

Case Report**Drug Substitution and Non-Surgical Therapy in Phenytoin Induced Gingival Enlargement**

Roopa DA, Choudary S, Shukla P, Gopal S

ABSTRACT: Gingival enlargements can occur as a result of the administration of certain anticonvulsants, immunosuppressants, and calcium channel blockers. These enlargements are complicated by the presence of inflammation caused by plaque. Management of drug induced gingival enlargement includes consultation with patient's physician, drug substitution, non surgical or surgical therapy. This case report deals with the management of phenytoin induced gingival enlargement with non surgical therapy and drug substitution.

Keywords: Drug-induced; Gingival; Enlargement; Phenytoin; Non-surgical therapy; Mandibular.

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.¹ Gingival enlargement is associated with multiple factors including inflammatory (acute and chronic), idiopathic, drug-induced, neoplasia (benign and malignant tumors), hormonal disturbances, ascorbic acid (vitamin C) deficiency and with dental eruption.² There are three classes of drugs that are well-established causes of gingival enlargement, being responsible for most cases: antiepileptic drug (AED) phenytoin, antihypertensive calcium antagonists and immunosuppressant cyclosporine.³

Drug-induced gingival enlargement (DIGE) associated with chronic use of the AED phenytoin was first reported in 1939 by Kimball.⁴ DIGE may be complicated by the presence of local factors like plaque and the treatment consists of non surgical or surgical management of gingival enlargement along with replacement of the drug with another class of drug. We present here a case of phenytoin induced gingival enlargement which was managed by scaling and root planing and phenytoin was changed with carbamazepine.

CASE REPORT

A 24-year-old male patient reported to the outpatient department of Periodontics, Rama Dental College, with a chief complaint of generalized swollen gums, which bled on slight provocation (for the last 2 years).



Figure 1(a,b,c): Preoperative view showing pale pink to reddish gingiva that is soft and edematous with bulbous enlargement of the interdental papillae extending to marginal gingiva. The gingival enlargement is more in the maxillary and mandibular anterior region. Abundant plaque and calculus is also visible.

The patient's medical history revealed epilepsy since the age of 20, controlled with medication (phenytoin 100 mg BID) for the last 4 years. Gingival tissues were pale pink with reddish areas, enlarged, soft, and edematous with loss of stippling. Gingival enlargement had typical mulberry shape arising from interdental papillae and involving the marginal gingiva [Fig. 1(a,b,c)].



Figure 2 (a,b,c): Postoperative views of patient's gingiva after scaling and root planing and drug substitution. As compared to the preoperative appearance, gingiva is healed, enlargement has subsided, regaining the normal colour, contour and texture of gingiva.

It was confined to maxillary and mandibular anterior region. Enlargement was superimposed by inflammation because of presence of heavy calculus and plaque. Generalized bleeding on probing was present. Complete hemogram results were under normal limits. A diagnosis of generalized drug-induced gingival enlargement induced by phenytoin was made.

Before starting any periodontal treatment, the patient was referred to his physician for consultation regarding periodontal therapy and replacement of phenytoin. Phenytoin was replaced with carbamazepine (Tegretol 200mg BID) by the doctor and there was no contraindication for periodontal treatment. There was reduction in inflammation after initial oral prophylaxis and chlorhexidine mouth rinsing. Thorough scaling and root planing was repeating over a period of three months. There was reduction in gingival enlargement during 6 months follow up which was maintained at one year follow up too [Fig 2 (a,b,c)]. The patient was managed by non surgical periodontal therapy, not requiring gingivectomy. During this period patient reported no episodes of seizures.

DISCUSSION

An increasing number of medications are associated with gingival enlargement. Currently, more than 20 prescription medications are associated with gingival enlargement. 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.⁵

Gingival enlargement is one of the most frequent adverse effects associated with the administration of phenytoin. Incidence rates have ranged from 3 to 93%, but about 50% of patients on long-term phenytoin therapy develop gingival enlargement.⁶ Among the many suggested theories regarding the etiology of phenytoin induced gingival overgrowth (PIGO) are serum vitamin C deficiency, direct local effect of the drug or metabolite, depression of the pituitary -- adrenal axis, and immunological reaction. It has also been proposed that phenytoin induced folic acid deficiency could render the gingival tissues more susceptible to local factors (e.g., plaque), thus enhancing the response of the

gingiva to specific exaggerated inflammatory reactions as in gingival overgrowth.⁷

The growth starts as a painless, bead-like enlargement of the interdental papilla, and extends to the facial and lingual gingival margins. As the condition progresses, the marginal and papillary enlargements unite; they may then develop into a massive tissue fold covering a considerable portion of the crowns, and they may interfere with occlusion. When uncomplicated by inflammation, the lesion is mulberry shaped, firm, pale pink, and resilient, with a minutely lobulated surface and no bleeding tendency. The presence of the enlargement makes plaque control difficult, often resulting in a secondary inflammatory process that complicates the gingival overgrowth caused by the drug. The resultant enlargement thus becomes a combination of the increase in size caused by the drug and the complicating inflammation caused by bacteria. Secondary inflammatory changes not only add to the size of the lesion caused by the drug, but also produce a red or bluish red discoloration, obliterating the lobulated surface demarcations and increasing the tendency to bleed. The enlargement is usually generalized throughout the mouth but is more severe in the maxillary and mandibular anterior regions.⁸

Ideally, the treatment of choice for medically induced gingival overgrowth would be discontinuation of the associated medication. Nevertheless, this approach is often not possible. Given the significance of plaque and calculus as a risk factor for exacerbation of gingival overgrowth, initial periodontal therapy should be aimed at reducing the inflammatory component comprising comprehensive oral hygiene advice to ensure optimal home care along with regular professional debridement. Prophylaxis includes oral hygiene instructions with frequent and correct brushing of teeth, and use of floss and rinses. The use of a 0.2% chlorhexidine mouthwash has been shown to be a highly beneficial adjunctive regimen to mechanical oral hygiene methods. The administration of folic acid (topical and/or oral) could ameliorate gingival overgrowth in

some cases, but its specific role has not clearly been established.^{2,3}

Definitive treatment involves surgical elimination of the excess gingival tissue through implementation of either the gingivectomy procedure or periodontal flap approach. The clinician's decision to choose between these two surgical techniques should be made on an individual basis, encompassing careful consideration of the following aspects: the extent of area requiring surgery; the presence of periodontitis and osseous defects; the amount of keratinized gingiva, and the position of the base of the pockets in relation to the existing mucogingival junction; the use of carbon dioxide laser surgical therapy is also becoming more common in treatment of gingival overgrowth due to its advantages in postoperative haemostasis.¹ Relapse may occur 3-6 months after surgical treatment, but in general maintenance of surgical results for at least 12 months are reported.⁹

The present case had typical clinical findings of drug induced gingival enlargement caused by phenytoin. The drug replacement and scaling and root planing produced drastic reduction in the enlargement, requiring no surgical intervention. The most effective treatment of drug-related gingival enlargement is withdrawal or substitution of medication. Unfortunately, not all patients respond to this mode of treatment, especially those with longstanding gingival lesions.¹⁰ Substitution of phenytoin with a different anticonvulsant has long been suggested as the treatment of choice for the severely affected gingiva.

The feasibility of phenytoin substitution has increased with the addition of a new generation of anticonvulsants such as lomatrigine, gabapentin, sulthiame, and topiramate. For this patient his doctor substituted phenytoin with carbamazepine and drug substitution helped in improving the clinical findings. The patient maintained good oral hygiene and the results were sustained during six months and one year follow ups. This case report emphasizes that drug substitution and non surgical management can

be used for the treatment of phenytoin induced gingival enlargement.

CONCLUSION: Every case of drug induced gingival enlargement should be treated in a step-wise manner inclusive of due consultation with patient's physician, substitution of the drug, non-surgical therapy, and surgical therapy (if needed), followed by supportive periodontal therapy at 3-month intervals.

Author affiliations: 1. Dr. Dinnahalli Adinarayan Roopa, MDS, Professor and HOD, 2. Dr. Shweta Choudary, MDS, PG Student, 3. Dr. Prerna Shukla, PG Student, 4. Dr. Saumiya Gopal, MDS, Reader, Dept. of Periodontics, Rama Dental College and Research Centre, Kanpur, Uttar Pradesh

REFERENCES:

1. American Academy of Periodontology. Informational paper: drug associated gingival enlargement. *J Periodontol.* 2004; 75:1424-31.
2. Brunet L, Miranda J, Farré M, Berini L, Mendieta C. Gingival enlargement induced by drugs. *Drug Saf.* 1996; 15:219-31.
3. Lin K, Guilhoto L.M. F. F., Yacubian E.M.T. Drug-induced Gingival Enlargement – Part II. Antiepileptic Drugs: Not Only Phenytoin is Involved. *J Epilepsy Clin Neurophysiol* 2007; 13(2):83-88.
4. Kimball OP. The treatment of epilepsy with sodium diphenyl hydantoinate. *J Am Med Assoc.* 1939; 112:1244-5.
5. Dongari-Bagtolou A. Dug-associated gingival enlargement. *J Periodontol* 2004;75:1424-1431.
6. Seymour RA, Smith DG, Turnbull DN. The effects of phenytoin and sodium valproate on the periodontal health of adult epileptic patients. *J Clin Periodontol.* 1985; 12:413-9.
7. Steinberg AD. Clinical management of phenytoin-induced gingival overgrowth in handicapped children. *Pediatric Dentistry* 981; 3 (special issue): 130-136
8. Carranza FA. *Gingival enlargement: Carranza's Clinical Periodontology.* 10th ed. W.B.Saunders. Philadelphia; 2006. p 373-390
9. Silverstein LH, Garnick JJ, Szikman M, Singh B. Medication induced gingival enlargement: a clinical review. *Gen Dent.* 1997;45:371-6.
10. Marshall RI, Bartold PM. A clinical review of drug-induced gingival overgrowth. *Aust Dent J* 1999;44:219-232.

Corresponding author:

Dr. Saumiya Gopal,
Reader,
Dept. of Periodontics,
Rama Dental College and Research Centre,
Kanpur, Uttar Pradesh.
Phone: 09918006605.
E-mail: saumiyagopal@gmail.com

How to cite this article: Roopa DA, Choudary S, Shukla P, Gopal S Drug Substitution and Non-Surgical Therapy in Phenytoin Induced Gingival Enlargement. *Rama Univ J Dent Sci* 2015 June;2(2):39-42.

Sources of support: Nil

Conflict of Interest: None declared