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CASE REPORT

DRUG INDUCED GINGIVAL ENLARGEMENT: A CASE REPORT

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ABSTRACT

Drug induced gingival enlargement, a common adverse drug reaction in patients treated with anticonvulsants, calcium channel blockers and immunosuppressant. The management requires a proper understanding of case and a multidisciplinary approach including medical, surgical and supportive care. Here, we are discussing a case report of drug induced gingival enlargement in a patient undergoing antiepileptic drug and antihypertensive drug. The treatment options included substitution of drug, measures to improve oral hygiene and gingivectomy.

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INTRODUCTION

Enlargements of the gingiva and soft tissues of periodontium depends on the various interactions between the host and environment (William *et al.,* 1999 & 2000). Among the several known etiologies of gingival enlargements, drug induced gingival enlargement is the most recognized one. The common causative drugs inducing gingival enlargement are phenytoin, phenobarbitone, amlodepine, nifedepine and cyclosporine (Devesh d gosavi *et al.,* 2000). Kimball in 1939 reported the first drug-induced gingival enlargement associated with chronic usage of the anti-epileptic drug phenytoin (Seymour *et al.,* 1996). The treatment of epilepsy, a common chronic neurological disorder is based on drug therapies aiming to reduce the seizure attacks. As the common drug of choice in treatment for grand mal, temporal lobe, and psychomotor epilepsy is phenytoin, the side effect of gingival enlargement has to be suspected (Devesh d gosavi *et al.,* 2000). Even though the exact pathogenesis of drug induced gingival enlargement is unknown, these enlargements are seemed to be induced through disruption in homeostasis of collagen synthesis and degradation of gingival connective tissues (Ching-Wen Chang *et al.,* 2012). Amlodipine, a third-generation calcium channel blocker(CCB) is a common drug of choice as it shows longer action and weaker side effect compared to the previous generations. The various side effects of amlodipine includeheadache, dizziness, edema, flushing, palpitation and rarely gingival enlargement.

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The first case of amlodipine induced gingival enlargement was reported in 1994 by Ellis and Seymour et al., 2014. The combined use of cyclosporine and calcium channel blockers have shown increased prevalence and severity of gingival enlargement compared with the monotherapy of the both. Very few reports are available about the gingival enlargement in patients receiving a combination of phenytoin and a CCB (Ching-Wen Chang *et al.,* 2012). This case report presents a case of probable synergism with the combined use of phenytoin and amlodipine that caused extensive gingival overgrowth in a patient with epilepsy.

CASE REPORT

A 29 years old male patient was referred to department of periodontology KVG Dental College with a chief complaint of swollen gums in his upper and lower front teeth region since one year. The patient is uncomfortable with the enlargement as it was interfering with his routine tooth brushing and mastication. The medical history revealed that the patient is epileptic since childhood hypertensive since 2 years and mild mentally retarded. Patient was under medication on phenytoin sodium 100mg twice daily and amilodipine 10 mg twice daily since 4 years. The enlargement was noticed from past 1 year which increased its size gradually. Intraoral examination showed gingival enlargement in upper and lower anterior tooth region extending from canine to canine region. In maxilla the enlargement was seen interdentally and to a small extend in marginal gingiva suggestive of grade 1 (angelopouloos and goaz grading). In mandible the gingival enlargement showed a

grade 3 gingival enlargement including interdental gingiva, marginal gingiva and attached gingiva covering more than half of the clinical crown (fig: 1 and fig2). Periodontal pockets measured more/equal to 5 mm in both upper and lower anteriors. Lobulated interdental papilla with blunt edges were seen in lower anterior tooth region. On the basis of medical history, drug history and clinical examination of the patient, a provisional diagnosis of phenytoin-amlodipine combined drug induced gingival enlargement was given. A complete hemogram of the patient was done and all parameters were in normal range. Orthopantomogram was done to check the bone loss which revealed mild horizontal bone loss in upper and lower anteriors.

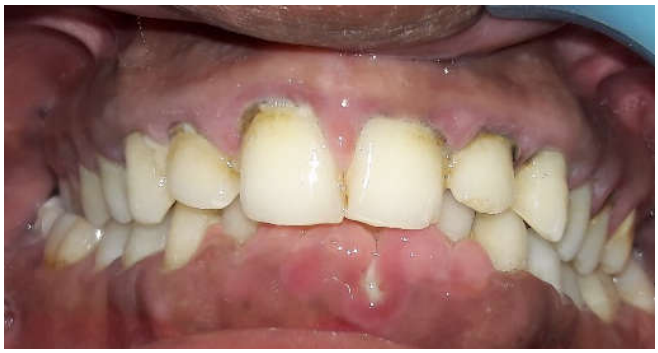

Figure 1.

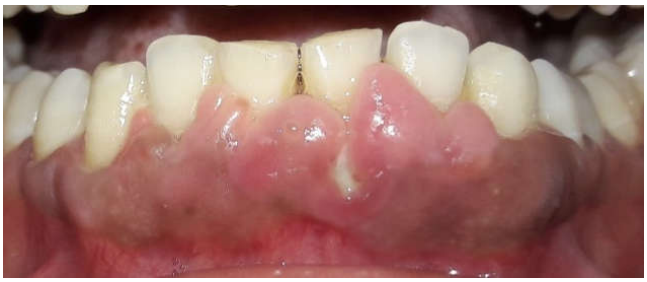

Figure 2.

Treatment

Patient was referred to physician for opinion regarding dental treatment procedures and local anesthetic injection. The substitution drug was given for the patient and opinion regarding dental treatment procedures were obtained. The initial treatment started with phase 1 therapy which included scaling and root planning with adjunctive prescription of chlorhexidine mouthwash 0.2% twice daily. Patient was advised tomaintain good oral hygiene and brushing techniques was shown. A drastic change was noticed after 3 weeks in the revisit. The regression of maxillary gingival enlargement size to grade 0 was a remarkable change. The mandibular enlargement reduced from grade 3 to grade 2 with some amount of fibrotic component left unresolved (fig 3 and fig 4).

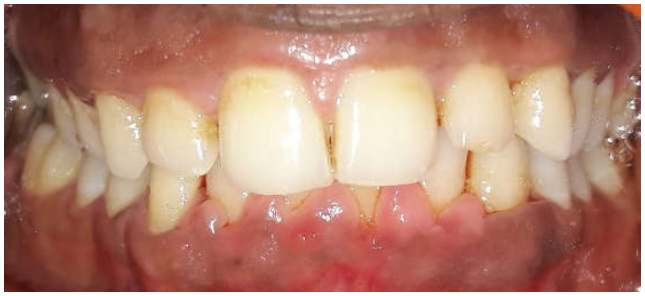

Figure 3.

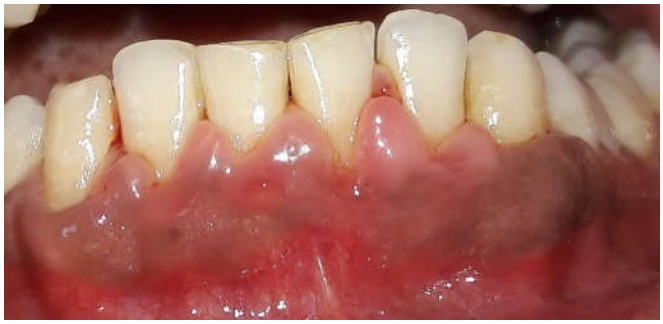

Figure 4.

A surgical therapy was planned to completely eliminate the remaining amount of enlargement in the lower anteriors. A 980nm diode laser was used with 400nm fiber optic cable at 3.00 unit power to remove the remaining enlarged portions. After the procedure, a betadine irrigation was done and periodontal dressing given for 7 days.

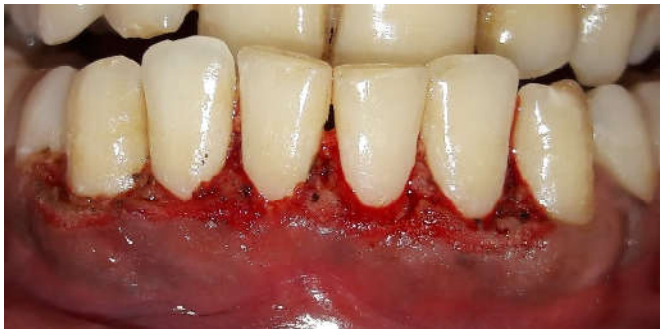

Figure 5.

Post-operative instructions and analgesics were given. Healing was uneventful without any post operative complications (fig 5).

DISCUSSION

The increase in gingival tissue size in whole or in part from systemic drug use is termed as drug induced gingival enlargement. Till now more than 20 drugs have been identified as a causative agent for gingival enlargement (Informational Paper Drug-Associated Gingival Enlargement, 2004). The common drugs causing gingival enlargements includes anticonvulsants like phenytoin, immunosuppresants like cyclosporine and calcium channel blockers like nifedipine, amlodipine, few cases are also reported in association with valproic acid, erythromycin, vigabatrin (Michelle *et al.,*). The prevalence rate of drug induced gingival enlargement varies among different drugs as follows: 3-84.5% for phenytoin (Fermin), 25-75% for cyclosporine (Fermin) and 0.5-83% for nifedipine[7] 1.7-3.3% for amlodipine (Informational Paper Drug-Associated Gingival Enlargement, 2004). Epilepsy is the repeated occurrence of sudden, excessive and/or synchronous discharges in cerebral cortical neurons resulting in disruption of consciousness, disturbance of sensation, movements, impairment of mental function, or some combination of these signs (James Bowman, 2001). The antiepileptic drugs will act either by depressing the neuronal activity at the focus of origin or by blocking the spreading mechanisms of seizures (Ravi PrakahSasankoti Mohan, 2013). Gingival enlargement was identified as an adverse effect of the most common antiepileptic drug phenytoin in 1939, just a year after its introduction (Arya and Gulati, 2012). Calcium channel

blockers, a commonly prescribed medication for conditions like hypertension and angina have shown an adverse effect of gingival enlargement. The first reported case of CCB induced gingival enlargement was by lederman et al and ramon et al in 1980 in nifedipine medicated patient, followed by diltiazem, verampamil and in amlodipine and felodipine induced gingival enlargement (Shobana *et al.,* 2017). Gingival enlargement caused by amlodipine begins at the interdental papilla, usually starting within 6 months of drug intake (Shobana *et al.,* 2017). The phenytoin induced enlargements usually starts within 3 to 6 months of drug intake with first sign of enlargement affecting the interdental papilla (Arya and Gulati, 2012). The present case gives a history of enlarged gums since 1 year.

Regardeless of the etiology gingival enlargement may be problematic and leads to increased risk for dental decay, periodontal diseases and esthetic disfigurements (Informational Paper Drug-Associated Gingival Enlargement, 2004). Seymour et al proposed a multifactorial model for explaining the pathogenesis of drug induced gingival enlargement which involves an interaction of several factors depending on the interaction between drug and metabolite with the gingival fibroblasts. The predisposing factors includes age, genetic predisposition, pharmacokinetic variables, drug-influenced alterations in gingival connective tissue homeostasis, histopathology, ultrastructural factors, inflammatory changes and drug-induced actions on growth factors (Shobana *et al.,* 2017). GE appears to be a result of interaction of susceptible subpopulation of fibroblasts, keratinocytes and collagen present in gums with drug & its metabolite. Matrix metalloproteinases (MMP`s) may also play a role because of their major role in cellular proliferation, migration, differentiation, and angiogenesis (Devesh Gosavi *et al.,* 2000). The treatment for drug induced gingival enlargement should always starts with substitution of drug if possible and nonsurgical therapy, aiming at reducing inflammation thereby resolving the condition.

Although gingival enlargement and oral hygiene has not shown significant relationship, several authors have mentioned the importance of good oral hygiene in controlling the drug induced gingival enlargement as it may cause secondary inflammatory responses (Ana *et al.,* 2014). In this present case scenario, the patient's physician have given the substitution drugs and kept the patient on observation period of one month. The substitution drug and phase 1 therapy have shown a drastic change in the gingival enlargement. The remaining fibrotic enlargement in mandibular anterior region was eliminated surgically.

The decision of choosing the technique whether scalpel gingivectomy, electrocautery or laser depends on the clinician. Here, in this case laser gingivectomy was done to remove the excess tissues.

Conclusion

A combination of non surgical and surgical therapy was used in the present case and showed complete healing with less postoperative complications. In this present case the combined non-surgical and surgical therapy resulted in complete healing of the gingival overgrowth with less post operative complication. Yet there is a need of post therapy maintenance and long term follow up in order to evaluate the long term recurrence of gingival overgrowth in patients who had not replaced Amlodipine with alternative drug. Within the limitation of the present study it has been concluded that combined treatment would be more effective in managing combined enlargement cases.

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