International Journal of Research in Health and Allied Sciences

Journal home page: www.ijrhas.com

Official Publication of "Society for Scientific Research and Studies" [Regd.]

ISSN: 2455-7803

# Case Report

## Clinical management of Patient with phenytoin induced gingival enlargement- a case report

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***ABSTRACT:***
Several distinct classes of drugs, such as anticonvulsants, immunosuppressants, and calcium channel blockers, caused gingival overgrowth. One of the main drugs associated with the gingival overgrowth is the anti-epileptic such as phenytoin, which affects gingival tissues by altering extracellular matrix metabolism. In our study, we evaluate the effect of phenytoin, a drug whose active substance is phenytoin, on gingival fibroblasts of healthy volunteers. Gene expression of 29 genes was investigated in gingival fibroblasts' cell culture treated with phenytoin compared with untreated cells. Among the studied genes, only 13 genes (CXCL5, CXCL10, CCR1, CCR3, CCR5, CCR6, IL-1A, IL-1B, IL-5, IL-7, IL-6R, BMP-2, and TNFSF-10) were statistically significant. All but one gene resulted downregulated after 24 h of treatment with phenytoin. BPM2 was the only, although weakly, up-expressed gene. Probably, we have not highlighted overexpression of the other inflammatory molecules because the study was performed on healthy people. Many studies show that phenytoin induces the overexpression of these cytokines but, probably, in our study, the drug does not have the same effect because we used gingival fibroblasts of healthy people.
**Keywords**: Gingival enlargement, phenytoin, platelet rich fibrin

Received: 23 Jan, 2023 Accepted: 30 Jan, 2023

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**This article may be cited as:** Sharma H, Jyoti D, Bansal N, Meenakshi, Kumar K. Clinical management of Patient with phenytoin induced gingival enlargement- a case report. Int J Res Health Allied Sci 2023; 9(3):17- 21

### INTRODUCTION

The term "gingival hyperplasia" usually used to describe an oral phenomenon seems nowadays used inappropriately because enlargement is not the result of an increase in the number of cells, but rather an increase in extracellular tissue volume.[1] "Gingival enlargement" is the term used to describe medication-related gingival overgrowth or gingival hyperplasia. It can be defined as an abnormal growth of the periodontal tissue.[2]

This pathology causes not only aesthetic changes and clinical symptoms, such as pain, tenderness, bleeding, speech disturbances, and abnormal tooth movement, but also dental occlusion problems, enhancement of caries development, and periodontal disorders.[3] Drug-induced gingival enlargement (DIGE) connected with the chronic use of the anti-epileptic drug (AED) phenytoin (PHT) was reported first in 1939 by Kimball.[4] In the same year, Faurbye[5] and in 1959, Strean and Dilantin[6] suggested that the alkalinity of PHT could be the cause of the gingival side effect.

### CASE REPORT

A 29-year-old female patient with swollen gums arrived complaining mostly of difficulties speaking, chewing, pain, and bleeding during brushing. The patient's medical history revealed that she had epilepsy and had been treated with an AED combination for 12

years, including phenytoin, carbamazepine, and eslicarbazepine, as well as phenobarbitone for the previous 8 months. The family history was not relevant. The patient noted the enlargement over the last 8 months, which was painless and progressing in nature, according to the history of the current sickness. In the few places that covered the occlusal surface of the posterior teeth, there was generalised gingival expansion during the intraoral clinical examination. Except for the locations where inflammation was overlaid, the expansion had a faintly lobulated surface, was firm, and durable. The patient experienced difficulties speaking, chewing, and maintaining good oral hygiene as a result of the growth. Bone loss was seen uniformly over the orthopantomogram. Routine blood tests came out negative for any anomalies. So, it was determined that the gingival hypertrophy was caused by AED. The proposed treatment strategy included phase I therapy to minimise inflammation, and a phase II gingivectomy operation to remove bulk tissue.

Every appointment, spaced by a week, a quadrant was scheduled for the gingivectomy process. An accurate identification of the pockets on all surfaces of all teeth within the surgical field was carried out with the aid of a pocket marker at three points/tooth (mid radicular, mesial, and distal line angles) on the facial/buccal and lingual/palatal aspects after achieving adequate anaesthesia using 2% xylocaine HCl with adrenaline (1:2,00,000). Using a no. 15 Bard Parker blade and a surgical periodontal knife from Kirkland, an external bevel incision was made. Using an Orbans knife, interdental incisions were performed, and curettes were used to remove the majority of the tissue. Surgical scissors made by Goldman Fox were used to cut off tissue tags. Next, the surgical site was examined for any lingering debris. In locations where bone exposure was seen, PRF was put and covered with a tin foil shield before periodontal dressing was applied. Following surgery, the patient was given instructions to take analgesics (a 500 mg paracetamol and 50 mg diclofenac combination) three times per day for three days. The remaining quadrants underwent the same surgical treatment. Healing went smoothly.

**Figure 1: Preoperative photograph**

**Figure 2: Preoperative OPG**

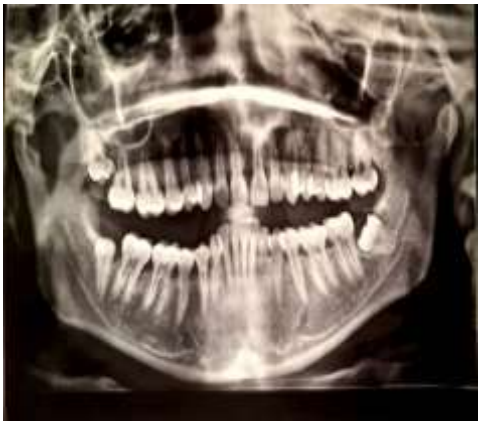


**Figure 3: Post phase 1 therapy**

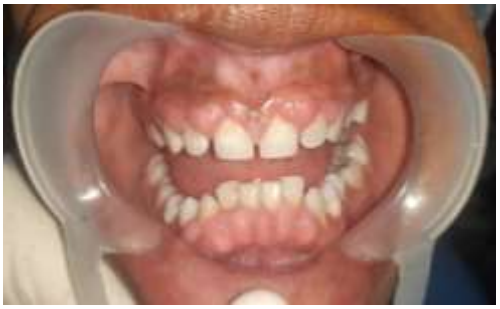


**Figure 4: POCKET DEPTH MEASUREMENT**

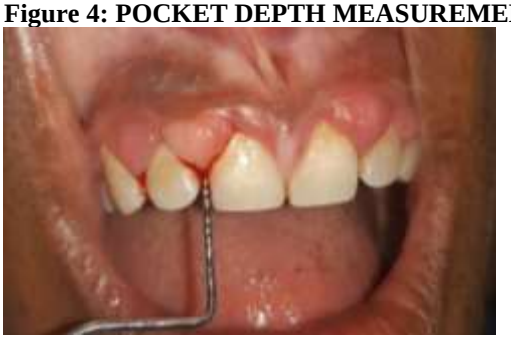


**Figure 5: POCKET MARKING**

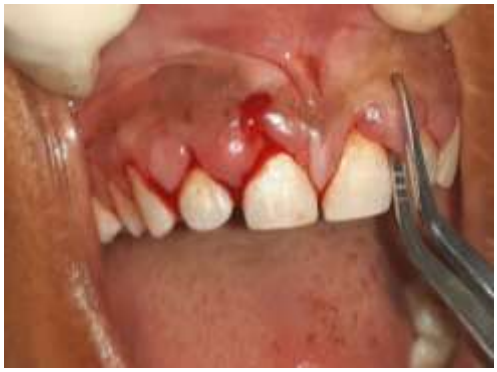


**Figure 6: BLEEDING POINTS**

**FIGURE 7**

**FIGURE 8**

**FIGURE 9**

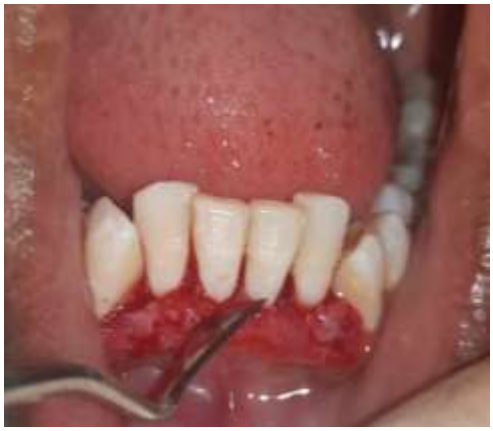


**FIGURE 10**

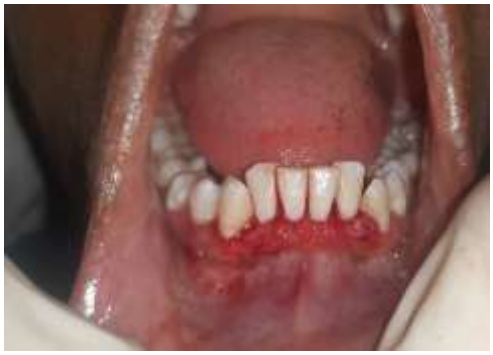
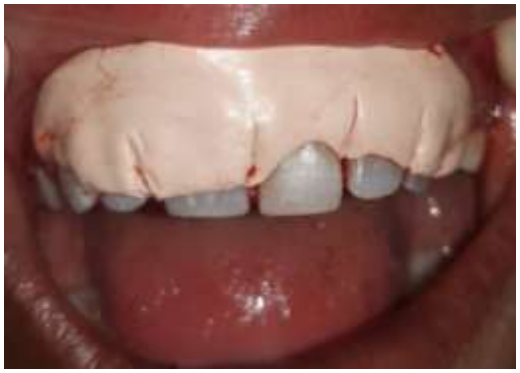


**FIGURE 11:** COE-PAK PLACED



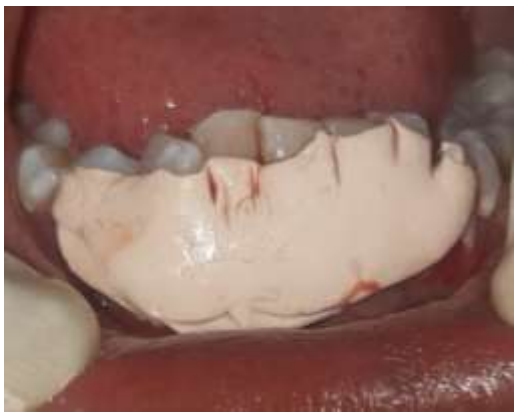


**FIGURE 12 : INCISION DONE**

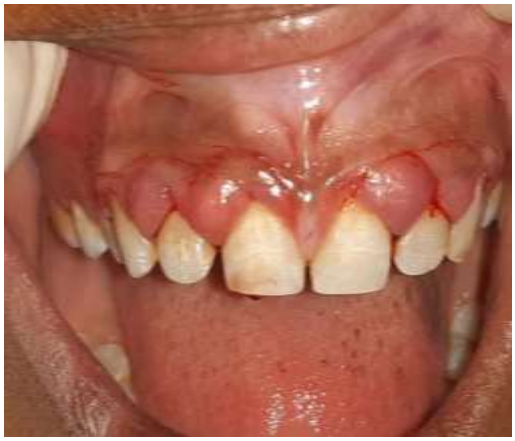


**Figure 13:** GINGIVECTOMY DONE

**FIGURE 14: POST-OPERATIVE PHOTOGRAPH AFTER 6 MONTHS**

**Discussion**

Drug-induced gingival overgrowth is a common complication of the continuous use of medications, such as anticonvulsant phenytoin, antihypertensive calcium channel blockers (nifedipine), and immunosuppressant cyclosporine-A therapy.[7] Reports about the possible etiological mechanisms of drug-induced gingival overgrowth have been suggested such as an imbalance in collagen synthesis and the degradation of gingival connective tissue, predominantly due to the inhibition of collagen phagocytosis of gingival fibroblasts.[8,9,10] Additionally, cytokines and connective tissue growth factors could have an important role in gingival overgrowth. Deregulation of these balances could cause an abnormal differentiation of fibroblasts, resulting in their accumulation with proliferative and synthetic phenotypes Phenytoin is a commonly prescribed medication for the treatment of patients with epilepsy. Kato et al.[11] have suggested that Phenytoin-induced gingival overgrowth (PIGO) was probably due to an imbalance in collagen degradation, rather than an increase in collagen synthesis. These authors demonstrated that the gingival fibroblasts demonstrate a specific extracellular matrix metabolism, stimulating, for example, prostaglandin E2 (PGE2) production and accumulation by gingival fibroblasts. One such study done by Ramesh Kumar et al.[12] showed that the presence of systemic autoimmune diseases may pose a risk for the development of periodontal diseases. He also showed that all patients with APS had periodontal disease. Epilepsy is the utmost common chronic neurological disorder in human, and phenytoin remains the drug of choice for control of seizures in cerebral palsy.[13] Clinically, gingival enlargement frequently appears within 1 year of the initiation of treatment with the phenytoin drug.[14]

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