

Role of Phenytoin on Gingiva of Epileptic Patients

Abhinav Jha, K.M.K. Masthan and Sudha Jimson

Department of Oral Pathology and Microbiology,
Sree Balaji Dental College and Hospital, Bharath University, Chennai, India

Abstract: Epilepsy is defined as a chronic neurological disorder which is associated with recurrent seizures of cerebral origin. Epilepsy is one of the most common neurologic disease affecting almost 45 million people worldwide. More than often epilepsy demonstrates bimodal age distribution consisting of infancy and old age. Till today, the scenario surrounding the management of epilepsy is complex. Phenytoin is one of the oldest anti-epileptic drugs used for its management. Like any other drugs, phenytoin causes gingival enlargement which is the most common adverse effect seen in epileptic patients.

Key words: Epilepsy • Phenytoin • Gingival Enlargement

INTRODUCTION

Phenytoin induced gingival enlargement is seen in almost 50-100% of patients. Clinically, gingival overgrowth usually starts from papillary region. Gingival enlargement which is induced due to anti-epileptic drugs appears to be firm and pale due to fibrous component. Despite of many recent anti-epileptic drugs, phenytoin is the drug of choice for many clinicians. Apart from other adverse effects, gingival enlargement is the most common of them [1]. The pseudopockets are formed due to gingival enlargement, produces plaque bearing areas, which ultimately enhances the patient's susceptibility to dental caries, inflammation of gingival and periodontal problems [2, 3].

Phenytoin and Gingiva: Epilepsy is neurological disorder of brain with an abnormal electrical activity. The enlargement of gingiva can occur due to various stimuli. The treatment of epilepsy is based upon freedom from seizures. The first reported case of gingival enlargement due to phenytoin was in 1939 [4]. Phenytoin-induced gingival enlargement can be noticed within 3 months of its use and reaches a static point within a year of its administration [5]. Phenytoin-induced gingival growth is seen mostly in children and adolescents but with little or no difference in relation to its gender or ethnic groups [6]. Intra-orally gingival overgrowth is mostly seen in buccal surfaces of both

upper and lower anterior teeth region [7]. Recently, it has been noted that phenytoin enhances the production of IL-6 and IL-8 by fibroblasts [8]. Another most important link was noticed between systemic fibrotic diseases to gingival growth is the presence of myofibroblasts in phenytoin induced tissues [9]. The management of gingival growth depends upon its etiopathogenesis. However, lots of studies indicated multifactorial etiology which includes oral hygiene status of the epileptic patients [10]. All patients on phenytoin therapy did not show gingival growth as it is dependent on other associated risk factors. The histopathology shows hyperplasia of both epithelium and connective tissue. Acanthosis of epithelium with elongated rete-pegs shows densely arranged collagen fibers with new blood vessels and fibroblasts. Few areas may show abundance of amorphous-like material [11]. The treatment modality includes gingivectomy/gingivoplasty, scaling, root planning and maintenance of oral hygiene. Other adverse effects of phenytoin includes ataxia, weakness, skin rashes, sedation, agranulocytosis, pruritis, slurred and in some cases it may lead to confusion.

CONCLUSION

World-wide various drugs are associated with gingival enlargement among which phenytoin is one of them and this should be identified prior to the treatment. A thorough investigation should be made and the

Corresponding Author: Abhinav Jha, Department Of Oral Pathology and Microbiology,
Sree Balaji Dental College and Hospital, Bharath University, Chennai-600100, Tamil Nadu, India.

clinician should be aware of the etiology and pathogenesis related to the unwanted side-effects of phenytoin induced gingival overgrowth. Currently, ongoing studies are based upon the pathogenic mechanisms of phenytoin induced gingival enlargement which focuses on direct and indirect effects on gingival fibroblasts mechanism. Finally, this review indulges and encourages the clinicians worldwide to work on the histology and mechanism of this drug and in future come out with other therapeutic options to overcome the adverse effects and make the management of epileptic patients more feasible and accurate.

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