Non-Surgical Approach to Phenytoin-Associated Gingival Enlargement: A Case Report

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Abstract

Aim: The aim of this case demonstrate a possible case of phenytoin-induced gingival overgrowth.
Methodology: In this case report, the initial results of periodontal treatment are reported for a 24-year-old epileptic male patient who presented at the Periodontology Department of the Dentistry Faculty, Dicle University, suffering from gingival enlargement. Phenytoin was changed to carbamazepine after consultation with his physician. After the patient received oral hygiene instruction, initial periodontal treatment (scaling and root planing) was performed.
Results: In periodic follow-up visits, appropriate oral hygiene and reduction of the enlargement were observed. Thus, no periodontal surgical treatment was planned. Follow-up visits at 3-month intervals are ongoing.
Conclusions: This case report illustrate that prescribing phenytoin is an important issue for epileptic patients.

Introduction

Epilepsy is a condition in which a person has recurrent seizures, due to a chronic underlying process. A seizure is a paroxysmal event, due to abnormal central nervous system activity, which can have various manifestations, ranging from dramatic convulsive activity, with or without loss of consciousness, to phenomena not even discernible by an observer (1-3).

There are significant side effects of many of the drugs used to treat epilepsy. One of these is gingival tissue growth.

Indeed, gingival enlargement or overgrowth has been associated with multiple factors, including inflammation, adverse drug effects, and neoplastic conditions. Chronic inflammation due to the accumulation of dental plaque frequently causes gingival overgrowth (4-7).

Drugs associated with gingival enlargement include anticonvulsant drugs such as phenytoin (8, 9), the immunosuppressant cyclosporin (10, 11), and calcium channel blockers such as dihydropyridines (12, 13), verapamil (14), and diltiazem (15, 16). Of these, phenytoin is known to stimulate responsive subpopulations of gingival fibroblasts to accumulate extracellular matrix components, including collagen and glycosaminoglycans, resulting in gingival enlargement (17). The clinical features of drug-induced gingival enlargement include marked, lobulated enlargement of the buccal and lingual
gingiva, most noticeable in the anterior region of the oral cavity. Secondary inflammation, caused by local irritation, frequently results in pain, redness, bleeding, and further progression of the hyperplastic gum tissue.

In addition to being a painful condition, gingival hyperplasia may result in tooth displacement and periodontal disease, which can interfere with eating, speech, and the patient’s physical appearance (18). The treatment of gingival enlargement depends on the severity of the problem. Appropriate oral hygiene is recommended for all patients to control the inflammatory component and decrease the severity of gingival enlargement. However, in severe cases of persistent gingival enlargement, amelioration may require periodontal surgical or laser intervention to remove gum tissue (gingivectomy) or conversion of the anticonvulsant regimen from phenytoin to an alternate anticonvulsant (19).

**Case Report**

A 24-year old male patient who presented at the Periodontology Department of the Dentistry Faculty, Dicle University, was evaluated for swollen and bleeding gums. His past medical history included epilepsy with irregular seizures. He was treated with phenytoin. After drug treatment, the patient complained of progressive gingival enlargement. Oral examination revealed marked buccal and lingual gingival enlargement in the anterior and posterior maxillary and mandibular regions. The enlargement was more significant interdentally. Areas of acute gingivitis related to plaque deposits were also identified (Fig. 1 and 2).

We consulted with his physician regarding the conversion from phenytoin to an alternate anticonvulsant. After consultation, the patient was prescribed tegretol. Following this conversion, initial periodontal treatment was begun (scaling and root planing). Twice daily chlorhexidine rinses were recommended and the patient was instructed how to maintain proper oral hygiene. After 3 weeks, the patient's gingival enlargement had regressed. After initial periodontal treatment, resolution of the gingival lesions was observed (Fig. 3 and 4). Thus, no periodontal surgical treatment was performed. The patient was referred to the Department of Conservative Dentistry and Endodontics for the treatment of caries. The 26th tooth was extracted because of deep caries. Follow-up visits at 3-month intervals are ongoing.

**Figure 1.** Appearance of the upper jaw before therapy.

**Figure 2.** Appearance of the mouth before therapy.

**Figure 3.** Radiological images.
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Discussion

It has been suggested that gingival enlargement occurs only when phenytoin is present in the antiepileptic regimen. Since 1939, when gingival enlargement was first described by Kimball and Kimball (9) a 57% incidence of gingival enlargement has been reported in patients treated with phenytoin (9). Phenytoin is known to stimulate responsive subpopulations of gingival fibroblasts to accumulate extracellular matrix components, including collagen and glycosaminoglycans, resulting in gingival overgrowth (17, 20).

In clinical studies, the incidence of gingival enlargement in patients treated with phenytoin has ranged from 3-93% (21, 22). This wide range may be attributed to the small number of cases reported in some publications, to large variations in the dose of phenytoin prescribed, to variations in the length of phenytoin exposure, and to differences in the ages of the patients (1, 8).

Also, the pathogenic mechanisms of phenytoin-induced gingival overgrowth seem to be affected by other factors, including dental plaque. Indeed, several studies have found a relationship between the quantity of accumulated dental plaque and phenytoin-induced gingival overgrowth (5, 20, 23-25).

A logical approach to the control of gingival enlargement induced by drugs should be reduction of the dose of the drug or substitution of the drug for another (1, 26-28). Phenytoin is the drug of choice for some types of epilepsy; however, it can often be replaced with second-generation drugs such as carbamazepine and sodium valproate (1). On the other hand, several investigations have demonstrated the use of effective periodontal therapy and anti-plaque agents for the prevention of phenytoin-induced gingival overgrowth (29).

This report demonstrates a possible case of phenytoin-induced gingival overgrowth. Our results are consistent with those in the studies above. Before initial periodontal treatment, phenytoin was replaced with carbamazepine. In this way, the treatment of gingival overgrowth was achieved without surgery. Research and this case study illustrate that prescribing phenytoin is an important issue for epileptic patients, and that physicians should consider the issue of gingival overgrowth when prescribing it.

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References