RESEARCH PAPER

Drug-induced gingival overgrowth

Omar Sadiq*

*Faculty of Medicine, Arab American University Jenin, Palestine

Received: 24th April, 2026; Revised: 20th May 2026; Accepted: 30th June, 2026; Available Online: 10th July, 2026

ABSTRACT

Drug-induced gingival overgrowth (DIGO) is a pathological growth of gingival tissue, primarily associated with calcium channel blockers and immunosuppressants. Consequently, it is mainly seen in cardiovascular and transplanted patients. Nifedipine remains the main calcium channel blocker related to the development of this unpleasant side-effect. As for immunosuppress-ants, cyclosporin is the leading causative agent, whereas other drugs from this druggroup, including tacrolimus, have better safety profiles. Accumulated collagen with inflammatory infiltrates is the histological hallmark of this condition. Several factors are involved in the pathogenesis and can increase the risk, such as male gender, younger age, pre-existing periodontal inflammation, and concomitant use of other DIGO-inducing medications. Patients with DIGO may experience severe discomfort, trouble with speech and mastication, pain, and teeth loss, aside from cosmetic implications. Furthermore, these patients also have an increased risk for cardiovascular diseases. The interdisciplinary approach and cooperation with dental care experts are necessary for patient management. Treatment includes discontinuing the drug and switching to one with a better profile, improving oral hygiene, and surgical removal of enlarged tissue. This article throws light on respective drugs and their association with gingival overgrowth and approaches to treatment based on current knowledge and investigative observations.

Keywords: Drug-induced gingival overgrowth; Calcium channel blocker; Nifedipine

How to cite this article: Sadiq O, Drug-induced gingival overgrowth. Int J Drug Deliv Technol. 2026;16(74s): 232-240. DOI: 10.25258/ijddt.16.74s.26

Source of support: Nil.

Conflict of interest: None

INTRODUCTION

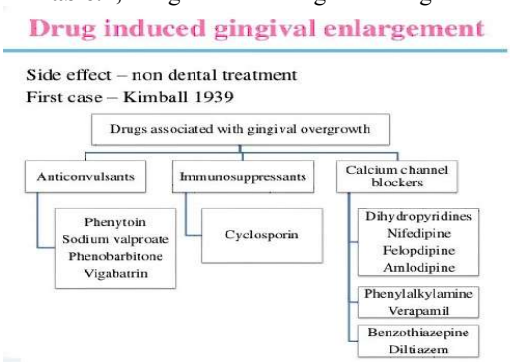
Drug-induced gingival overgrowth (DGO) belongs to the group of iatrogenic gingival diseases with functional and aesthetic repercussions which can alter the quality of life. This periodontal disease was reported for the first time in 1939 as an adverse effect of phenytoin. Subsequently, gingival enlargement was attributed to other drugs**.[1,2]**

Drug-induced gingival enlargement is classified by the American Academy of Periodontology as a dental plaque-induced gingival disease, as evidence suggests that existing gingival inflammation may be necessary for its development and that proper plaque control and effective oral hygiene can lessen its severity or potentially prevent its occurrence altogether. **[1-3]**

Currently, there are over 20 medications from three pharmaceutical categories including anticonvulsants, calcium channel blockers and immunosuppressants that are associated with gingival enlargement**.[4]** It is the responsibility of the dental practitioner to recognize the potential of these medications to contribute to gingival enlargement and to provide the proper prophylactic care or appropriately refer the patient for periodontal therapy. A team approach involving a consultation with a periodontist and the patient's physician is a critical step in successful treatment. **[1-6]**

This article throws light on respective drugs and their association with gingival overgrowth and approaches to treatment based on current knowledge and investigative observations.

Table.1; Drug-Induced Gingival Overgrowth



Etiology of Drug-Induced Gingival Overgrowth

*Author for Correspondence: Omar Sadiq

Gingival overgrowth is caused by three categories of drugs: anticonvulsants, immunosuppressants, and calcium channel blockers. These drugs are sometimes taken for the remainder of a patient's life because of chronic health conditions being treated, such as organ transplantations**.[1-5]** A clear understanding of the etiology and pathogenesis of drug-induced gingival overgrowth has not been established.6 The three different classes of drugs that produce gingival overgrowth might share some common metabolic pathway, or they could produce a similarly appearing clinical condition from totally different mechanism**.[1-7]** Some theories have focused on the direct effects of the drug or its metabolites on specific gingival cells of the periodontium, particularly on gingival fibroblasts. Spoildorio et al. reported that as the severity of overgrowth increases, there are parallel increases in collagen and fibroblasts and a decrease in blood vessel content, possibly explaining the light pink appearance of the enlarged gingival tissue. **Table.1**

Seymour et al. **[3]** concluded that the following factors might contribute to the expression of drug-induced gingival overgrowth:

Genetics

Age

Gender

Concomitant Medication

Drug Variables

Periodontal Variables

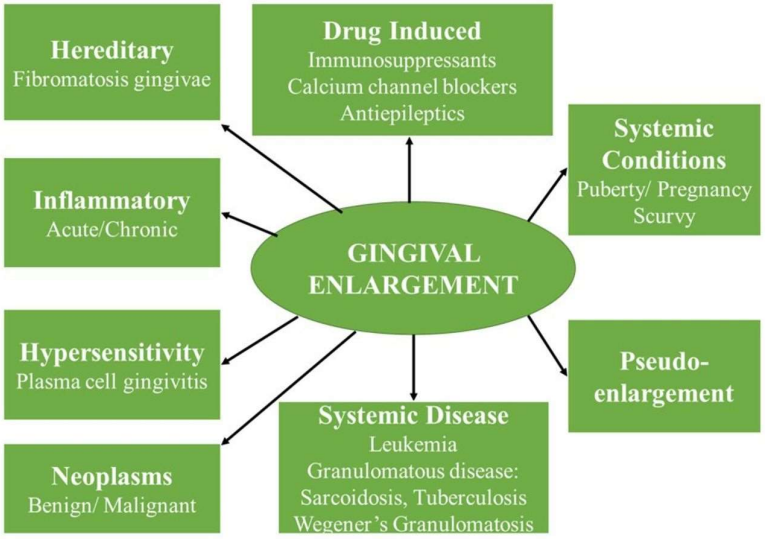
Genetics, gender, and age are factors that cannot be modified. Drug combinations, such as therapy with immunosuppressants and calcium channel blockers, are often necessary and can result in additive effects.Drug variables such as dose, serum, tissue, and salivary concentrations of the medication suggest that the effect could be dosage-dependent but that, in order for a drug to be effective, it must reach certain threshold levels. A reduction in the dose of medication is not warranted

just to prevent the adverse side effects.

There appears to be a significant correlation between plaque/gingivitis and gingival overgrowth.However, Thomason et al. questioned whether the gingival overgrowth is the cause or the result of the increased inflammation and plaque levels.

In a study of the prevalence and risk of gingival overgrowth in patients treated with anticonvulsant drugs, Brunet et al. found that gingival inflammation is a significant risk factor for gingival overgrowth in these patients.9 Periodontal variables, in contrast to the previously mentioned factors, may be modified and controlled. Therefore, oral health care professionals play an important role in the prevention and control of this condition because of the significant correlation noted between plaque/gingivitis and gingival overgrowth.

Table .2; Clinical And Pathogenesis

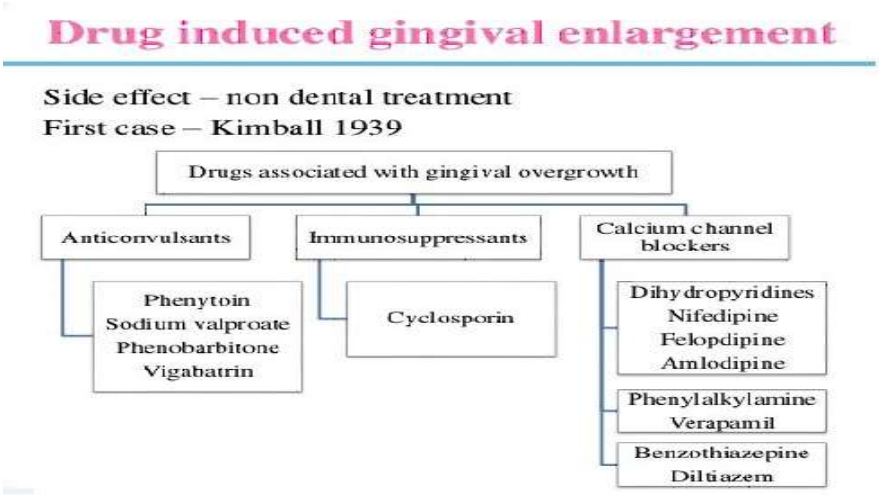


CLINICAL FEATURES AND PATHOGENESIS

DIGO usually starts as painless enlargement of interdental papillae and progresses towards facial and lingual margins, covering the teeth crowns**.[1-7]** Fully developed, itforms generalized changes throughout the mouth, affecting the anterior gingiva the most, although it can also occur as a localized lesion. A possible explanation for the predominance of lesions in anterior regions could be higher exposure of anterior gingiva to the irritation resulting from plaque. DIGO initially appears as pink, **[8,9]** lobulated, and thickened gingival tissue without concomitant inflammation, with no tendency to bleed. In its course

it becomes inflamed with red or bluish-red discolorations and frequent bleeding. As it progresses, it spreads both vertically and horizontally and affects mastication and speech.In advanced forms, enlarged gingiva may even interfere with the occlusion. **[8,9-12] TABLE.2**

Table .3; Possible-mechanisms-for-drug-induced-gingival-overgrowth-Modified-from-Harel-Raviv-et



Patients with DIGO have problems maintaining oral hygiene, which leads to susceptibility to infections and periodontal disease and may result in the loss of the teeth. A weakened immune system predisposes to more severe periodontal diseaseand puts patients on immunosuppressants at additional risk. Furthermore, periodontitis may potentially carry a risk for cardiovascular diseases, including myocardial infarction, peripheral artery disease, stroke, and heart failure. In theory, possible mechanisms behind this association could be dissemination of oral pathogens into the bloodstream and invasion of cardiovascular organs and tissues to induce inflammatory response on a local and systemic level. Additionally, periodontitis is associated with endothelial dysfunction, an important factor in atherosclerosis development. Aside from the functional impairment and cardiovascular risk, gingival changes also represent a significant esthetic problem for the patients. **Table.3**

Etiopathology of DIGO is multifactorial and not fully understood. Genetic factors(cytochrome P450, HLA, and MDR1 gene polymorphisms) influence the interindividual difference in gingival response to DIGO-inducing drugs and could have a role in identifying patients at risk. The main histological finding in DIGO isthe accumulation of collagen in the gingiva's extracellular matrix, along with the infiltration of inflammatory cells. Most of the DIGO-inducing drugs act as inhibitors of calcium ion influx. An inhibited influx of cations into fibroblasts causes a decrease in cation-dependent folic acid uptake. Folic acid is necessary for the proper function of matrix metalloproteinases, which activate collagenase. With no collagenase, there is no collagen breakdown, and it accumulates in connective tissue[1]. Furthermore, studies demonstrated a drug-induced increase in glycosaminoglycans[1] and collagen production along with the proliferation of gingival fibroblasts. These changes are mainly mediated by inflammatory cytokines that are a part of an inflammatory response to drugs. Inflammatory infiltrates found in the gingiva mainly consist ofplasma cells. Periodontal status is a significant predictor of DIGO, as bacterial plaque induces inflammation. A significant correlation was found between bacterial plaque and a higher risk for DIGO in patients treated with CCBs or cyclosporin.

In gingival tissue of patients with CCBs-associated DIGO, higher expression of androgen receptors accompanied with higher levels of type I collagen were detected, implying androgens' role in its pathogenesis.

Medications That Cause Gingival Overgrowth

Drug-induced gingival enlargement or otherwise called drug-influenced gingival enlargement[6] or “drug induce gingival overgrowth” abbreviated as “DIGO.”

Gingival enlargement is mostly associated with administration of three different classes of drugs producing almost similar response which are phenytoin, cyclosporine, and dihydropyridines.[8] “Phenytoin-induced gingival overgrowth” is a most common side effect of phenytoin[9] observed in epileptic patients in taking phenytoin affecting 50% of patients. Cyclosporine is an immunosuppressant causing gingival enlargement in 25–80% of patients. The calcium channel antagonist, in particular, dihydropyridines (e.g. nifedipine, felodipine, and amlodipine) is also commonly associated with gingival enlargement.

Table.4; Common-drugs-that-cause-gingival-enlargement

Class of drugs	Generic name
Immunosuppressants	Cyclosporine
	Everolimus
	Sirolimus
	Mycophenolate mofetil
Calcium-channel blockers	Amlodipine
	Benidipine
	Nicardipine
	Nifedipine
Anticonvulsants	Carbamazepine
	Diazepam
	Phenytoin
	Clobazam
	Gabapentin
	Phenobarbital
	Topiramate
	Valproic acid
	Primidone
	Zonisamide
	Levetiracetem

- Anticonvulsants (such as phenytoin, phenobarbital, lamotrigine, valproate, vigabatrin, ethosuximide, topiramate, and primidone**).[10]**
- Calcium channel blockers, such as nifedipine, amlodipine, and verapamil. The dihydropyridine derivative is radipidine can replace nifedipine and does not induce gingival overgrowth**.[10]**
- Cyclosporine, an immunosuppressant**.[10]**

Almost all cases of drug-induced gingival enlargement are due to phenytoin, cyclosporine, and calcium channel blockers. Cyclosporine is an immunosuppressant which is used in organ transplant recipients and for treating psoriasis. Other calcium channel blocker agents such as nifedipine and amlodipine also induce gingival enlargement.

Nifedipine tends to have an additive effect when used in combination with cyclosporine in transplant recipients with hypertension**.[13]** Not all patients on phenytoin, cyclosporine, and calcium channel blockers develop gingival enlargement**.[14]** The etiology of drug-induced gingival enlargement is multifactorial. The pathogenesis and etiology of gingival enlargement are not clearly known**.[15-17]**

Anticonvulsants

Phenytoin, known better by its brand name Dilantin (Parke-Davis Company), has been widely used to control convulsive disorders since 1938, when it was first used as an anticonvulsant/antiepileptic drug. Scientists have developed other drugs for the treatment of seizures, but phenytoin is still the preferred drug for the treatment of epilepsy, particularly with grand mal, temporal lobe, and psychomotor seizures. It is also widely used in the treatment of some forms of neuralgia and cardiac arrhythmias. Phenytoin is usually prescribed for chronic use, and approximately 50% of those who take it develop gingival overgrowth. **[1,8,9]**

Gingival changes resulting from phenytoin drug therapy usually begin within two weeks to three months. The marginal gingiva and the interdental papillae appear to be the areas predisposed to enlargement. The appearance of phenytoin-induced gingival overgrowth, in the absence of gingivitis, is usually firm, pink, and somewhat pebbly. In severe cases, teeth surfaces are completely covered with gingival overgrowth. Figure 4 illustrates gingival overgrowth on a patient who has been taking phenytoin for several years. Studies of patients taking this anticonvulsant drug suggest that bacterial plaque is an important determinant of the severity of phenytoin-induced gingival overgrowth and stress the importance of instituting preventive plaque control programs, principally in young patients taking this drug.17-19

Immunosuppressants

Cyclosporine is a fungal derivative with immunosuppressive effects.3 This medication has a wide range of biological activities, showing antiparasitic, antifungal, anti-inflammatory, and antiproliferative action. In addition to its primary use to prevent organ rejection, cyclosporine's antiproliferative action has led to its use to treat severe psoriasis, ichthyosis vulgaris, and rheumatoid arthritis. **[1-15]**

While cyclosporine has been the drug of preference since the beginning of the transplant era, a number of longitudinal and crossover studies have reported an incidence of gingival problems associated with the drug in the range of 25% to 70%.20 Other frequent side effects associated with cyclosporine are damage to the liver and kidneys, increased hair growth, and trembling of the hands. However, cyclosporine has been a revolutionary drug that enables patients to receive lifesaving organs without rejection. **[15-19]**

Another major side effect associated with cyclosporine is increased blood pressure. Calcium channel blockers, such as nifedipine, are usually the drugs of choice to

control this problem. As mentioned previously, calcium channel blockers have their own adverse effects on the gingival tissues.3 In combination, cyclosporine and calcium channel blockers produce an intensified effect, and the severity of the gingival enlargement is greater. **[19-24]**

Fortunately, advances in pharmacology are providing new options to physicians for the immunosuppression of transplant patients. New drugs are being evaluated, and there is a possibility that, in the future, adequate immunosuppression will be achieved without such noxious side effects as gingival overgrowth. **[1-5,8,9]**

Calcium Channel Blockers

Calcium channel blockers are used for the treatment of many cardiovascular disorders, including angina, arrhythmias, hypertension, and acute myocardial infarction. They are considered first-choice anti-hypertensive drugs for patients who also exhibit problems with angina or peripheral vascular disease. The most frequently prescribed calcium channel blockers, the most common side effects of which include headache, dizziness, facial flushing, edema, and gingival overgrowth. A direct relationship between plasma concentration of the drug and the degree of gingival enlargement does not seem to exist, but the prevalence of gingival overgrowth with the use of calcium channel blockers has been reported as high as 38%.**[19-25]**

In calcium channel blocker-induced gingival overgrowth, the onset of gingival enlargement is commonly noticed between the first and second months of drug therapy. The severity of enlargement is usually greater in the anterior of the mouth, as in the patient who is taking nifedipine (Procardia®, Adalat®). This severity is usually greater with nifedipine (20% to 30%) than with other calcium channel blockers.Several clinical studies show that patients taking nifedipine are at a high risk for gingival overgrowth, and that gingivitis acts as a factor that predisposes patients to develop gingival overgrowth when they are taking calcium channel blockers. **[25-29]**

DIFFERENTIAL DIAGNOSIS

A differential diagnosis requires thorough medical and dental histories, a careful evaluation of nature of enlargement, and an identification of the etiologic factors. A biopsy specimen may be required to confirm diagnosis. **[29-34]**

Drug-induced gingival overgrowth must be differentiated from inflammatory enlargement: Acute inflammatory enlargement appears as a localized gingival swelling characterized by acute pain of rapid onset suggesting an abscess. Chronic inflammatory enlargement appears as deep red or bluish red and soft, friable with smooth, shiny surface along with bleeding tendency. Inflammatory enlargements usually are a secondary complication to any of the other types of enlargement, creating a combined gingival enlargement. **[2-36]**

Idiopathic or familial or hereditary gingival enlargement: It affects the attached gingiva, as well as the gingival margin and interdental papillae. The facial and lingual surfaces of the mandible and maxilla are generally affected, but the involvement may be limited to either jaw. The color of enlarged gingivais pink with firm and leathery in consistency. The etiology is unknown**.[20-27]**

Conditioned enlargement: It occurs when the systemic condition of the patient exaggerates or distorts the usual gingival response to dental plaque. It includes hormonal-like pregnancy, puberty, nutritional conditions like associated with Vitamin C deficiency, and allergic conditions like plasma cell gingivitis. The gingiva shows features of chronic inflammatory enlargement, especially interproximally. Plasma cell gingivitis consists of lesion located in the oral aspect of attached gingiva**.[21-27]**

Systemic diseases induced gingival enlargement: Several systemic diseases, namely, leukemia, sarcoidosis, tuberculosis, and other granulomatous diseases can result in gingival enlargement. Hematological investigations as in leukemia and histopathological examination such as leukemic infiltrate in leukemia, foreign body giant cell in sarcoidosis, tuberculosis are useful in establishing the diagnosis. **[34-38]**

Neoplastic enlargement or gingival tumors: It may appear as slowly growing spherical mass that tends to be firm and nodular or hard and wart-like protuberance from gingival surface. **[38-44]**

False enlargement: These are not true enlargements of the gingival tissues but appear as such. These result due to increase in size of the underlying osseous or dental tissues. The gingiva usually presents with no abnormal clinical features except the massive increase in size of area.

Gingival overgrowth induced by various drugs is differentiated from one another as:

In phenobarbitone treated patients, gingival overgrowth occurs without lobulations of the interdental papillae. The lesions develop often in posteriors than in anteriors.

In individuals, immunosuppressed with cyclosporin, sometimes pebbly or papillary lesions appear on the surface of larger lobulations, which have been associated with the presence of Candida hyphae invading the gingival epithelium. In phenytoin-induced gingival overgrowth, gingival tends to bleed rapidly whereas in cyclosporine-induce

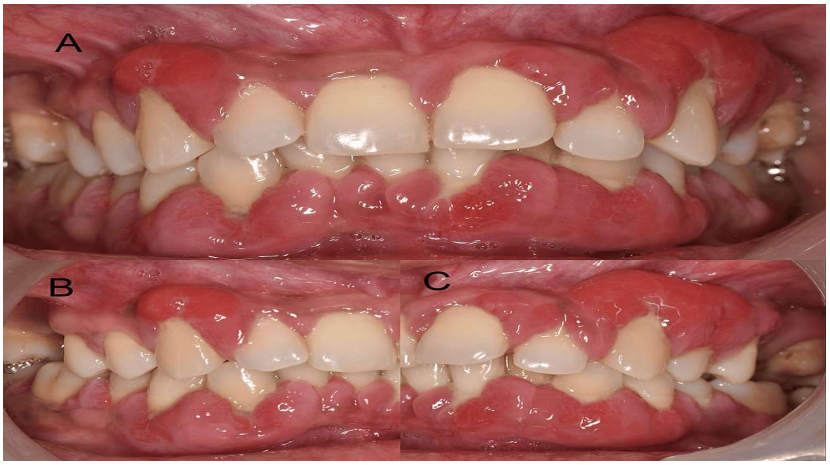


Figure.1;The Management of Drug-Induced Gingival Enlargement in a Patient

Diagnosis

The diagnosis of drug-induced gingival overgrowth is mainly based on the clinical appearance of the gingivae and on the medical history.

The histopathological features of the phenytoin drug-induced gingival enlargements are mainly characterised by proliferation of morphologically normal fibroblasts and by an increased amount of collagen. **[3,4-38]** Figure.1

The histopathological features of the other drug-induced gingival enlargements are similar and characterised by a collagenous connective tissue with little or no inflammatory exudates. The connective tissue is highly vascularised and there are focal accumulations of inflammatory infiltrates dominated by plasma cells, that may be suggestive of a neoplastic process. **[1-8]**

INVESTIGATIONS

1. Complete hemogram: patient with severe gum bleeding to rule out any hematological cause (leukemia) or complication (anemia due to bleeding gums).
2. Liver function tests for increased transaminases.
3. Plasma, serum and salivary concentration of the PNT.
4. Culture – to rule out oral candidiasis.
5. Tissue biopsy –in an unusual presentation of gum overgrowth.
6. Imaging – Periapical (full mouth series) or Panorex (panoramic view) prior to treatment to evaluate the status of periodontal tissue or any compromised teeth.

Treatment

The management of GE is essentially multidisciplinary which includes medical, surgical and supportive care. **[1-7]**

Medical Care

The most effective treatment of drug-induced gingival enlargement is withdrawal or substitution of medication i.e. PNT. Unfortunately, not all patients respond to this mode of treatment, especially those with longstanding gingival lesions .

A) Antimicrobials: These are used for the treatment of GE and associated infections like periodontitis. But some of them are interfering with collagen synthesis and breakdown also.

1. **Macrolides-** Azithromycin, Roxithromycin: Arrests GE by inhibition of expression of gene for collagen type 1 leading to decreased protein synthesis. Also there is up regulation of mRNA for MMP -2, a cell surface associated type 1 collagen degrading MMP causing increased degradation of collagen. It is also thought to reduce the viability of gingival fibroblast as it blocks the DNA synthesis. **[22-24]**

2. Tetracyclines-

Doxycycline/Minocycline- inhibits specific MMP`s required for collagen synthesis. **[25,34-38]**

B) Antiseptic mouth wash-

I. Chlorhexidine gluconate-

II. Biotene (Lysozyme, lactoferrin, glucose oxidase, lactoperoxidase[**38-44]**

C) Folic acid 1 mg one to three times a day is to be supplemented to take care of PNT induced folate deficiency.

SURGICAL CARE: This option is sought in moderate to severe cases, not resolving even after decreasing the dose of the offending drug, maintaining proper oral hygiene, not responding to professional debridement with scaling or root planning. It includes Flap procedures which clean the root of a tooth and repairs bone damage caused by gum diseases. The other option is Gingivectomy. If carried out with routine surgical methods, there is rapid recurrence . Hence the preferred

method is the use of CO2 or YAG Laser , an approach which provides rapid postoperative hemostasis. **[32-44]**

SUPPORTIVE CARE: This involves informing patients of the risk of developing GE secondary to PNT therapy and the role of oral health in minimizing complications.

Routine dental examination is recommended to control development of dental plaque. As presence of plaque predisposes to GE, removal of DRUG INDUCED GINGIVAL ENLARGEMENTplaques, calculus deposits often reduces mild and moderate GE thus avoiding surgical intervention.

Proper cleaning (brushing and flossing) of each tooth separately is must. Patients should practice thorough oral hygiene twice a day (i.e., before breakfast, before going to bed) and rinse mouth with plain water after each meal.

Tooth staining due to mouthwash is an anticipated side effect. It can be prevented by brushing teeth prior to rinsing out with chlorhexidine. No dietary restriction as such are there for preventing GE but once it ensues; one should minimize sweets, carbohydrates, soft drinks.

PROGNOSIS: The condition has a better prognosis if patients maintain regular oral hygiene and plaque control.

SPECIAL CONSIDERATIONS:

1. Many cases of GE will respond to local treatment i.e. scaling and root planning. But if it poses considerable trouble to the patient i.e. enlargement covers more than about a third of the tooth surface, one should consider substituting the drug if dose reduction is not possible in an individual case. This approach may bring about partial or complete regression of the lesion. Most patients will observe an alteration in the soft tissues within a few days .
2. Several alternatives to PNT are available, but they may not be as well tolerated or they may not control seizures as well. Recently, the feasibility of phenytoin substitution has increased with the addition of a new generation of anticonvulsants such as lomatrigine, gabapentin, carbamazepine and topiramate. Some patients can switch to a lower dose of PNT combined with another anticonvulsant.
3. There is every possibility of recurrence of GE despite periodontal treatment if a person continues taking the same offending medication.
4. Discontinuance of PNT therapy may result in gradual regression of the lesion over the time (26).
5. Dental plaque is not essential for the development but related to its severity, though not all the patients with poor oral hygiene develop this.

CONCLUSION

Drug-induced gingival enlargement is a common sequela to treatment with anticonvulsants, calcium channel blockers and immunosuppressants. Evidence suggests that gingival inflammation is critical in its pathogenesis. While it may be prevented through meticulous periodontal maintenance and home care, it is essential for the dentist to work together with the patient's physician and periodontist in order to successfully treat this condition once it occurs.

REFERENCES

1. Newman MG, Takei HH, Klokkevold PR, Carranza FA. Carranza's Clinical Periodontology. 11th ed. St. Louis, Mo.: Elsevier/Saunders; 2012. p. 84-96.
2. G. & C. Merriam Company. Merriam-Webster's Medical Desk Dictionary. Springfield, MA: G. & C. Merriam Company; 2002. p. 367-8.
3. Seymour RA, Ellis JS, Thomason JM. Risk factors for drug-induced gingival overgrowth. J Clin Periodontol. 2000 Apr;27(4):217-23. doi: 10.1034/j.1600-051x.2000.027004217.x. PMID: 10783833.
4. Lindhe J, Lang NP, Karring T. Clinical Periodontology and Implant Dentistry. 5th ed. Oxford: Blackwell Munksgaard; 2008. p. 641.
5. W.B. Saunders. Carranza'a Clinical Periodontology. 9th ed. Philadelphia, PA: W.B. Saunders; 1996.
6 . Hardman JG, Limbird L. Goodman Gillman A. The pharmacological basis of therapeutics. 10th Ed. NewYork: McGraw-Hill Co.; 2003.
7. Droździk A, Droździk M. Drug-Induced Gingival Overgrowth-Molecular Aspects of Drug Actions. Int J Mol Sci. 2023 Mar 13;24(6):5448. doi: 10.3390/ijms24065448. PMID: 36982523; PMCID: PMC10052148.
8. Ciancio SG, Bartz NW Jr, Lauciello FR. Cyclosporine-induced gingival hyperplasia and chlorhexidine: a case report. Int J Periodontics Restorative Dent. 1991;11(3):241-5.
9. Trackman PC, Kantarci A. Molecular and clinical aspects of drug-induced gingival overgrowth. J Dent Res. 2015 Apr;94(4):540-6. doi: 10.1177/0022034515571265. Epub 2015 Feb 13. PMID: 25680368; PMCID: PMC4485217.
10. Mavrogiannis M, Ellis JS, Thomason JM, Seymour RA. The management of drug-induced gingival overgrowth. J Clin Periodontol. 2006 Jun;33(6):434-9. doi: 10.1111/j.1600-051X.2006.00930.x. PMID: 16677333.
11. Dalal R, Garg S, Gupta A. Nonsurgical Management of Drug-induced Gingival Overgrowth in a Young Patient. Int J Clin Pediatr Dent. 2023

12. Moffitt ML, Bencivenni D, Cohen RE. Drug-induced gingival enlargement: an overview. Compend Contin Educ Dent. 2013 May;34(5):330-6. PMID: 23991852.ov;16(Suppl 3):331-334. doi: 10.5005/jp-journals-10005-2482. PMID: 38268630; PMCID: PMC10804294.

13. Damdoum M, Varma SR, Nambiar M, Venugopal A. Calcium Channel Blockers Induced Gingival Overgrowth: A Comprehensive Review from a Dental Perspective. J Int Soc Prev Community Dent. 2022 Jun 29;12(3):309-322. doi: 10.4103/jispcd.JISPCD_57_22. PMID: 35966914; PMCID: PMC9369783.

14. Bajkovec L, Mrzljak A, Likic R, Alajbeg I. Drug-induced gingival overgrowth in cardiovascular patients. World J Cardiol. 2021 Apr 26;13(4):68-75. doi: 21569887. 10.4330/wjc.v13.i4.68. PMID: 33968305; PMCID: PMC8069521

15. Nakib N, Ashrafi SS. Drug-induced gingival overgrowth. Dis Mon. 2011 Apr;57(4):225-30. doi: 10.1016/j.disamonth.2011.03.010. PMID: 21569887.

16. Tungare S, Paranjpe AG. Drug-Induced Gingival Overgrowth. 2022 Sep 19. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan–. PMID: 30860753.

17. Kanade K. Drug Induced Gingival Enlargement. Munich: GRIN Verlag, 2018: 2-10

18. Desai P, Silver JG. Drug-induced gingival enlargements. J Can Dent Assoc. 1998 Apr;64(4):263-8. PMID: 9594464.

19. Chojnacka-Purpurowicz J, Wygonowska E, Placek W, Owczarczyk-Saczonek A. Cyclosporine-induced gingival overgrowth-Review. Dermatol Ther. 2022 Dec;35(12):e15912. doi: 10.1111/dth.15912. Epub 2022 Oct 17. PMID: 36208445.

20. Tungare S, Paranjpe AG. Drug-Induced Gingival Overgrowth. 2022 Sep 19. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan–. PMID: 30860753.

21. Seymour RA, Ellis JS, Thomason JM. Risk factors for drug-induced gingival overgrowth. J Clin Periodontol. 2000 Apr;27(4):217-23. doi: 10.1034/j.1600-051x.2000.027004217.x. PMID: 10783833.

22. Thomason JM, Kelly PJ, Seymour RA. The distribution of gingival overgrowth in organ transplant patients. J Clin Periodontol 1996;23(4):367-371.

23. Galas-Zgorzalewicz B, Borysewicz-Lewicka M, Zgorzalewicz M, Borowicz-Andrzejewska E. The effect of chronic carbamazepine,valproic acid and phenytoin medication on the periodontal condition of epileptic children and adolescents. Funct Neurol 1996;11(4):187-193.

24. Katz J, Givol N, Chaushu G, Taicher S, Shemer J. Vigabatrin-induced gingival overgrowth. J Clin Periodontol 1997;24(3):180-182.

25. Pernu HE, Pernu LM, Knuuttila ML. Effect of periodontal treatment on gingival overgrowth among cyclosporine A-treated renal transplant recipients. J Periodontol 1993;64(11):1098-1100.

26. Russo J. Periodontal laser surgery. Dent Today 1997;16(11):80-81.

27. Guelmann M, Britto LR, Katz J. Cyclosporin-induced gingival overgrowth in a child treated with CO2 laser surgery: a case report. J Clin Pediatr Dent 2003;27(2):123-126.

28. Ikawa K, Ikawa M, Shimauchi H, Iwakura M, Sakamoto S. Treatment of gingival overgrowth induced by manidipine administration. A case report. J Periodontol 2002;73(1):115-122.

29. Lozada-Nur F, Vacharotayangul F. Drug-induced gingival hyperplasia. eMedicine Medical Library. Available at http://www.emedicine.com/derm/topic645.htm [cited 2004 Aug 23].

30. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4(1):1-6.

31. Hodges K. The importance of PM. Dimensions of Dental Hygiene 2003;1Suppl 7:12-20.

32. Slots J. Update on general health risk of periodontal disease. Int Dent J 2003;53Suppl 3:200-207.

33. Omar Sadiq*, Rand Ghoul**,Abu-Hussein Muhamad*;(2025)Dental Management of Cardiovascular Compromised Patient.Journal of Neonatal Surgery,14(22s), 154-168

34. Abu-Hussein Muhamad., et al. “Analgesics and Orthodontic Tooth Movement". Acta Scientific Dental Sciences 9.4 (2025): 84-97.

35. Omar Sadiq, Rand Ghoul , Nidal Ghannam and Abu-Hussein Muhamad, (2025 Effect Of Hormone on Orthodontic Tooth Movement: A Review. Journal of Neonatal Surgery, 14 (22s), 189-201

36. Watted N, Ghoul R, Sadiq O, Watted A, Abu Hussein M. Chemico-Biological Approach Accelerating Orthodontic Tooth Movement. Int J Drug Deliv Technol. 2026;16(61s):907-913. DOI: 10.25258/ijddt.16.61s.99

37. Muhamad Abu Hussein, Nezar Watted. (2025) Biology of Tooth Movement. Int Dent Jour. ;4(4):1- 14. (4):458–68.

38. Muhamad Abu-Hussein, Nezar Watted, Edlira Zere, Péter Borbély(2016); Periodontally Accelerated Osteogenic Orthodontics with Piezoelectric Surgery: A Case Report. Journal of Dental and MedicalSciences 2016,15,2, 64-68

39. Abu-Hussein M, Watted N, Hegedűs V, Péter B, BillJ. (2015) Orth odontic Treatment of Periodontall y Damaged Teeth – An Interdisciplinary Approach. JAdv Med Dent Scie Res ;3(3):1-6

40. Abu-Hussein M., Watted N,. HegedűsV., Borbély P(2015); Corti cotomy in the Modern Orthodontics, Journal of Dental and Medical Sciences , 14 (11), 68-80

41. Abu-Hussein Muhamad etl(2025), Platelet-Rich Plasma in Orthodontics, International Journal of Electrical Power System and Technology, Volume 49 Issue 4 (December),3430- 44

42. Abu-Hussein Muhamad etl(2025), Role of StemCells in Orthodontics International Journal ofElectrical Power System and Technology, Volume 49 Issue 4 (December ,2955-79

43. Mask AGJr.. Medical management of the patient with cardiovascular disease. Periodontol 2000 2000;23:136-141.

44. Pilatti GL, Sampaio JE. The influence of chlorhexidine on the severity of cyclosporine A-induced gingival overgrowth. Erratum in: J Periodontol 1998 Jan;69(1):102] . J Periodontol 1997;68(9):900-904.23.