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AMLODIPINE INDUCED GINGIVAL ENLARGEMENT: A CASE REPORT

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ABSTRACT

Enlargement of the gingiva can be due to interactions between host and the environment. Drug-induced gingival enlargement is a serious concern both for the clinician and the patient. It is found that an increasing number of medications associated with gingival enlargement, which is used to treat common medical conditions. This case reports highlights the gingival overgrowth induced by Amlodipine and its management.

KEYWORDS: Amlodipine, Gingival enlargement, Overgrowth.

INTRODUCTION

Nowadays, the drug induced gingival lesions are called “gingival enlargement” or “gingival overgrowth”, as the earlier terms like “gingival hyperplasia” or “gingival hypertrophy” did not accurately reflect the histologic composition of the pharmacologically modified gingiva. Several medications can cause gingival enlargement as a side effect in susceptible individuals. Mainly three types of medications are associated with the gingival enlargement; calcium channel blockers like nifedipinne for treatment of hypertension or angina pectoris, anticonvulsants such as phenytoin for the control of seizures and immunosuppressant like cyclosporine A for the prevention of graft rejection. [1,2]

Amlodipine is a long-acting calcium channel blocker belonging to third generation dihydropyridine group, which is used commonly for the treatment of hypertension and angina. Pharmacokinetic profile characteristic of these group medications is an increased oral bioavailability and extended clearance time.[3] In the present report, a case of amlodipine‐induced gingival overgrowth is discussed.

CASE REPORT

A 45-year-old male patient came to the hospital with the complaint of swellings of gums since last one month period (Fig 1 to 3). On taking history of that patient, it was found that patient was hypertensive since 4 to 5 years and there was change in his medications to amlodipine by his physician since last one and half months. Since the gingival enlargement started since the time of taking amlodipine, it was assumed to be due to amlodipine. Also other etiological factors were considered. The current medications were ceased and the patient was prescribed angiotensin receptor blockers, with the consent from his physician. During follow up, a remarkable improvement of the patient's gingival enlargement was observed.

Gingivectomy was done for few pieces of remained enlarged tissue from the labial part of the teeth 32, 33 and the palatal parts of tooth 23 were sent for histopathological examination (Fig. 4). Patient was prescribed analgesics, antibiotics for three days and mouthwash Chlorhexidine Gluconate 0.12% for two weeks after surgical procedure. The histopathological examination demonstrated proliferated stratified squamous epithelium with elongated rete ridges and irregular fibrous overgrowth composed of collagenous connective tissues with a diffuse chronic inflammatory cell infiltrate (Fig. 5).

Fig.1: Mandibular anterior region showing gingival enlargement on labial side.

Fig. 2: Occlusal view of mandibular anterior region showing gingival enlargement on labial and lingual side.

Fig. 3: Occlusal view of maxillary teeth, showing gingival enlargement on both buccal and palatal aspect.

Fig. 4: Biopsy specimen sent for the histopathological examination.

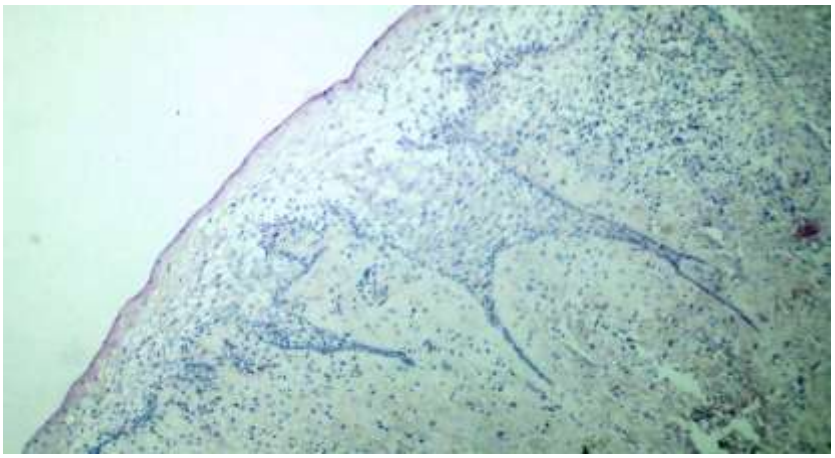

Fig. 5: Histopathological picture. (H & E, X 100, original magnification)

DISCUSSION

Medication induced gingival enlargement was first reported in the early 1980s associated with the nifedipine and was soon also described with diltiazem, verapamil, and in rare cases with amlodipine and felodipine. Amlodipine induced gingival enlargement was first reported in 1993. The clinical features of gingival enlargement usually presented as enlarged interdental papillae and resulting in a lobulated or nodular morphology. [2-4]

Till date, the exact pathogenesis of drug-induced gingival enlargement is not known, but some well-known risk factors are gingival inflammation resulted from poor oral hygiene, presence of dental plaques, the dose and duration of the drug used. The underlying mechanism remains to be fully understood. However, two pathways i. e. inflammatory and non-inflammatory have been suggested. The non-inflammatory mechanisms include decreased uptake of folic acid leading to defective collagenase activity, blockage of aldosterone synthesis in adrenal cortex and consequent feedback increase in ACTH level, and upregulation of keratinocyte growth factor. Alternatively, inflammation may develop as a result of direct toxic effects of concentrated drug in crevicular gingival fluid and/or bacterial plaques. This inflammation could lead to the upregulation of several cytokine factors such as TGF-ß1. [3,5-7] Another important factor having an impact on the development of gingival enlargement is gender. It occurs three times more common in men than in women. [3]

Histologically, it shows epithelial proliferation with irregular rete ridges, slight to moderate hyperkeratosis, fibrosis of underlying connective tissue with fibroblastic proliferation, increase in the number of capillaries with slight chronic inflammation. [1,6]

The primary treatment of drug-induced gingival enlargement involves drug substitution and good oral care. By the start of these things, gingival enlargement often improves. Usually, surgical approach is not needed. In the situations requiring surgery, gingivectomy or periodontal flap is performed. [3,8-11]

CONCLUSION

It is important to identify and explore possible risk factors relating to both prevalence and severity of drug‑induced gingival enlargement. Newer molecular studies are needed to clearly establish the pathogenesis of gingival overgrowth, so as to provide necessary information for the design of future preventive and therapeutic modalities. Also, when prescribing amlodipine, the side effect of amlodipine should be considered and patients should be recommended to pay attention to oral hygiene.

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