Surgical Management of Amlodipine Induced Gingival Enlargement - A Case Report

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ABSTRACT

Amlodipine, a calcium channel blocker, used with increasing frequency as antihypertensive and anti-angina drug has been found associated with gingival enlargement. It causes unesthetic appearance and formation of new niches for periodonto-pathogenic bacteria. If severe, it causes difficulty in mastication, speech and oral hygiene maintenance. Its treatment is still largely limited to meticulous oral hygiene maintenance, drug substitution, professional scaling and root planning and surgical excision of enlarged gingival tissue. There are very few reports of amlodipine-induced gingival enlargement at a dose of 5 mg. This case report discusses amlodipine-induced gingival enlargement and its management.

Keywords: Amlodipine, gingival enlargement, surgical excision.

INTRODUCTION

An increase in size of gingiva is called as gingival enlargement or gingival overgrowth. 1 One of the common cause for gingival enlargement are drugs which was first reported in 1939 by Kimball with chronic usage of phenytoin. 2 Amlodipine, a commonly used calcium channel blocker, is reported to cause gingival enlargement with an incidence rate of 3.3%. 3 In Nepal, recent study reported prevalence rate of 2.5%. 4 Amlodipine reduces $\alpha 2\beta 1$ integrin mediated phagocytosis of collagen thereby increasing collagen content. 5 Its management may be nonsurgical or surgical (excision). Here, we report a case of Amlodipine induced severe gingival enlargement which was managed surgically.

CASE REPORT

A 68 years old female presented to dental out patient department with the chief complaint of swollen gums since three years. It was associated with discomfort during speech and mastication, and bleeding on brushing. She

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also complained of multiple loose teeth. She first noticed swelling of gums in lower front teeth three years back which progressively increased in size and also appeared in posterior areas, covering almost entire tooth surface at multiple sites.

The patient was diagnosed with hypertension three years back and was initially under Amlodipine (5 mg) once daily. After three-four months of its use, she noticed swelling of gums which was progressive. She had swollen hands and feet as well. Her cardiologist substituted the medication with losartan (50 mg). She was unable to make follow-up visits to the dentist. She soon had hemiplegia of left side of body and took almost two-three months to recover from it. The gingival enlargement persisted and progressed to the present size.

Intraoral examination revealed generalised enlargement of the gingiva of both the upper and the lower jaws involving marginal gingiva, attached gingiva and the interdental papilla (Figure 1). Both buccal and lingual/palatal gingiva was involved. The enlarged gingiva was erythematous, had lobulated surface and showed bleeding on probing. The enlarged gingiva covered more than three-quarters of the tooth surface that is, Grade III enlargement as per Bokenkamp and Bohnhorst (1994). Moderate plaque and calculus deposits were seen. Few teeth (26,35 and 46) had grade III mobility.

Panoramic radiography revealed generalized horizontal bone loss and furcation involvement with respect to 46 (Figure 2). Non-surgical periodontal therapy was performed after which slight reduction in gingival enlargement was noticed.

Since the enlargement was severe, unesthetic and caused problem during function, it was planned for quadrant-wise







Figure 1a: Generalized diffuse gingival enlargement.

Figure 1b: Upper arch.

Figure 1c: Lower arch.



Figure 2: Generalized bone loss.



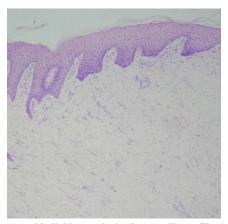
Figure 3a: Undisplaced flap surgery.



Figure 3b: Suture placed after flap surgery.

excision. Undisplaced flap surgery was started from the third quadrant and second premolar which was extremely mobile was extracted simultaneously (Figure 3a). The flap edges were approximated and sutured (Figure 3b). A part of excised gingiva was sent for histopathological evaluation which revealed elongated rete ridges, underlying connective tissue with dense collagen fibres and chronic inflammatory cell infiltrate chiefly lymphocytes and plasma cells (Figure 4).

Similar procedures were carried out in first, second and fourth quadrants (Figure 5, 6, 7 respectively) and extractions were done with respect to 26 and 46 during the procedure. Sutures (3-0 silk) were removed in one week and patient was followed up in one month, three months and six months, with oral hygiene reinforcement and instructions in each visit. Marked improvement in gingival and periodontal health was achieved with no recurrence over six months (Figure 8).



Figure~4: Gingival~biopsy~examination~shows~epithelial~hyperplasia, dense~collagen~fibres~and~chronic~inflammatory~cell~infiltrate.



Figure 5a: First quadrant presurgical.



Figure 5b: After debridement.



Figure 5c: Suture placed after flap surgery.



Figure 6a: Second quadrant presurgical.



Figure 6b: After debridement and extraction of 2.



Figure 6c: Suture placed after flap surgery.



Figure 7a: Fourth quadrant pre-operative. Figure 7b: After debridement and extraction of 46.





Figure 7c: Suture placed after flap surgery.



Figure 8a: At 6 month follow up.



Figure 8b: Occlusal view (upper arch).



Figure 8c: Occlusal view (lower arch).

DISCUSSION

Amlodipine, a dihydropyridine calcium channel blocker, is commonly used now-a-days due to its longer duration of action and minimal cardiac depressant activity unlike nifedipine. However, the prevalence of gingival overgrowth associated with amlodipine is lower than that associated with other calcium channel blocking agents including nifedipine.3 Also, there are less data on reports of hyperplasia with amlodipine at a dose of 5 mg even when used for more than six months.3,6 The present case occurred with a low dose of amlodipine (5 mg) after duration of only three- four months.

Drug induced gingival enlargement is multifactorial and involves interaction of several factors (age, sex, genetic predisposition, pharmacokinetic variables, periodontal variables, concomitant medication) which influences the interaction between drug and metabolite with gingival fibroblasts.7 Calcium channel blockers inhibit the intracellular Ca2+ uptake thereby stimulating gingival fibroblasts proliferation, reducing collagen phagocytosis by fibroblast and inactive collagenase production favouring collagen deposition.

All patients receiving the same drug may not develop

gingival enlargement as their susceptibility may be governed by existence of differential proportions of fibroblast subset (high activity: low activity fibroblasts) which is genetically determined.⁸

Treatment includes non-surgical periodontal therapy for reducing inflammatory component and thereby avoiding the need of surgery. Drug substitution, if possible, can be done with the patient's physician consent. If any drug substitution is attempted, it is important to allow for 6–12 months to elapse between discontinuation of the offending drug and the possible resolution of gingival enlargement before a decision to implement surgical treatment is made. However, if functional, aesthetic and maintenance complications persist then surgery is needed.⁹

If gingival enlargement still persists after non-surgical therapy including drug substitution, surgery is done, either by gingivectomy or flap surgery. In present case patient was subjected to drug substitution $2\frac{1}{2}$ years back after which patient lost follow-up but the enlargement persisted and increased in size. Non-surgical periodontal therapy was done and since the patient had esthetic and functional problems, surgery was planned. Undisplaced flap surgery was done as there was presence of pockets, and bone loss. There was complete resolution of the enlargement which was maintained even at six months follow-up.

Conflict of Interest: None.

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