Management of amlodipine influenced gingival overgrowth

Surgical Management of Amlodipine influenced gingival overgrowth in Hypertensive patient.

Abstract:

Drug-influenced gingival overgrowth (DIGO) is a serious concern both for the patient and the clinician. A number of local and systemic factors such as plaque, hormonal changes, drug ingestion, heredity can cause or influence gingival overgrowth. Certain anticonvulsants, immuno-suppressive drugs and a number of calcium channel blockers have been shown to produce similar gingival overgrowths in certain susceptible patients. Amlodipine is a comparatively new calcium channel blocker may induce gingival overgrowth in case of underlying inflammatory component. A 38-year-old hypertensive female patient on amlodipine (10 mg/day, single dose orally) since eight months, sought dental attention because of the resultant gingival overgrowth. Clinical examination, Medical history and histological assessment further helped to formulate a diagnosis of DIGO. Six weeks after phase-I therapy and drug substitution, undisplaced flap surgery was performed. The patient’s gingiva seemed to be normal at six month follow-up visit, with no signs of recurrence.

Key words: Gingival overgrowth, Hypertension, Amlodipine, Undisplaced flap surgery. Drug influenced gingival overgrowth.

Introduction:

There are many factors (causal or modifying) involved in gingival overgrowth. Plaque accumulation on teeth causes gingival inflammation and

may lead to inflammatory enlargement. Gingival overgrowth can be seen in patients with familial hereditary gingival fibromatosis, pregnancy, and leukemia. DIGO is a well-documented side effect of some pharmacologic agents, including, but not limited to, calcium channel blockers (CCBs), phenytoin, and cyclosporine[1, 2]. It can be a serious concern for patients due to the concomitant unesthetic appearance and the formation of new niches for the periopathogenic bacteria [3]. Despite the relatively high prevalence of nifedipine-influenced gingival overgrowth, [4] amlodipine has less frequently been reported as the potential etiologic cause of gingival overgrowth[5] . Amlodipine is a comparatively new long acting dihydropyridine calcium channel blocker that is used in the management of both hypertension and angina. Unwanted effects associated with chronic usage of

amlodipine are few and are mainly related to vasodilation. The pharmacological effects of these drugs are specific but the clinical and histological features of the enlargement caused by the different drugs are similar. The clinical appearance of DIGO is usually characteristic, although variants are seen depending on the location of lesions, the irritants involved and the extent of inflammation. As the condition progresses, the marginal and papillary gingival overgrowth and may interfere with speech, mastication and aesthetics. In the patients with preexisting periodontitis and DIGO the deepening of periodontal pockets and associated subgingival microbiota may increase periodontal

attachment and bone loss. The surgical treatment is a definitive therapy for DIGO, in absence of spontaneous regression following drug substitution and

phase-I Therapy. The common surgical technique is the simple excision of the excessive gingival tissue with– external bevel gingivectomy (EBG) or internal (reverse) bevel gingivectomy (IBG). The surgical approach of undisplaced full thickness flap, in this context, is more suitable to eliminate periodontal pockets (Pocket wall) in presence of adequate attached gingiva and to improve the alveolar bone morphology. In the present report, a case of amlodipine-influenced gingival overgrowth (AIGO) has been presented wherein the AIGO was treated in the following phases: (1) substitution of the drug , (2) thorough Phase-1 therapy, (3) surgical excision of the residual gingival overgrowth and (4) maintenance and supportive therapy. Case Description:

A 38-year-old female patient was referred to us with complaint of swollen and bleeding gums in the upper and lower jaw. Past medical history revealed hypertension for which the patient received amlodipine (10 mg/day, single dose orally) for the last eight months. The patient had noted a gradual and painless enlargement of the gingiva for first 4 months and then she noticed bleeding gums. A generalized fibrous gingival enlargement with edematous marginal gingiva, owing to superimposed inflammatory component, was found throughout the maxillary and mandibular gingiva (Fig. 1A, B, C, D). Presence of generalized periodontal pockets (≥7-8mm) and clinical attachment loss (≥5-6mm) was a prominent feature of gingival overgrowth indicating a vertical enlargement of gingiva. Purulent discharge and bleeding on probing were detected which were in accordance with the inflammation.

Treatment:

On request, patient’s physician substituted amlodipine with Beta Adrenergic blocker (Atenolol), after which, patient was recalled for through scaling and root planing. Oral hygiene instructions, chlorhexidine mouthwash 0. 2% of 10ml twice a day was prescribed. At follow-up after six weeks, residual inflammatory component of the enlargement resolved(Fig-2) but the gingival overgrowth needed definitive surgical treatment. Under adequate local anesthesia (xylocaine 2%), the pocket depth was marked, (Fig-3) an internal bevel incision was taken up to the alveolar crest. (Fig-4) Crevicular and interdental incision along the base of the pocket wall was released and full thickness mucoperiosteal flap was reflected. (Fig-5) The excised mass was stored in formalin for further histopathologic investigation. Scaling, root planning and curettage were completed. Osseous resective surgery, using carbide burs, along with copious saline irrigation was done to recontour thickened bony plates, ledges and deep interdental craters. (Fig-6) Flaps were trimmed and approximated using interrupted silk sutures. Routine post surgical instructions, a course of antibiotics and analgesics (Cap. Amoxycillin 500mg three times a day for five days and Ibufrofen 400 mg three times a day for three days) and 0. 2% chlorhexidine was prescribed twice a day for fifteen days. Microscopic inspection of the gingival biopsy specimens demonstrated a connective tissue hyperplasia, acanthosis of overlying epithelium and elongated rete ridges together with inflammatory cells. Sutures were removed after 1 week. Healing was uneventful and the patient’s appearance and overall function improved considerably at six month follow up. (Fig-7) Oral hygiene instructions were given from first visit and reinforced in all subsequent visits.

Discussion:

Amlodipine is a second-generation dihydropyridine CCB that can cause gingival overgrowth. The prevalence of amlodipine-influenced gingival overgrowth has been shown to be between 1. 7% and 3. 3%[6, 7]. Lafzi *et al.* (2006) had reported rapidly developing gingival hyperplasia in patient receiving 10 mg/day of amlodipine within 2 month of onset. [8] The incidence of gingival overgrowth with nifedipine therapy has been reported to be as high as 20%, [9] and a study by Prisant (2002) [10] reported that the prevalence with the use of CCBs might be as high as 38%. Gingival overgrowth considered to be 3. 3 times more common in men than in women [10] . The most common form is bacterial plaque–influenced gingival disease, which presents as gingivitis. Use of phenytoin, cyclosporine, and CCBs, as well as vitamin C deficiency, can also predispose to development of gingival overgrowth, as can hormonal shifts during pregnancy. The reason for these adverse events is not absolutely known, but mechanisms involving inflammatory and non inflammatory pathways have been suggested [11]. For example, individual sensitivity to a drug’s metabolic pathway might be a trigger [11]. Untreated gingival overgrowth might lead to bleeding, infection, abscess, ulceration, cosmetic deficiency and/or functional difficulty (eg, chewing, talking) [10]. Treatment of drug-influenced gingival overgrowth includes cessation/replacement of the drug and decreasing other risk factors with meticulous mechanical and chemical plaque control. Replacing the affecting drug with another agent is also recommended when possible[12]. In present case of DIGO patient was under treatment for hypertension since last 8 months and was prescribed tablet Amlodipin 10mg/day by her

physician. Thorough SRP and replacing the Amlodipin with Atenolol was done. Drug substitution and thorough SRP did not result into regression of the enlargement.

The surgical treatment is a definitive therapy for DIGO, in absence of spontaneous regression following drug substitution and phase-I Therapy. Classic gingival surgery primarily deals with the treatment of pockets – i. e., gingival sulci that are deepened due to a proliferation or an increase in bulk of gingival tissue in a coronal direction, with or without apical migration of the epithelial attachment. External bevel gingivectomy (EBG) and internal bevel gingivectomy (IBG) should be reserved for cases not responding to non surgical methods or severe cases that affect oral hygiene or functionality, or can be performed for cosmetic reasons. IBG approach has the benefit of limiting the large denuded connective tissue wound that results from the external gingivectomy, thereby minimizing postoperative pain and bleeding. It is accepted that gingival surgery (both EBG and IBG) is essentially limited to the treatment of pseudopockets. But if true pockets associated with bone defects are present then undisplaced flap surgery can be the treatment modality for the massive enlargement. The advantages of this technique are removal of pocket wall and osseous contouring simultaneously eliminating the gingival overgrowth and pocket in presence of adequate attached gingiva. In this case report undispalced flap surgery was performed for eliminating pocket and osseous contouring in presence of adequate attached gingiva. However regardless of the treatment option employed, regular maintenance and recall follow up are mandatory to achieve the long term success.

Conclusion

Gingival overgrowth is an overlooked but potentially harmful side effect of treatment with amlodipine and other calcium channel blockers and every physician should be aware of this, particularly if adverse oral symptoms arise during drug use. The amlodipine influenced gingival overgrowth in this case completely resolved when the patient was switched to Beta Adrenergic blocker (Atenolol) followed by surgical excision of the overgrowth. Another factor contributing to the excellent response to the therapy is the patient compliance in maintaining the oral hygiene. Lastly the patients' documented data should be shared with the physician to gain his confidence and respect for the dental community. In addition, he will be motivated to refer patients with complains of gum swelling at a much earlier stage or in fact, advice dental consultation for improvement of oral hygiene before prescribing the list of drugs that may influence gingival overgrowth in presence of preexisting gingival inflammation.

References

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