CASE REPORT

Management of Drug Induced Gingival Enlargement: A Case Report

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ABSTRACT

This article reviews the etiology, pathogenesis and AED associated with gingival overgrowth, in addition to its multidisciplinary management and prevention. Although the pharmacologic effect of each of these drugs is different and directed toward various primary target tissues, all of them seem to act similarly on a secondary target tissue, i.e., the gingival connective tissue, causing common clinical and histopathological findings.

INTRODUCTION

“Gingival enlargement” is the term now used to describe medication-related gingival overgrowth or gingival hyperplasia and can be defined as an abnormal growth of the periodontal tissue. The term “gingival hyperplasia” is an inappropriate term because enlargement is not the result of an increase in the number of cells, but rather an increase in extracellular tissue volume. Gingival enlargement produces aesthetic changes and clinical symptoms including pain, tenderness, bleeding, speech disturbances, abnormal tooth movement, dental occlusion problems, enhancement of caries development and periodontal disorders. It may be caused by medications, including antiepileptic drugs (AED) genetic abnormalities, such as hereditary gingival fibromatosis, proliferative lesions, etc.*

CASE REPORT

A 16-year-old male reported to the Department of Periodontology with swollen and bleeding gums. Past medical history revealed that patient was known case of epilepsy and was under medication for same (100 mg capsule TID). Preoperative intra oral view reveals appearance of generalised gingival enlargement. Gingival enlargement is bulbous, diffuse and gradually progressed to present stage. As per Bokenkamp’s grading, maxillary anterior region showed grade II enlargement (involving papilla and marginal gingiva) (Fig. 1) while mandibular anterior showed grade III enlargement (covering three quarters or more of the crown) (Fig. 2). Enlargement is not so significant in lingual and palatal region (Fig. 3).

Treatment

Scaling and root planning was performed. Drug substitution was carried as per physician’s consent. External bevel gingivectomy was carried in maxillary and internal bevel gingivectomy was carried in mandibular anterior region (Figs. 5a–b). Bleeding points were marked with Krane Kaplans pocket marker. External bevel gingivectomy was carried from 2 to 3 mm from gingival margin by extending incision apical to bleeding points to eliminate the pocket lining (Fig. 4). The incised tissue was excised, tissue tags were removed and debridement was done. As this created an open gingivectomy wound that heals by secondary intension periodontal pack was applied (Fig. 6). The patient was given postoperative instructions. In lower anterior segment internal bevel gingivectomy was carried by ledge and wedge technique (Fig. 7). Flap was reflected and debridement was done (Fig. 8).

Flap was approximated, interrupted sutures were placed (Fig. 9) and periodontal pack was applied.

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Figure 1  Gingival enlargement in maxillary anterior shows Bokenkamp’s Grade II.

Figure 2  No significant enlargement seen in maxillary palatal region.

Figure 3  Mandibular anterior shows Boken Kamp’s Grade III involvement.

Figure 4  External bevel gingivectomy performed in maxillary anterior region.

Figure 5 (a–b)  External bevel gingivectomy performed in maxillary anterior.

Histological Features

The excised tissue was sent to department of oral pathology for histological examination. Histological picture shows stratified squamous epithelium, often acanthotic with thin long rete pegs extending deep in to the lamina propria. Lamina propria shows proliferation of fibroblasts and increased collagen formation and an increase in non-collagenous proteins (Fig. 10).

Granulation tissue composed of numerous young capillaries and fibroblasts and irregularly arranged collagen fibrils with occasional lymphocytes. After careful case follow-up phenytoin was substituted by lamotrigine. As the recurrence of gingival enlargement is very common oral hygiene practice reinforcement was stressed to care takers of the patient during regular follow-up visit.
Management of drug induced gingival enlargement

DISCUSSION

Phenytoin (Dilantin, Pfizer, New York, USA) commonly prescribed for control of seizures in cerebral palsy, was the first drug reported to produce gingival enlargement with the incidence ranging from 3% to 85%. The facial surfaces of the gingiva in anterior sextants are often most severely involved, and the patient may present with inflamed, fibrotic masses spreading from the interdental papilla to the attached gingiva that may interfere with mastication, speech, and esthetics. Phenytoin induced gingival overgrowth develops as a result of an increase in the connective tissue extracellular matrix.

Earliest signs of gingival change, soreness and tenderness start occurring 2–3 weeks after therapy. During the 6–9 months there is enlargement of interdental papillae, which starts as a painless, beadlike enlargement of the interdental papilla. As the condition progresses, the marginal and papillary enlargements unite and results in massive tissue fold covering most part of the crowns. When uncomplicated by inflammation, the lesion is mulberry shaped, firm, pale pink, and resilient, with a minutely lobulated surface and no tendency to bleed. Facial gingiva of the anterior sextant is more commonly affected and the colour is coral pink to deep bluish red with a granular or lobulated surface. Tissue culture experiments indicate that phenytoin stimulates proliferation of fibroblasts like cells and epithelium. These phenytoin induced fibroblasts show increased synthesis of sulfated glycosaminoglycans. Also phenytoin may induce a decrease in collagen degradation as a result of production of inactive fibroblastic collagenase.

The usual therapeutic plasma level of phenytoin necessary to maintain effective seizure control is 10–20 μg/ml, which exceeds the minimal threshold dose below
which gingival overgrowth does not occur. Daily dosage consist of 300 mg and giving a plasma concentration of 10 ng/ml. Prevalence of gingival enlargement with phenytoin (Dilantin®) use has been shown to be up to 50%, while other anticonvulsants such as Valproic acid, Phenobarbital® and Tegretol® have been shown to be rarely associated with the disorder.

Regular use of CHX mouthwash helps to reduce the recurrence rate after surgery (O’Neil & Figures 1982). However, the bacterial resistance and taste disturbance limits its long-term use.

Systemic antibiotics short courses of azithromycin and metronidazole have been evaluated in the management of DIGO in organ transplant patients. However, the results are conflicting. Azithromycin (3–5 days, 250–500 mg/day) appears to be more effective than Metronidazole in the management of gingival enlargement (Chand et al. 2004). Two possible mechanisms by which Azithromycin may act: first, by reducing concomitant bacterial infection and hence inflammation (Mesa et al. 2003). Second, by increasing the phagocytic activity of gingival fibroblasts, thereby reversing the ability of cyclosporin to decrease collagen degradation (Paik et al. 2004). Recurrence may occur as early as 3–6 months, but in general results are maintained for at least 12 months. To prevent recurrence, patient is advised to follow a meticulous home care regimen (Nishikawa et al. 1991).

CONCLUSION

Use of medications with the potential to contribute to the development of gingival overgrowth will likely increase in the years to come. Its clinical appearance is similar in most cases and the comprehensive management may be challenging and multidisciplinary in nature.

Treatment is generally targeted on drug substitution and effective control of local inflammatory factors such as plaque and calculus. On failure of these measures to cause resolution of the enlargement, surgical intervention is recommended. These treatment modalities, although effective, do not necessarily prevent recurrence of the lesions. Newer molecular approaches are needed to clearly establish the pathogenesis of gingival overgrowth and to provide novel information for the design of future preventive and therapeutic modalities.

REFERENCES


Statement of originality of work: The manuscript has been read and approved by all the authors, the requirements for authorship have been met, and that each author believes that the manuscript represents honest and original work.

Source of funding: None

Conflict of interest: None

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