Gingival Stillman's Cleft-Revisited

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Abstract:

Stillman's clefts are apostrophe shaped indentations extending from and into the gingival margin for varying distances. The etiology of this cleft is still not clear. They may repair spontaneously or persist as surface lesions of deep periodontal pockets that penetrate into the supporting tissues. Here we report a case of stillman's cleft in the mandibular left lateral incisor region treated with de-epithelialisation.

Keywords: Stillman's cleft, inflammatory, occlusal trauma, developmental, gingival clefts, simple clefts.

Introduction

Stillman's cleft is a term used to describe a specific type gingival recession consisting of a narrow triangular-shaped gingival recession. As the recession progresses apically, the cleft becomes broader, exposing the cementum of the root surface. When the lesion reaches the mucogingival junction, the apical border of oral mucosa is usually inflamed because of the difficulty in maintaining adequate plaque control at this site.¹

Stillman's cleft has been a forgotten entity and references reporting it had considered it being a result of occlusal trauma² but however the present consensus consider it being associated with inflammatory changes of the marginal gingiva due to accumulation of local factors.3 This article documents the case of Stillman's cleft and discusses the causative factors and treatment of Stillman's cleft

Case Report

A 23 years old male patient reported to outpatient department, department of Periodontology, Subharti Dental College and Hospital, Meerut, Uttar Pradesh with chief complaint of swollen gums in lower left front tooth region since 1 month. Medical history and past dental history were non-contributory. Patient was systemically healthy and was not taking any medications. Intra-oral examination revealed poor oral hygiene with no occlusal

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trauma. Stillman's cleft was seen in relation to mandibular left lateral incisor on the labial aspect extending from marginal gingiva towards muco-gingival junction. Radiographic examination revealed no evidence of bone loss #32. Scaling and root planing was performed and during re-evaluation of Phase Stillman's cleft I, persisted. Gingival de-epithelialisation was performed with gingivoplasty with conventional technique under local anaesthesia. Healing was satisfactory.(Fig.1,Fig.2,Fig.3)



Fig.1: Pre-operative photograph showing Stillman's cleft #32

Fig.2: De-epithelialisation and gingivoplasty done #32.





Fig.3: Clinical photograph showing complete healing

Discussion

Stillman's clefts are apostrophe shaped indentations extending from and into the gingival margin for varying distances. The clefts generally occur on the facial surface. One or two may be present in relation to a single tooth. The margins of the clefts are rolled underneath the linear gap in the gingiva and the remainder of the gingival margin is blunt instead of knife edge. Originally described by Stillman and considered to be the result of occlusal trauma, these clefts were subsequently described by Box as pathologic pockets in which the ulcerative process had extended through to the facial surface of the gingiva. The clefts may repair spontaneously or persist as surface lesions of deep periodontal pockets that penetrate into the supporting tissues. Their association with trauma from occlusion has not been substantiated. The clefts are divided into simple, cleavage in a single direction which is most common and compound cleft where cleavage in more than one direction.4 The length of the clefts varies from a slight break in the gingival margin to a depth of 5 to 6 mm or The putative cause of a Stillman's cleft was occlusal trauma, and the recommended treatment was occlusal adjustment. Several textbooks also stated that occlusal forces might be a factor in gingival recession.⁴ Energiee determined that tooth position and anatomy of the supporting bone were a major factor in the occurance of recession and that the literature did not support occlusal forces as a factor in the etiology of gingival recession. However, he indicated that occlusal force might be a factor in some instances and should be evaluated as possible factor in recession.⁵

Another study by Prasad et al⁶ suggested that occlusal interferences in maximum intercuspation and eccentric movements in one form or the other and absence of mutually protected occlusion could contribute to gingival lesions such as gingival recession and clefts.

A positive relationship between occlusal trauma and gingival recession was shown by Kleber and Schenk. Their study revealed that more than 70% showed functional disturbances to be present on the teeth with gingival recession. However, Gormon evaluated gingival recession in 164 patients and related to the presence of recession to various clinical factors, including occlusal trauma. But occlusal trauma was not found to be a significant cause in gingival recession.⁸

In another interesting study by Klaiber et al employing instrumental functional analysis showed that no

conclusive relationship exist between traumatizing occlusion and appearance of Stillman clefts.⁹

It is claimed that TFO (trauma from occlusion) may lead to thickening of the free gingival margin (Mccall's Festoon), gingival dehiscence (Stillman's cleft) or gingival recession have not been supported by research findings. Such concepts seem to lack scientific evidence based on current knowledge of periodontal physiology and pathology. It has been suggested that clefts are usually the result of calculus or faulty tooth brushing. 10 Interdental clefts or invaginations contribute to orthodontic relapse and poor periodontal health in cases. Treatment extraction options include de-epithelialization, gingivectomy, gingivoplasty by conventional or using electrosurgical unit if the cleft is shallow and narrow.¹⁰ Six weeks after phase I therapy, cleft areas could be surgically treated with either a lateral pedicle graft or a connective tissue graft for root coverage.

Conclusion:

Clefts may cause hindrance in oral hygiene maintenance, and if left untreated leads to further progression of periodontal diseases. Gingival clefts or Stillman's clefts are less discussed in the literature. Studies to establish the etio-pathogenesis are needed.

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