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Case Study

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EXPLORING AMLODIPINE- INDUCED GINGIVAL ENLARGEMENT: A CASE REPORT

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ABSTRACT

Gingival overgrowth is a well-recognized adverse effect associated with phenytoin, cyclosporine, and calcium channel blockers. The exact pathogenesis of drug- induced gingival overgrowth remains unclear, and no single unifying hypothesis currently links these three commonly implicated drugs. Key factors influencing this model include age, genetic predisposition, pharmacokinetic variables, plaque-induced inflammatory and immunological responses, and activation of growth factors. A deeper understanding of the underlying mechanisms may pave the way for more effective strategies to manage and control this undesirable condition.

INTRODUCTION

Increase in size of the gingiva is a common feature of gingival disease.[1] **Gingival enlargement** or **gingival overgrowth** are currently preferred terms in the clinical description of this condition. These terms are used to describe the abnormal increase in the size of the gingiva (gums) without implying any specific underlying pathological mechanism, which makes them more accurate and neutral compared to older terms like "hypertrophic gingivitis" or "gingival hyperplasia."

Drug-induced gingival overgrowth was the first reported in 1939 by Kimball, associated with chronic usage of the anti-epileptic drug phenytoin.[2] This enlargement is a well- known consequence of the administration of some anticonvulsant, immunosuppressant, and calcium channel blockers and may create speech, mastication, tooth eruption, and aesthetic problems. Clinical and microscopic features of the enlargements caused by the different drugs are similar. The enlargement of gingiva is associated with more than 20 drugs used for various diseases.[3]

CASE REPORT

A 62-year female patient was reported in the outpatient department of Periodontology and oral Implantology with a chief complaint of enlarged gums in upper anterior teeth region from last 6 months. As the patient having hypertension, she was under treatment of essential hypertension for 2 years and was taking amlodipine 5mg (OD)+telmisartan 40 mg (OD).

No pain, blood discharge, was noticed. However, the patient had moderate plaque & halitosis present. The oval reddish, sessile overgrowth of the left side extending from mesial aspect of 23 to distal aspect of 24 with its extension till the upper second molar. The overgrowth of the right side was seen in edentulous canine region. Patient having hypertension and on medication for the same systemic disease. The gingival overgrowth started slowly as a small nodular, sessile growth which gradually increased in size in overall maxillary arch. Patient was having difficulty in speech and mastication.

Intraoral clinical examination shows an exophytic growth in right and left maxillary anterior region, which extends in posterior left region. (Fig 1) The phase 1 therapy of the patient was performed and recalled after seven days. Simultaneously she was sent for physicians opinion regarding drug dosage. And the alternative drug was prescribed by the physician.

The area was anesthetized using 2% lidocaine hydrochloride without epinephrine. Excision of right-side overgrowth was done with scalpel. (fig. 2) Patient recalled after 7 days for follow up & left side overgrowth was operated. Excision followed by extraction of posterior mobile teeth was done. (Fig 3) Suture were given. And the specimen was sent for histopathological analysis. (Fig 4) Patient was recalled after 7 days for reevaluation. (Fig 6) There was no recurrence after three months. (Fig 5)

Histopathologically, the given H & E-stained section shows stratified squamous epithelium. The underlying connective tissue shows inflammatory cell infiltrate. The connective tissue is fibro-cellular in nature. Some areas show calcification & blood vessels. Para-keratinized epithelium of variable thickness covers the connective tissue stroma and epithelial ridges may penetrate deep into the connective tissue, creating irregularly arranged collagen fibers. (Fig 7)

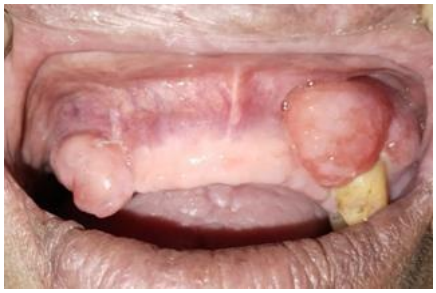


Figure 1: Pre-operative Intraoral View.

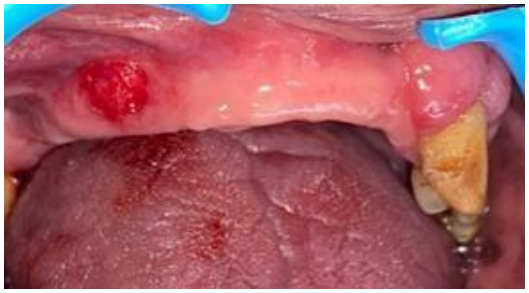


Figure 2: Excision of right-side overgrowth.

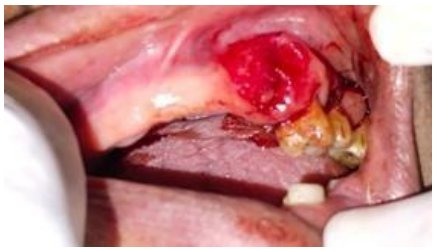


Figure 3: Excision of left side overgrowth followed by extraction of posterior teeth.

Figure 4: Biopsy Specimen.

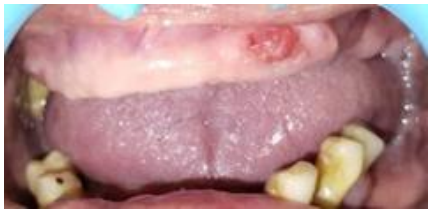


Figure 5: 7 days follow up.

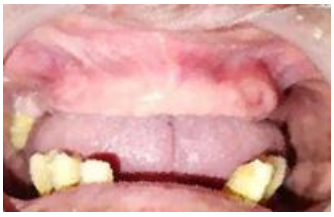


Figure 6: 3 months follow up.

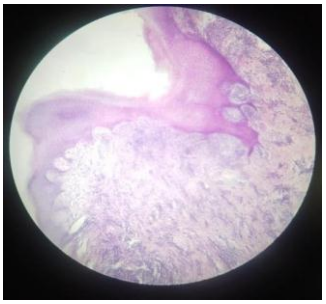


Figure 7: Histopathological Appearance.

DISCUSSION

The etiology of gingival enlargement is essential for effective management. As we mentioned, certain medications can contribute to this condition. Calcium channel blockers, particularly nifedipine, are well-documented causes, and amlodipine has been noted to potentially cause more severe cases of gingival overgrowth.

Other medications, like cyclosporine and phenytoin, also play significant roles in this condition. It's important for healthcare providers to review a patient's medication history when assessing gingival enlargement, as this can guide treatment decisions.

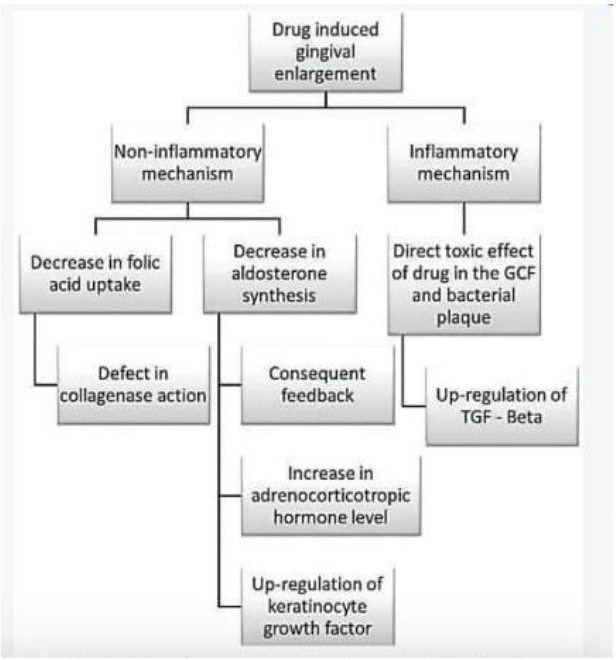
Additionally, addressing oral hygiene practices and considering alternative medications can help manage and mitigate the effects of these drug-induced enlargements.

Drug-induced gingival overgrowth appears to be more prevalent in children and adolescents and has a predilection for the anterior gingival tissues, Gingival changes can occur within 3 months of dosage.[7] The pattern of overgrowth development shows intra patient variation, but may reach a "state of equilibrium" often within the first year of commencing medication. Change in drug therapy, or systemic ill ness may alter this state and lead to further gingival changes.

By providing alternative drug for these conditions, gingival enlargement significantly reduces. Treatment for mild or moderate disease includes medication discontinuation or dose reduction in addition to plaque control and good oral dental hygiene. Scalpel gingivectomy remains the treatment of choice in severe cases and necessitates referral to a general dentist or periodontist. Metronidazole, azithromycin, and

azithromycin toothpaste have shown variable degrees of success in treatment. A recent study found that oral folic acid decreased the incidence of phenytoin-induced gingival overgrowth in children.[8]

The incidence in the general population is unknown.[1] The exact mechanism of action is not understood. Medications are thought to produce changes in fibroblast function increasing the extracellular matrix of the gingival connective tissue. Dental plaque causes inflammation and increases the likelihood of medication-induced gingival overgrowth. This process is not related to the systemic dose of the medication but to, its concentration in the saliva and gingival connective tissue. Clinically, it can lead to significant disfigurement and difficulty in chewing and speech.[9]



The non-inflammatory and the inflammatory mechanism for drug induced gingival enlargement

CONCLUSION

Drug-induced gingival overgrowth (DIGO) is a significant issue that can affect many individuals taking certain medications. It's one of the more common adverse effects on the periodontal tissues, especially among patients using medications like phenytoin (for epilepsy), calcium channel blockers (e.g., nifedipine), and immunosuppressive agents like cyclosporine. However, despite its prevalence, the precise pathogenesis behind the development and severity of DIGO remains unclear. Identifying potential risk factors that influence both the prevalence and severity could greatly enhance our understanding of this condition and help clinicians manage or mitigate its effects more effectively.

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