Case Report

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SURGICAL TREATMENT OF DRUG INDUCED GINGIVAL ENLARGEMENT: A CASE REPORT

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ABSTRACT:

Drug induced gingival enlargement may be defined as abnormal growth of the gingiva due to an adverse drug reaction in patients treated with anticonvulsants, immunosuppressants and calcium channel blockers. Patients undergoing treatment for epilepsy with Phenytoin (PHT) are frequently affected. Occurrence of such lesions among younger age group is more than the adults. Here in this case report we present the clinical, histopathological features and treatment of inflammatory gingival enlargement which disturbed the aesthetics and masticator function of the patient. A 27-year-old male reported with the chief complaints of swelling and mild pain in the gums of all the teeth in both the jaws. The gingival enlargement was firm and fibrotic accompanied by an inflammatory component probably due to inability to maintain adequate personal oral hygiene and using anti-epileptic drugs like PHT. Surgical therapy was carried out to provide a good aesthetic outcome. No recurrence was seen at the end of 3 months.

Keywords: Anticonvulsants, Drug-Induced, Epilepsy, Gingival Enlargement, Phenytoin.

INTRODUCTION:

Epilepsy, the most common chronic neurological disorder in humans has a prevalence of approximately 1% in developed countries, rising to 2% in less developed nations.[1] The term 'Drug-induced gingival enlargement' (DIGE) refers to gingival hypertrophy or hyperplasia caused due to the long term use of a drug such as Phenytoin (PHT).[2] Merritt & Putnam introduced PHT as an anti-epileptic drug in 1938.[3] Kimball in the year 1939 discovered that administration of diphenylhydantoin (Phenytoin) on a long term regimen for control of grand mal seizures is often accompanied by enlargement of the soft tissues surrounding the teeth.[4] This condition is frequently referred to as Phenytoin-induced gingival enlargement (PIGE). It is estimated that about 30% to 50% of patients taking PHT develop significant gingival alterations.[1] Other anticonvulsants such as sodium valproate, phenobarbitone, vigabatrin and primidone have also been associated with gingival enlargement but the cases of gingival changes after long term use of these drugs in adult patients have been rarely reported.[5]

CASE REPORT:

A 27-year-old male reported to the Department of Dentistry, Shaheed Hasan Khan Mewati Govt. Medical College, Nuh with the chief complaints of generalized swollen and painful gums in upper and lower jaws since 1 year.

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The patient hesitated in meeting people because of an unaesthetic appearance. Medical history showed that he had epilepsy since the age of 9 years and had tried various types of medical treatments but did not get much benefit. For the last 1 year, he had been put on PHT (Eptoin 100 mg three times a day). The patient did not receive any prior dental treatment. On intra oral examination, the gingival tissues were bead shaped, pale pink in color, enlarged, firm, and fibrotic. Generalized bleeding on probing was present. Oral hygiene was poor. Gingival enlargement was covering almost full part of crown of the teeth in both of upper and lower jaw till 1st molars (excluding maxillary incisors) [Fig.- 1]. Full mouth periodontal charting which includes assessment of probing depth and clinical attachment level was done and it showed false pockets which also confirm with Orthopentograph (OPG) (Fig. 2).

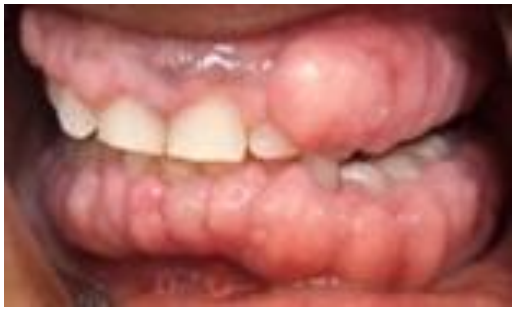

Figure: 1. Fibrotic gingival enlargement in upper and lower arches

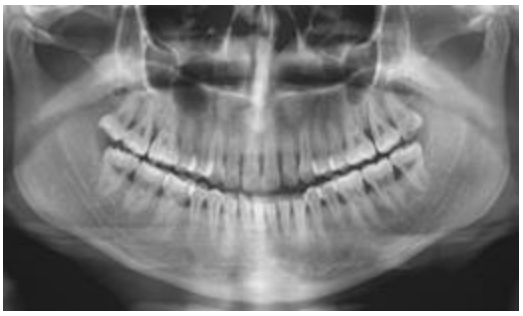

Figure: 2. Orthopentograph showing generalized false pockets

Case management:

After the clinical examination, the patient was referred to the medicine department for opinion, if any form of dental procedures could be conducted and if the patient needed any change in the medication to control the gingival enlargement. The concerned doctor tapered down his drug dose of PHT 100 mg to once in a day for one month along with sodium valporate 400 mg two times a day. The change of drug was well compensated showing no episode of recurrent seizure activity. Complete hemogram showed the values were under normal limits. Hence, the patient was considered fit to undergo any dental procedure under local anesthesia. After taking consent from the patient, complete oral prophylaxis was performed and 0.2% chlorhexidine gluconate mouthwash (10 ml twice a day) was prescribed to the patient. Following the administration of local anesthesia (2% lignocaine with 1:80,000 adrenaline), the pockets on each surface were explored with a periodontal probe

and marked with a pocket marker. Each pocket was marked in several areas to outline its course on each surface. The initial scalloped internal bevel incision was then made starting apical to the points marking the course of the pockets with a number 15 blade including the creation of new interdental papillae. The incision was carried out to a point apical to the alveolar crest. Thinning of the flap was done along with the initial incision in the buccolingual direction up to the mucogingival junction. Care was taken to retain enough amount of attached gingiva after removal of the pocket wall. A crevicular incision was made from bottom of pocket to the bone followed by interdental incision to detach the connective tissue from the bone. The triangular wedge of tissue thus created was removed with the help of curettes [Fig.-3]. Following scaling and root planing, the flaps were positioned on the root-bone junction and co-pack was placed over the surgical site [Fig.-4]. The patient was prescribed chlorhexidine gluconate mouthwash 0.2% twice daily for four weeks and was also advised to avoid vigorous brushing on the surgical site. A course of antibiotics – Amoxicillin 500 mg thrice daily for 5 days and Analgesics (Ibuprofen 400 mg, Paracetamol 325 mg combination) were prescribed thrice daily for 3 days. The same procedure was carried out for both upper and lower arch with a gap of 7 days between the two periodontal surgeries. The 3 months postoperative results were satisfactory with adequate plaque control [Fig.-5].

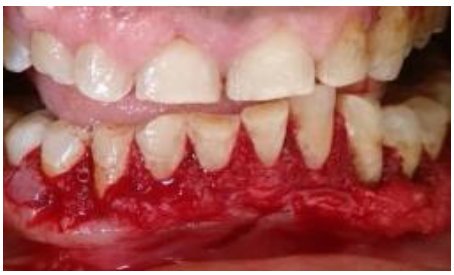

Figure - 3. Immediate postoperative surgical site

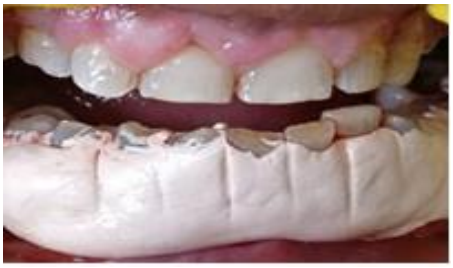

Figure - 4. Co-pack placement done over the surgical site in mandibular segment

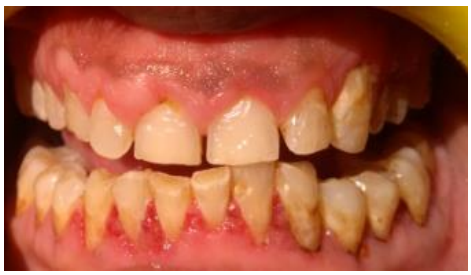

Figure - 5. 3 months postoperative view

Histopathological examination:

The excised tissue was sent for histopathological examination. The microscopic sections of the soft tissue specimen revealed gingival hyperplasia showing hyperplastic epithelium with thin elongated rete pegs penetrating deep into the connective tissue. The

connective tissue showed dense collagen fibers bundles along with blood vessels and chronic inflammatory cells [Fig.-6]. The lesion was diagnosed as fibroepithelial hyperplasia and was suggestive of PHT induced gingival hyperplasia based on clinical and histological evidences.

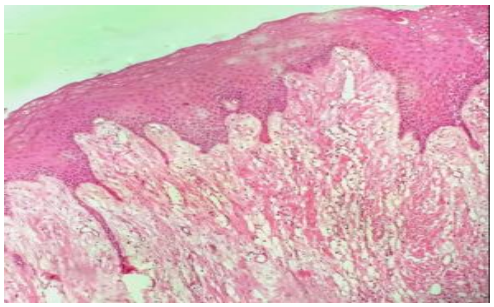

Figure- 6. Histopathology of the gingival tissue exhibiting hyperplasia of the epithelium and elongated rete ridges with underlying dense connective tissue. (H & E, 20x)

DISCUSSION:

PHT therapy was given to the epileptic patients for the treatment of epileptic shock but the common side effect of PHT is the gingival enlargement. The major clinical and microscopic characteristics of gingival enlargement caused by various drugs are similar.[3] PIGE is a soft tissue growth which initially starts as a painless, bead-like, diffuse swelling at the region of the interdental papilla, which later on enlarges and combines to form a nodular form of a swelling. As the swelling progresses to the marginal gingiva it forms a massive tissue fold which sufficiently covers a huge portion of the tooth structure.[6] It does not cause any form of tenderness unless it is secondarily infected. Secondary inflammatory changes not only adds to the size and tenderness of the lesion caused by the drug, but also produce a red or bluish red discoloration, obliterating the lobulated surface demarcations and increasing the tendency to bleed. It has been reported that gingival overgrowth in individuals prescribed with PHT can be minimized with appropriate personal oral hygiene and professional maintenance.[7,8] The presence of the enlargement makes the plaque control and maintenance of the area difficult, which results in secondary inflammatory response which further complicates the overgrowth.[9] A recent study concluded that the CYP2C9 gene polymorphism is responsible for modification of the inflammatory response to PHT.[10] The hypothesis that DIGE is a side effect with a multifactorial etiology is supported by the review of various investigations into its pathogenesis. The inflammatory changes that occur within gingival tissue appears to coordinate the communication between drugs and fibroblasts.[4] PIGE usually develops in susceptible individuals at usually within 1 to 3 months of starting the medication. But the pathogenesis of PIGE is yet unsure.[11] The pharmacokinetics of inducing drugs and

the gingival binding affinity of these drugs are determinants in the pathogenesis of DIGE.[12] Hall conducted a study in which he found sodium valproate as the safest alternative to PHT for the treatment of adult-onset epilepsy.[9] In our case also we found sodium valporate as a suitable alternative to PHT.

CONCLUSION:

PIGE requires appropriate treatment planning. Treating the excessive tissue enlargement alone without considering other factors such as the drug and local factors such as plaque and calculus will not completely benefit the patient. It is very crucial to include drug substitution and control the local inflammatory factors as an initial part of the treatment protocol. However, when this sequence of treatment fails to resolve the problem, surgical intervention can provide favorable patients results.

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