The gingival Stillman's clefts: histopathology and cellular characteristics

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Summary

Aim of the study. **Stillman's cleft is a mucogingival triangular-shaped defect on the buccal surface of a root with unknown etiology and pathogenesis. The aim of this study is to examine the Stillman's cleft obtained from excision during root coverage surgical procedures at an histopathological level.**
Materials and method. **Harvesting of cleft was obtained from two periodontally healthy patients with a scalpel and a bevel incision and then placed in a test tube with buffered solution to be processed for light microscopy.**
Results. **Microscopic analysis has shown that Stillman's cleft presented a lichenoid hand-like inflammatory infiltration, while in the periodontal patient an inflammatory fibrous hyperplasia was identified.**
Conclusion. **Stillman's cleft remains to be investigated as for the possible causes of such lesion of the gingival margin, although an inflammatory response seems to be evident and active from a strictly histopathological standpoint**.

Key words: Stillman's cleft, recessions, gingival margin, histological analysis, inflammation.

Introduction

Stillman's cleft is a mucogingival triangular-shaped defect predominantly seen on the buccal surface of a root, first described by Stillman as a recession related to occlusal trauma, either associated with marginal gingivitis or with mild periodontitis (1). It can be found as a depression or as a sharply defined fissure extending up to 5-6 mm of length (Fig. 1). This particular type of ulcerative gingival recession occurs as single or multiple cleft and it can be classified as *simple* (one direction shape) or *composed* (multiple and differently directed shape) (2, 3). Other possible etiological factors are assumed to be periodontal inflammation (2), which leads to proliferation of the pocket epithelium into the gingival corium and its subsequent anastomosis with the outer epithelium (4). In addition, the traumatic tooth-brushing and the incorrect use of the interdental floss have been described among the possible causes (5, 6). A recent systematic review concluded that although the majority of the observational studies confirmed a relationship between tooth brushing and gingival recessions the data to support or question the association are inconclusive (7). To date, the etiology and pathogenesis of this defects remain unclear even though the assumptions are related to chronic factors that ulcerate the epithelium and healing occurs through the anastomosis of the external and internal epithelium in the gingival sulcus, creating a triangular defect (8). When flossing trauma is involved, superficial gingival tissue clefts are 'red' because the injury is confined within connective tissue. In this case the lesion is reversible: flossing procedures have to be interrupted for at least 2 weeks and chemical plaque control only (i.e. chlorexidine rinses) should be performed. If the cleft appears 'white' the whole connective tissue thickness is involved and the root surface becomes evident; in this case the gingival lesion is irreversible (9, 10).
In case of Stillman's clefts, home oral hygiene could become very difficult to be performed and bacterial or

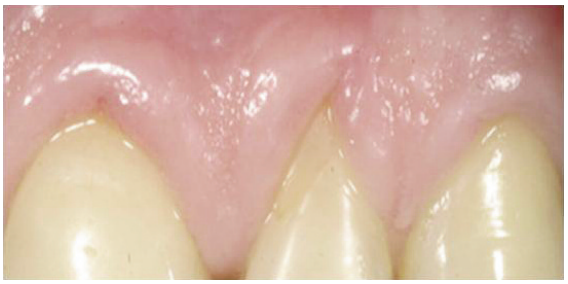

Figure 1. Stillman's cleft.

viral infections may induce the formation of a buccal probing pocket of sufficient depth to reach the periapical areas of the tooth. Sometimes a delayed diagnosis is made only when an endodontic abscess occurs (10). The prognosis of the clefts is variable: they can heal uneventfully or remain as superficial lesions combined with deep periodontal pockets.
In 2013 Pilloni showed how a laterally moved, coronally advanced technique could modify and eliminate this kind of anatomical lesion. He demonstrated that such surgical approach was effective in treating an isolated Stillman's cleft and the result remained stable over a 5-year period (11).
Analysis of gingival clefts indicate an apically-directed spread of an inflammatory exudate through the gingival connective tissues, with concurrent epithelial resorptive and proliferative reactions, with collagen resorption being mediated by an hydrolytic enzymatic activity (8).
The aim of this study was to examine the Stillman's cleft histological features in two different patients and compare them with the clinical aspects (healthy *vs* non healthy periodontal tissues).

Case report

Two patients with in common the presence of an asymptomatic Stillman's cleft on a vital and stable element, without any restoration, were selected for the study. They presented with two different periodontal conditions: patient A was periodontally healthy, meanwhile patient B was healthy but previously treated for mild periodontitis. Patient B showed a deeper lesion (5 mm) than patient A (2 mm).

The surgical protocol consisted of the excision of the cleft, as indicated by previously validated techniques (12), in order to create a better manageable contour of the gingival recession for subsequent treatment. Preparation of the surgical site followed the same surgical protocol of the treatment of a single gingival recession with a subepithelial connective tissue graft, that allows the coverage of the exposed root surface (11). Patients received ibuprofen twice daily for three days and a 0.12% chlorhexidine rinse every 12 hours for 7 days. No systemic antibiotics were used.
The tissue samples were fixed in 10% neutral buffered formalin for 24 hours and than were oriented in order to correctly identify the cleft and sectioned perpendicularly longitudinally by 2 mm cuts. The biopsies were sampled *in toto* in two histological biocasettes: the representative sample of cleft was placed within the first one (one or two samples) and the lateral part of surgical biopsies into the second one. Finally, they were embedded in paraffin wax and 4 μm serial sections were cut at different levels and stained with haematoxylin and eosin for each block. The slides were examined with a Nikon Eclipse E1200 light microscope and pictures taken with a Nikon camera system.
Patient A: at scanning magnification, a lichenoid band-like inflammatory infiltrate is observed with focal epithelial ulceration corresponding to cleft floor.
At higher magnification, the epithelium shows reactive atypia with many mitoses, spongiosis, acantosis and occasional diskeratotic cells.
The inflammatory infiltrate is mainly constituted by small lymphocytes with sligthly irregular nuclei and only scarce plasma cells. The lamina propria shows fragmentation of elastic fibers (Fig. 2).

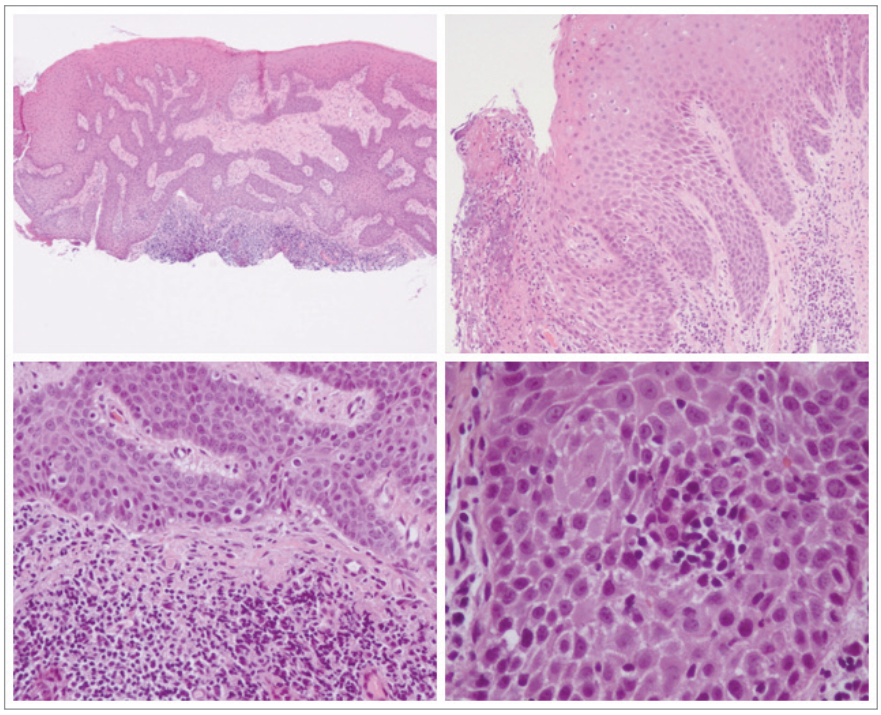

Figure 2. Histological analysis patient A.