

Gingival Oral Focal Mucinosi s Associated with Severe Periodontal Destruction and Metabolic Dysregulation: A Rare Clinicopathological Case Report

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Abstract

Oral focal mucinosis (OFM) is a rare benign connective tissue lesion regarded as the oral counterpart of cutaneous focal mucinosis and often mimics inflammatory or reactive gingival enlargements clinically. This case report highlights a rare presentation of gingival OFM associated with severe periodontal destruction and metabolic dysregulation in a 49-year-old male patient. The patient presented with mobility of mandibular anterior teeth and weakening of the gums for the past two months. Clinical examination revealed localized erythematous gingival enlargement associated with deep periodontal pockets, attachment loss, and severe alveolar bone loss. Hematological and biochemical investigations demonstrated elevated levels of erythrocyte sedimentation rate, HbA1c, and dyslipidemia. The patient underwent scaling and root surface debridement as a part of periodontal therapy followed by provisional splinting and excisional biopsy of the lesion. Histopathological examination revealed loosely arranged myxoid connective tissue with spindle and stellate fibroblasts suggestive of oral focal mucinosis. Periodontal flap surgery and supportive periodontal maintenance therapy were subsequently performed. Follow-up evaluation at 8 months demonstrated satisfactory healing without recurrence. This case highlights the diagnostic challenge associated with gingival OFM and underscores the significance of clinical and histopathological examination together with appropriate periodontal management to achieve successful treatment outcomes.

Keywords

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Introduction

Oral focal mucinosis (OFM) represents an unusual benign connective tissue lesion of the oral cavity, initially described by Tomich in 1974 as the oral equivalent of cutaneous focal mucinosis[1]. The histopathological features include focal myxoid

changes in the connective tissue, resulting from increased hyaluronic acid production by fibroblasts and reduced collagen formation. This alteration results in the accumulation of a mucin-rich extracellular matrix within the connective tissue stroma, producing characteristic myxomatous changes microscopically [2].

OFM is considered an uncommon lesion, with relatively few cases reported in the literature. Clinically, the lesion usually appears as a slow-growing, asymptomatic, sessile or pedunculated soft-tissue enlargement with a smooth, normal-appearing surface or slightly erythematous mucosal coloration. Because of the absence of distinctive clinical features, OFM is frequently misdiagnosed as other reactive or inflammatory gingival lesions such as irritation fibroma, pyogenic granuloma, peripheral ossifying fibroma, peripheral giant cell granuloma, or inflammatory gingival hyperplasia[2,3]. In some cases, the lesion may also mimic periodontal or soft tissue pathologies associated with underlying inflammatory changes. Therefore, histopathological evaluation remains the definitive diagnostic standard.

The gingiva is the most commonly affected intraoral site, followed by the palate, tongue, and alveolar mucosa. Previous studies have reported a female predilection and a greater incidence in middle-aged adults [4,5]. Although OFM generally presents as a localized benign lesion without significant bone involvement, unusual presentations associated with periodontal destruction are exceedingly rare. The present case is noteworthy because the lesion was associated with severe periodontal breakdown, advanced attachment loss, tooth mobility, uncontrolled diabetes mellitus, dyslipidemia, and elevated ESR, a non-specific systemic inflammatory marker thereby clinically simulating an inflammatory periodontal lesion. Such coexistence of OFM with significant periodontal destruction and metabolic dysregulation has been rarely documented in the literature. This report emphasizes the importance of comprehensive clinical, radiographic and histopathological evaluation for accurate diagnosis and appropriate interdisciplinary management.

Case Report

A 49 year old male patient presented with a chief complaint of mobility of lower front teeth and weakening of gums for two months to the Department of periodontics. Detailed anamnesis revealed that the patient was a diabetic and had hypercholesterolemia for the past three years and was under medication. The patient reported that he had undergone scaling, 3 years back for bleeding gums. Furthermore, the patient disclosed that he consumes alcohol occasionally approximately once a month, for the past 10 years. He consumes mixed diet and brushes once daily using a hard bristled toothbrush.

Extraoral examination was non-contributory. Intraoral examination revealed a localized erythematous gingival enlargement in relation to the mandibular anterior region involving teeth 42 and 43. The lesion appeared sessile, non-tender, edematous, and soft to slightly firm in consistency with focal reddish areas over the surface (Figure 1). Periodontal examination demonstrated severe periodontal destruction associated with deep periodontal pockets and advanced clinical attachment loss in the mandibular anterior region. The probing pocket depth measured approximately 10-11 mm, while clinical attachment loss measured approximately 10-13 mm in relation to mandibular lower anteriors. Grade III mobility was observed in 41,42 and 43, Grade II mobility was observed in 31 and 32. A provisional diagnosis of pyogenic granuloma was given based on the clinical findings and a differential diagnosis of peripheral giant cell granuloma, fibrous epulis, oral sarcoidosis was considered. Panoramic radiographic examination demonstrated severe alveolar bone loss in the mandibular anterior region corresponding to the involved teeth (Figure 2).

Hematological investigations revealed elevated total leukocyte count (11,780 cells/cmm) and markedly elevated erythrocyte sedimentation rate (71 mm/hour). Biochemical investigations demonstrated poorly controlled diabetes mellitus with fasting plasma glucose level of 203 mg/dL and postprandial plasma glucose level of 251 mg/dL. Lipid profile analysis revealed elevated triglycerides (227 mg/dL), elevated LDL cholesterol (119.6 mg/dL), elevated VLDL cholesterol (45.4 mg/dL), reduced HDL cholesterol (33 mg/dL), and increased cholesterol/HDL ratio (6.0).

The initial periodontal treatment included Phase I therapy with oral hygiene instructions, plaque biofilm control, dietary counseling, scaling and root planing, and provisional splinting of mandibular anterior teeth. Following reevaluation after one-month, excisional biopsy of the lesion was performed.

Histopathological examination revealed stratified squamous epithelium overlying connective tissue exhibiting loosely arranged collagen fibers within a myxoid stroma. Numerous spindle-shaped and stellate fibroblasts were evident within the connective tissue matrix. Peripheral areas showed relatively dense collagen bundles. Dilated capillary-sized blood vessels, extravasated red blood cells, and focal chronic inflammatory infiltrate were also observed (Figure 3). These findings were suggestive of oral focal mucinosis. Subsequently, Phase II periodontal therapy

involving periodontal flap surgery for pocket management was performed in the second and fourth quadrants. The patient was later placed under supportive periodontal maintenance therapy.

Follow-up examination at 3 months demonstrated satisfactory healing with no evidence of recurrence (Figure 4). Six-month follow-up revealed stable periodontal healing and complete resolution of the lesion without recurrence (Figure 5).

Figure 1. Clinical photograph showing localized erythematous gingival enlargement in relation to mandibular anterior teeth.



Figure 2. Panoramic radiograph demonstrating severe alveolar bone loss in the mandibular anterior region.



Figure 3. Histopathological photomicrograph showing loosely arranged myxoid connective tissue stroma with spindle and stellate fibroblasts suggestive of oral focal mucinosis.

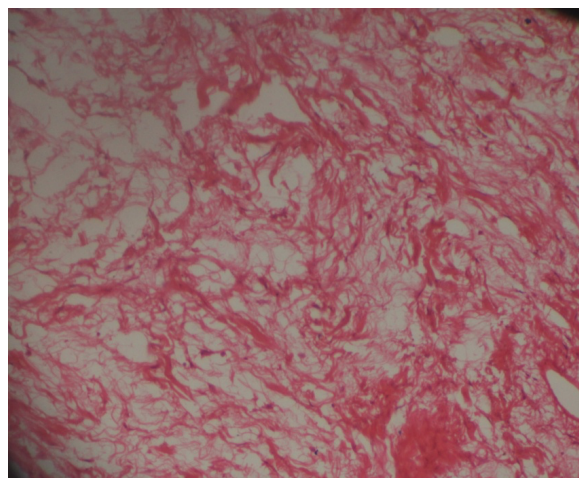


Figure 4. Three month postoperative follow-up demonstrating satisfactory healing and absence of recurrence.



Figure 5. Six month postoperative follow-up showing stable periodontal condition and complete healing.



Discussion

Oral focal mucinosis is an uncommon connective tissue lesion attributed to increased fibroblast mediated hyaluronic acid production accompanied by decreased collagen synthesis [1,2]. Histologically, this results in focal myxoid degeneration of connective tissue characterized by loosely arranged myxomatous stroma containing spindle and stellate fibroblasts [6].

The lesion commonly affects keratinized mucosa overlying bone, particularly the gingiva and palate [4,7]. Although OFM demonstrates female predilection in most studies, the present case occurred in a middle-aged male patient [4,5].

In the present case, the lesion clinically simulated inflammatory gingival enlargement due to associated erythema, severe periodontal destruction, mobility, and deep periodontal pockets. The clinical differential diagnosis of the present lesion included pyogenic granuloma, irritation fibroma, peripheral ossifying fibroma, peripheral giant cell granuloma, and oral focal mucinosis[2,3,8]. Pyogenic granuloma was considered because of the erythematous gingival enlargement and inflammatory appearance; however, histopathological examination did not reveal granulation tissue or marked vascular proliferation characteristic of pyogenic granuloma. Irritation fibroma was also considered due to the localized gingival enlargement, but the absence of dense collagenized connective tissue ruled out this lesion. Peripheral ossifying fibroma was excluded because histopathological examination did not demonstrate areas of calcification or ossification within the connective tissue stroma. Peripheral giant cell granuloma was considered because of the gingival location and erythematous appearance; however, multinucleated giant cells were absent microscopically. Histopathological examination ultimately revealed loosely arranged myxoid connective tissue containing spindle-shaped and stellate fibroblasts, which was consistent with oral focal mucinosis.

Histopathological evaluation remains the diagnostic gold standard, as clinical diagnosis alone can be challenging [4,9]. Characteristic histopathological features include myxoid connective tissue, stellate fibroblasts, loosely arranged collagen fibers, and Alcian blue-positive mucinous matrix [9,10,13].

The present case additionally demonstrated poorly controlled diabetes mellitus, dyslipidemia, leukocytosis, and elevated ESR, suggesting an underlying systemic inflammatory state. Chronic hyperglycemia and metabolic dysregulation are known to alter fibroblast function, collagen metabolism, inflammatory cytokine activity, and periodontal wound healing [11,12]. These alterations may potentially contribute to extracellular matrix changes and periodontal tissue destruction. The associated severe periodontal destruction observed in the present case represents an unusual presentation of OFM. Chronic periodontal inflammation may have

contributed to altered fibroblastic activity and connective tissue degeneration. However, further studies are required to establish any definitive association between OFM and metabolic disorders.

Complete surgical excision is the preferred treatment for OFM, with recurrence being uncommon[13,14,15]. In the present case, interdisciplinary periodontal management combined with surgical excision resulted in satisfactory healing without recurrence during follow-up.

Conclusions

Oral focal mucinosis represents a rare, benign lesion of connective tissue with nonspecific clinical presentation that may mimic inflammatory and reactive gingival enlargements. Gingival OFM associated with severe periodontal destruction and metabolic dysregulation is particularly unusual and diagnostically challenging. Histopathological examination remains indispensable for definitive diagnosis. Early interdisciplinary management involving periodontal therapy, surgical excision, and supportive periodontal maintenance contributes to favorable clinical outcomes and reduced risk of recurrence.

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