibroepithelial or epithelial hyperplasias and were reactive in nature.
Furthermore, the term fibro-epithelial hyperplasia should not be confused with focal epithelial hyperplasia which is caused by HPV virus. Focal epithelial hyperplasia, all alterations occur in the epithelial layer of the mucosa with virtually no alteration in the underlying connective tissue. The histopathological features are quite distinct but considerable overlap still exists among these lesions.

**Conclusion:**

The myriad histological entities that we observe of reactive hyperplasia may be due to connective tissue response to varied intensities of gingival irritation. This response may be influenced by the serum levels of certain endocrine hormones. Distinction between hyperplasia and neoplasia needs to be clearly defined as neoplasias are not self-limiting conditions and long standing hyperplastic lesions in presence of chronic irritation can get converted to neoplasia. In addition to the physical characteristics of the lesion, the patient’s demographics, presence of associated symptoms, related systemic disorders, and location and growth patterns of the lesion all give clues to adequately diagnose and treat their typical histopathologic architecture.

A biopsy will ensure a better and a more ideal treatment plan for the patient and prevent recurrence of these lesions. Molecular analysis allows a more conclusive diagnosis with polymerase chain reaction (PCR) as a useful tool. Treatment modalities commonly practised include scalpel surgery, cryotherapy, cauterization. Laser therapy is currently being undertaken and would soon become the norm, as it has many advantages like hemostasis, more patient comfort and better healing.

Because the condition described here, is of both epithelial and connective origin, chances that it may transform into neoplasia are quite high. Further studies are needed to confirm this hypothesis with longer follow up of the patients.

**References:**


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