

Management of Patients with Foreign Body Gingivitis: Report of 2 Cases with Histologic Findings

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A b s t r a c t

Foreign body gingivitis is an inflammation of the gingiva, characterized by foci containing particles of foreign material in the connective tissue, which can have either a granulomatous or a lichenoid microscopic appearance. In clinical terms, it differs from other immune-mediated gingival disorders in its limited involvement of tissues other than the gingiva, as well as its relative resistance to treatment by topical corticosteroids. Two cases are presented, with a review of the clinical features, including characteristic desquamation and mottling of the marginal gingiva and symptoms of localized tenderness and pain; gingival recession was observed in both of the reported cases. Histologic examination revealed damaged epithelium and degeneration of the basal layer, as well as a mixed inflammatory cell infiltrate in the connective tissue with refractile or opaque particles of foreign material. Gingival inflammation and the severity of gingival erosions improved dramatically with careful debridement, improved home care and more frequent, diligent periodontal maintenance therapy. Free gingival grafts, together with excision of affected tissues, served to stabilize and reinforce the marginal tissues, as well as eliminating further clinical signs of the disease; excision alone was not as effective. Patients require careful dental and periodontal management as well as appropriate oral home care to avoid further mechanical damage to the gingiva; in addition, the use of dental abrasives and polishing agents should be restricted, particularly if gingival lesions are present. Home care recommendations include avoidance of dentifrices with certain chemical additives and rinses with a high alcohol content.

MeSH Key Words: case report; gingivitis/pathology; foreign bodies/complications; granuloma, foreign-body/complications

© J Can Dent Assoc 2005; 71(2):105-9
This article has been peer reviewed.

Foreign body gingivitis (FBG) is an inflammation of the gingival tissues associated with the presence of foreign material in the gingival connective tissues.¹ Its clinical presentation is characterized by solitary or multiple red or red and white lesions, which may resemble and be mistaken for lichen planus; the affected tissues may appear ulcerated. The condition is much more common among women (mean age of 48 years), and patients may complain of swelling or pain in the region.^{1,2} The lesions usually involve the marginal and attached gingiva; the interdental papillae are also commonly affected. In some cases the onset of lesions can be correlated with recent dental treatment involving the proximal use of dental restorative materials or dental prophylaxis agents. The

condition often persists despite conventional periodontal therapy and excellent oral hygiene.¹

Because the clinical appearance of FBG resembles that of other desquamative gingival disorders, a definitive diagnosis requires microscopic evaluation of the tissue, possibly supplemented by another form of analysis, to confirm the presence and nature of foreign materials. Histologic examination characteristically shows granulomatous inflammation, with an intense lichenoid lymphocytic infiltrate. There may be well-formed granulomas, sometimes containing multinucleated giant cells, or only focal collections of histiocytes.¹ Foreign bodies in the granulomas vary in size, but most are less than 5 µm in diameter. They may be opaque or refractile or both. The most frequently identified

element in the foreign material is silicon, followed by aluminum and titanium, with several other elements found in fewer specimens.³ It has been suggested that FBG may be caused by the entry of dental materials into the connective tissues.¹⁻³

Previous work indicates that the condition persists despite conventional periodontal therapy and excellent oral hygiene and that the only successful treatment for FBG is surgical excision of the affected tissues.¹ The clinical findings and treatment histories of 2 patients, presented here, provide some insight into the response of the affected tissues to periodontal therapies; suggestions for management of similar cases are also provided.

Case Reports

Case 1

The first patient was a 40-year-old female nonsmoker who was in good general health and was not using any prescription medications. For the 6 months preceding her referral to our office, she had reported acute soreness in the gum region and aggressive bleeding when she brushed her teeth. She usually visited her dentist every 3 to 6 months for scaling and prophylaxis. Her most symptomatic gingival regions were the labial areas of the anterior maxilla, and the mandibular buccal premolar areas. Because of these problems, she found home dental hygiene difficult and discouraging.

Oral examination revealed that the gingiva was irregularly mottled, with red and white patches; desquamation and marginal ulceration were present at many sites (Fig. 1). The interdental papillae were flattened or missing entirely in some areas. Plaque control was fair overall, but poor in the regions most affected, especially interproximally. Supragingival calculus was light, but subgingival calculus deposits were moderate in many interproximal areas. Pocket probing depths were up to 5 mm anteriorly and 6 mm for the interproximal areas between molars; bleeding on probing was generally significant and heavy in regions of marginal gingival ulceration. The gingiva at these sites was atrophic and poorly keratinized, with generalized labial and buccal recession; there was no attached gingiva remaining at several sites, and there was significant root exposure. There were a few localized mobilities, up to grade 1, with Class I buccal furcation involvements. Initial diagnosis was early to moderate periodontitis, with recession and inadequate attached gingiva, complicated by a desquamative gingival disorder, suspected to be erosive lichen planus.

Incisional strip biopsy of sore, red buccal gingiva was performed before initiation of periodontal debridement. Histologic examination showed fibrous connective tissue containing a patchy chronic inflammatory infiltrate consisting primarily of plasma cells, with pockets of lymphocytes. The overlying epithelium exhibited atrophy,

with liquefactive degeneration of cells of the basal layer. Refractile fragments of foreign material were present within the connective tissue (Fig. 2). Topical use of a corticosteroid ointment (0.05% fluocinonide) was prescribed, to help relieve inflammation-mediated tissue pain; tenderness of the affected tissues improved with regular use of the medication, but tissue quality did not visibly change.

The patient's initial periodontal therapy consisted of quadrant root planing under local anesthetic, followed by appropriate instruction about oral hygiene. Instrumentation was thorough and definitive, but great effort was made to minimize soft-tissue trauma. Unfortunately, improvement in hygiene was incremental; therefore, a chlorhexidine rinse (0.12%) was also prescribed, to be used for 1 to 2 weeks after root planing. The response to initial therapy was good, with a significant reduction in general gingival inflammation, as evidenced by resolution of most of the severe initial gingival redness, swelling and soreness. The number of bleeding points, as well as the intensity of bleeding on probing, was significantly reduced. Some erythema of the marginal gingiva persisted in the anterior region and in many interproximal areas (most notably where the interproximal papillae were initially damaged).

Surgical therapy for this patient consisted of free gingival grafts for the areas judged to be most receded and unstable, the lower canine and premolar buccal regions. The posterior palatal gingiva was deemed clinically healthy and suitable as a donor site, as it showed no signs of inflammation or desquamation. Grafted sites healed well, and there was no evidence of recurrence of the initial gingival symptoms in the grafted areas up to 5 years later (Fig. 3). Two years after the initial procedures, the maxillary anterior labial and palatal gingiva still showed significant inflammation and ulceration. Because the mandibular gingival grafts had been so effective, a free gingival graft was also placed in this area (Fig. 4), in addition to labial gingival stripping (despite an adequate band of gingiva on the labial surface of the maxillary incisors). On the palatal aspect of this affected area, the tissue was thicker and more hyperplastic; therefore only a long-bevel gingivectomy was performed. Both sites initially healed well, but within a year much of the palatal gingiva exhibited recurrence of the initial redness, although signs of desquamation were reduced (Fig. 5). The labial aspect remained mostly free of inflammation.

The patient continued with a rigid maintenance schedule and was seen at the authors' office every 2 months. At the time of writing, 5 years after the surgery, the gingival tissues remain quite tender to the touch; therefore, topical or local anesthetic is applied liberally at each periodontal maintenance visit to allow adequate instrumentation. The tooth and root surfaces can be fully instrumented, but no dental polishing agents are used.

Case 1



Figure 1: On initial presentation, there was significant gingival erythema, loss of interdental papilla and marginal ulceration.

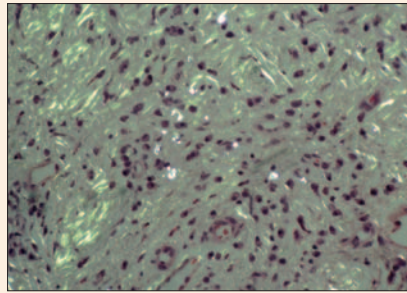


Figure 2: Many of the foreign particles exhibited birefringence when viewed by polarized light; this appearance indicated a crystalline structure. (Hematoxylin and eosin, magnification $\times 150$.)



Figure 3: Site of incisional biopsy (see Fig. 2 for histology), 5 years after free gingival grafts were placed in the buccal region of teeth 33 and 34.



Figure 4: After root planing and placement of free gingival grafts for teeth 21 and 22 (labial surface), most of the initially damaged interproximal papilla between the central and lateral incisors has re-formed.



Figure 5: Residual inflammation and interproximal palatal ulceration, at 12 months after the gingivectomy procedures.

Case 2

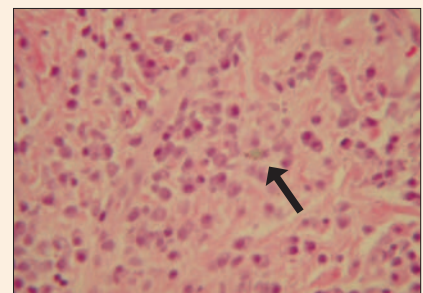


Figure 6: Histologic examination of chronic inflammatory cell infiltrate shows plasma cells, lymphocytes and histiocytes. There is a large refractile fragment in the centre of the slide, which is about 5 μ m in diameter. (Hematoxylin and eosin, magnification $\times 300$.)



Figure 7: Six months after augmentation with a free gingival graft in the premolar area. The anterior region was treated similarly at a later date. There is minimal clinical evidence of inflammatory problems in the grafted area (despite the patient's limited ability to perform dental hygiene), whereas the untreated anterior tissue is atrophic and highly inflamed.

Unfortunately, the patient's ability to perform oral hygiene remains a factor limiting overall periodontal stability, especially in the interproximal regions. The areas with the heaviest deposits appear to be positively correlated with sites of persistent FBG-type tissue response. When the patient experiences an increase in tissue soreness, she applies the corticosteroid ointment selectively. Attempts have been made to prolong the maintenance interval, but all of her gingival tissues appear to react strongly to moderate plaque accumulation.

Case 2

The second patient was a 45-year-old woman with a clear health history who was referred for management of persistent gingival inflammation and progressive gingival recession. She was undergoing scaling and prophylaxis at her general dentist's office every 6 months. She presented with multiple areas of gingival mottling, consisting of darker and lighter red patches, along with small ulcerations or desquamative areas, which were mainly visible marginally and interproximally; the most affected areas were the

anterior gingiva and the premolar buccal regions. Erosive lichen planus was suspected. Her chief complaint was of generally sensitive teeth, with some gum tenderness when she was brushing or eating hard foods; she had not noticed significant bleeding. Pocketing was minimal, but there was significant recession with exposure of roots, especially in the premolar and first molar areas. In these regions in particular, the gingiva was thin and poorly keratinized, and at several sites there was no attached gingiva remaining. The interproximal papillae were intact, without loss of interdental tissue (a feature that contrasted with case 1). Her oral hygiene at presentation was fair, with mostly soft deposits in the gingival third region; subgingival calculus was localized and moderate in the interproximal areas and on the lingual aspects.

At the start of periodontal therapy, an incisional strip biopsy sample was taken from the buccal marginal tissue most affected by the ulcerative lesions. The histologic report described a focally atrophic layer of parakeratinized and nonkeratinized squamous epithelium exhibiting reactive (inflammatory) changes. Focal absence of the basal cell layer was noted. The underlying connective tissue exhibited a mixed inflammatory cell infiltrate, consisting largely of plasma cells, lymphocytes and histiocytes. Numerous small particles of opaque foreign material, interpreted as dental prophylaxis paste, were present within the connective tissue (Fig. 6). The typical features of lichen planus were not seen.

Initial dental and root debridement was performed by half mouth, with local anesthetic. The patient's oral hygiene improved somewhat after extensive instruction, but has remained only fair subsequently. After root planing, the overall redness of the gingival tissues subsided significantly, but the uneven colouring or mottling of the gingiva persisted. No ulcerations could be seen clinically at this stage. Free gingival grafts were placed in the mandibular premolar buccal and anterior areas, which were the sites of the most severe and unstable recession (Fig. 7); these areas healed well, and there has been no progression of recession or recurrence of the ulcerations and redness. At the time of writing, the patient was attending maintenance visits every 3 months; the severity of the FBG type of gingival response becomes exaggerated if the maintenance interval is lengthened. She no longer complains of gingival soreness, but dental sensitivity continues because of the amount of root exposure.

Discussion

FBG should be included in the differential diagnosis of gingival disorders exhibiting desquamation or ulceration. Its clinical features may resemble those of oral lichen planus and other ulcerating immune mucosal conditions, but FBG pathosis tends to be limited to the gingiva, whereas lichen planus typically has a more migrating, widespread mucosal involvement. Localized swelling of the gingiva, often

painful, should also alert the clinician to the possibility of FBG, especially if there is a red and white colouration to the tissue.² It is important to distinguish between various desquamative and lichenoid problems, so that the condition can be treated appropriately, and to rule out disorders with systemic ramifications and malignant or premalignant conditions.^{4,5}

Previous authors have reported the persistence of FBG lesions despite conventional periodontal therapy and excellent oral hygiene.^{1,2} Both of the patients described here exhibited only fair dental hygiene, and there were significant accumulations of hard and soft subgingival bacterial deposits. A more critical assessment of hygiene status, particularly of subgingival deposits and root roughness, is needed for patients with FBG and other desquamative gingival conditions. When oral hygiene measures are complicated by pain and bleeding, inferior plaque control is inevitable and must be counteracted by more thorough and frequent professional instrumentation, as well as oral hygiene measures tailored to the patient's specific needs. These observations are consistent with the suggested management of other desquamative gingival disorders.^{6,7} The presence of dental plaque also adversely affects the course of oral lichen planus, and intensive oral hygiene procedures result in improvement in these lesions.^{8,9}

Surgical excision of affected tissues has been the only recommended treatment for FBG.¹ Although gingivectomy or surgical stripping may be the best option, when thick or hyperplastic tissues are involved, overlaying the damaged sites with healthy gingiva may be a better option for persistently erosive areas or when the gingival complex is atrophic. In case 1, the labial maxillary gingiva, where a gingival graft was overlaid, remained much healthier than the palatal area of the same region, where only an excisional procedure was performed. It has been demonstrated that if healthy tissue is transplanted to an area of oral lichen planus, that area will remain free of lesions and appear clinically healthy.¹⁰ In patients with lichen planus, free gingival grafts have been used successfully to reinforce marginal dental soft tissues and to help stabilize recession.^{11,12} In cases of more severe gingival fragility, typically encountered in cases of cicatricial pemphigoid and epidermolysis bullosa, coverage of exposed roots may be possible.^{13,14} The latter oral conditions, manifesting as desquamative gingivitis, have a systemic cause; therefore, finding donor tissues (for gingival grafting procedures) that are unaffected by the disease should theoretically be problematic. On the other hand, because the cause of FBG is more localized, gingival grafting with tissues taken from distant, unaffected sites should be successful.

The use of topical corticosteroids with other types of desquamative gingival conditions such as erosive lichen planus or cicatricial pemphigoid is common,^{4-6,13} but for

control of FBG, potential of these drugs appears limited. When a lichenoid type of inflammation is noted, as in case 1, corticosteroids may provide limited short-term relief of symptoms.²

The foreign bodies found in FBG tissue samples are usually consistent with dental materials, most frequently abrasives and often dental restorative materials.³ Prevention would be the ideal solution to the problem, and restorative dentists should therefore exercise additional caution when finishing or polishing restorations close to the soft tissues. Patients with gingival ulcerations or recent oral soft-tissue trauma might be at particular risk for impregnation of foreign materials; therefore, unless a rubber dam can effectively isolate the procedure site, treatment should be delayed until the epithelium is clinically intact. Dental prophylaxis, particularly air abrasion polishing, has the potential to direct particles against the gingival tissues with significant force and speed. If the marginal tissues are highly inflamed, or any ulceration is noted, thorough scaling should be performed without these adjunctive polishing procedures. Patients should be informed that effective removal of supragingival and subgingival plaque and calculus is the most beneficial part of their periodontal maintenance, not the dental polishing at the end of their appointment.

Oral hygiene procedures must be modified for patients with frequent or chronic gingival ulcerations, to maximize effectiveness despite the tissue tenderness and to minimize the potential for impregnation of foreign substances in the tissues. Home care efforts must be minimally aggressive, to prevent further gingival abrasion and to minimize gingival recession. An ultrasoft manual toothbrush provides more tactile control than an electric brush for patients with fragile and tender gingiva. Given that foreign body reactions to toothpaste abrasives have been demonstrated experimentally,¹⁵ brushing without a dentifrice is recommended if gingival erosions are present. A dentifrice with abrasive particles of high solubility, such as sodium bicarbonate, may be less likely to impregnate tissues. Various toothpaste ingredients cause oral ulcerations or gingival irritation, including toothpaste detergents^{16,17} and tartar control formulas.^{18,19} If a patient has suspected sensitivity to these agents, or if oral ulcerations are present, avoidance of products containing these chemical agents is recommended. Chemical antibacterial rinses may be necessary to assist with superficial plaque control when tissue soreness is severe, but products with high alcohol content should be avoided, to minimize chemical irritation and desiccation of the affected tissues.

Although FBG resembles other oral mucosal desquamative disorders, it has a distinct histologic profile. Careful and specific management is required to control symptoms and to limit periodontal tissue damage. ♦



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The authors have no declared financial interests.

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