Case Report

Drug induced gingival enlargement – A menace to the gingiva: Case Report
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Abstract
A case of gingival enlargement was reported to the department of Periodontics and Implantology with the chief complaint of difficulty in mastication and poor esthetics. Detailed medical history of the patient was taken and patient was clinically evaluated. After investigations patient was diagnosed as a case of drug induced gingival enlargement. With the physicians consent phenytoin was substituted by valproic acid. There was a decrease in the gingival enlargement after the drug substitution but it did not resolve completely. So after 6 months gingivectomy procedure was carried out, this gave better results. We had successfully treated the patient for his chief complaint. Antiepileptic drugs can cause drug induced gingival enlargement. So if enlargement occurs, consideration should be given to substitution of drugs with another drug that does not induce gingival enlargement or has a very low prevalence of enlargement. If the enlargement does not resolve completely by drug substitution, then surgical approach should be opted for treatment.

Keywords
Gingival enlargement, drug induced, gingivectomy, phenytoin, tuberculoma.

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Drug Induced Gingival Enlargement

Introduction

A well-known consequence of administration of certain calcium channel blockers, immunosuppressants and anticonvulsants is enlargement of the gingiva. Apart from the primary target tissues, the effects of these drugs are also directed towards gingival connective tissue causing histopathological and clinical abnormalities (1). Difficulties that are associated with gingival overgrowth are speech, aesthetics, tooth eruption, mastication, impaired nutrition, oral diseases and periodontitis.

The most common neurological disorder seen in humans is epilepsy (2). Treatment of choice for treating grand mal, psychomotor and temporal lobe epilepsy is phenytoin (dilantin) and it was first introduced in 1938 by Merritt and Putman. Dilantin is a hydantoin and first case of drug induced gingival enlargement were those reported by phenytoin (3). The prevalence of gingival enlargement in patients taking phenytoin is around 50%, although different authors have reported incidences ranging from 3% to 84.5% (4). Other hydantoins that induce enlargement of gingiva include mephenytoin and ethotoin.

Age and plaque has been suggested as important predisposing factors for development of gingival overgrowth. It occurs most often in younger age and occurrence and severity are not necessarily related to dosage after threshold levels have been exceeded. The clinical features of enlargement are presented as enlarged interdental papilla resulting in nodular or lobulated shape usually limited to marginal and attached gingiva and more frequently observed in the anterior region of the jaw. Due to the enlargement, plaque control becomes difficult and a secondary inflammatory process may set in. Due to the side effects of phenytoin on the periodontium and the secondary complications occurring, we have focused this case of gingival enlargement. In the current case report we have discussed the clinical picture and treatment of phenytoin induced gingival enlargement.

Case Report

A 13 year old male child who was a known case of seizure disorder since 2 years was referred to the department of Periodontics and implantology with the chief complaint of generalized swollen gums since 2 months (Fig 1a, 1b).

Patient's medical history revealed history of epilepsy since 2 years and was on medication for same. Detailed medical history and medical reports documented that patient had a seizure attack 2 years back and at the time of attack there was rigidity of jaws which lasted for 15 to 20 minutes.
Patient was immediately taken to hospital and CT scan and MRI investigations revealed tuberculoma of right parietal lobe (Fig 2).

![Image](image.png)

**Fig 2 - tuberculoma seen in the right parietal lobe shown by arrows.**

After the first attack there was no history of another seizure attack but medications for epilepsy and tuberculosis were started. Patient took antitubercular drugs (INH 150 mg and rifampicin 250mg) for 1 year after which the tuberculoma resolved and antitubercular drugs were stopped. For epilepsy, patient was on antiepileptic drugs (phenytoin 50 mg three times a day) and antiepileptic medication is still continued. The symptoms reported by the patient included difficulty in mastication, poor esthetics, and for these problems patient requested treatment. There was no history of bleeding from gums and swelling was not associated with pain. On examination the gingival tissues were firm, fibrotic, pale pink in colour, enlarged with pronounced stippling.

The investigations carried out in our institute included orthopantamograph and complete blood count. Complete haemogram reports showed the values were under normal limits. Based on the clinical examination and detailed medical history of the patient and after ruling the other causes of generalized gingival enlargement, child was diagnosed as a case of phenytoin induced gingival hypertrophy. Physicians consent was taken to switch the child to another antiepileptic drug i.e. Valproic acid (100mg TDS).

Complete professional oral prophylaxis was carried out and patient was prescribed with mouth rinse (chlorhexidine for 7 days). Patient was recalled after 15 days and it was seen that the gingival condition improved. Patient was asked to maintain oral hygiene and was recalled for supportive periodontal therapy after 1 month, 3 month and 6 months. Even at 6 month recall visit after maintaining hygiene and changing the drug, there was substantial enlargement of gingival tissues which interfered with patient’s esthetics. Patient’s physician was consulted and reports revealed that there was no systemic history that would contraindicate the surgical procedure (gingivectomy). A written consent was obtained from the patient for gingival surgery.

Surgical procedure – On clinical examination at 6 month recall visit, gingival enlargement was associated with only lower anterior region of jaw. A day prior to surgery local anaesthesia sensitivity test was done which revealed no sensitivity to local anesthesia. Under strictly aseptic conditions, 2-3 ml of local anaesthesia was infiltrated in the mandibular anterior region and gingivectomy was performed for the lower anterior sextant. Firstly, pocket marker was used to mark the deepest point in each pocket on the radicular and interproximal surfaces. A series of bleeding points were obtained after marking the pockets which served as a guideline for initial external bevel incision (using 15 no. blade) (Fig 3).
This incision was followed by a sulcular incision and orban’s knife was used to remove the interproximal tissue. After removing the enlarged tissue, gingivoplasty was done with surgical scissors (Fig 4).

Periodontal dressing was applied to the surgical area, postoperative instructions were given, analgesics (ibuprofen 400 mg and paracetamol 325 mg BID after meals for 3 days) and antibiotics (amoxicillin 500 mg BID for 5 days) were prescribed and patient was recalled after 8 days. After 8 days, surgical site showed uneventful healing. Tissue sample that was removed was sent for biopsy and histopathological examination revealed gingival hyperplasia with inflammatory cell infiltrate and predominance of fibrotic component (Fig 5).

Patient was recalled 1 month after surgery to see the healing and gingival tissues status (Fig- 6a, 6b).
Discussion

Phenytoin induced gingival enlargement is most commonly seen in younger age group. The proliferation of fibroblast like cells and epithelium is stimulated by phenytoin. There is production of inactive fibroblastic collagenase and thus decrease in collagen degradation (5). Also in vitro experiments have shown that there is increase synthesis of glycosaminoglycans (GAG’s) by fibroblast in gingival enlargement (6). Fibroblast are less active in non-inflamed gingiva and do not respond to circulating phenytoin. Whereas in an inflamed tissue these fibroblast are in an active state because of the growth factors and inflammatory mediators present (7).

The exact pathogenesis of phenytoin induced gingival enlargement is not known but some evidence links it to a direct effect on specific, genetically predetermined subpopulations of fibroblasts, inactivation of collagenase, and plaque-induced inflammation. Gingival enlargement is not observed in all patients taking phenytoin. When gingival enlargement occurs as a results of phenytoin administration it can be of 3 types.

Type I – Non-inflammatory hyperplasia. On substitution to another anti-epileptic drug, enlargement disappears in few months.

Type II – Chronic inflammatory enlargement not related to administration of phenytoin and can be caused by local irritants. It can be an inflammatory enlargement resembling that caused by phenytoin.

Type III – Combined enlargement. Hyperplasia is caused by inflammation due to local irritants and phenytoin administration (8).

Drug induced gingival enlargement in susceptible individuals can be ameliorated by strict plaque control and removal of local irritants but it cannot be prevented. Thus these susceptible individuals should be kept on proper periodontal maintenance therapy with recall visits every 3 months to reduce the prevalence of gingival enlargement and optimum patient care (9). Patients with drug induced gingival enlargement should be managed in a step wise manner with appropriate consultation of patient’s physician, substitution of drug, oral prophylaxis and strict plaque control and surgical therapy if required.

Conclusion

Gingival enlargement appears to be a result of interaction between keratinocyte, collagen and fibroblast which is present in gingiva with phenytoin and its metabolite. Drug substitution should be considered for patients presenting drug induced gingival enlargement followed by surgical therapy if needed.

References

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