Case Report

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Hereditary Gingival Overgrowth In Epileptic Patient: A Case Report

Abstract:
Gingival overgrowth (GO) is the abnormal enlargement of gingiva which may be caused due to different etiological factors such as inflammation, drug or inherited (hereditary) gingival fibromatosis (HGF). Gingival fibromatosis is a rare and heterogeneous group of disorders that develop as slowly progressive, local or diffuse enlargements within marginal and attached gingiva or interdental papilla. Unlike drug induced gingival overgrowth (DIGO) which usually occurs as a generalized diffuse enlargement, HGF is characterized by a slow, progressive growth of the gingival tissue. Gingival fibromatosis may be familial or idiopathic. Treatments vary according to the type of overgrowth and the extent of disease progression, thus, scaling of teeth is sufficient in mild cases, while in severe cases surgical intervention is required. Risk of recurrence of HGF exists. This article presents case report of gingival hypermatosis with distinctive facies, and diagnosis and treatment of the disease.

Key-words: Gingival Overgrowth, Phenytoin, Gingival fibromatosis, Levetiracetam.

Introduction:

Gingival overgrowth (GO) or gingival enlargement (previously called gingival hyperplasia or gingival hypertrophy) is characterized by enlarged gingival tissue with lobulated appearance that gradually extends along the labial, lingual, and coronal aspects to cover the entire anatomic crown of teeth. It may often associate with pain and bleeding gums, which in advanced cases may cause interference with speech, mastication, and aesthetics.[1]

The American Academy of Periodontology (AAP) defined drug-influenced gingival enlargement as "an overgrowth or increase in size of the gingiva resulting in whole or in part from systemic drug use".[2] 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.[3] These drugs have similar mechanism of action , where they inhibit intracellular calcium ion influx. They act on calcium and sodium ion flux that may prove to why three dissimilar drugs have a common side effect upon a secondary target tissue, such as gingival connective tissue.[4]

Gingival fibromatosis is a rare and heterogeneous group of disorders that develop as slowly progressive, local or diffuse enlargements within marginal and attached gingiva or interdental papilla. In severe cases, the excess tissue may cover the crowns of the teeth, thus causing functional, esthetic, and periodontal problems, such as bone loss and bleeding, due to the presence of pseudopockets and plaque accumulation.

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The diagnosis is mainly made on the basis of the patient's history and clinical features, and on histopathological evaluation of affected gingiva. Early diagnosis is important, mostly to exclude oral malignancy. Differential diagnosis comprises all pathologies in the mouth with excessive gingival overgrowth.[5]

This article presents case report of gingival hypermatosis with distinctive facies, and diagnosis and treatment of the disease.

Case Report:

A 28-year-old male patient reported to the Department of Periodontology in Career PG Institute of Dental Sciences and Hospital, Lucknow, Uttar Pradesh, India, with the chief complaint of swollen gums in both upper and lower front and back teeth region for 1 year. The patient also reported bleeding while brushing and bad breath. Medical history showed that the patient was epileptic since the age of 15 years, and from last 8 months, the patient had been put on Phenytoin (Epsod, 100 mg tds). He had reportedly not received any dental treatment.

On oral examination, generalized fibrotic gingival enlargement was seen in both upper and lower jaw. Gingiva was inflamed and was pinkish red in color with irregular margins. The interdental papillae were scalloped, giving lobulated appearance.(Figure 1)Probing pocket depths ranged from 2 to 10 mm around most of the teeth. Abundant plaque and calculus, and generalized bleeding on probing was evident.

On the basis of medical history and local examination, provisional diagnosis of phenytoin-induced gingival enlargement was made. Blood investigations values were within the normal limit.

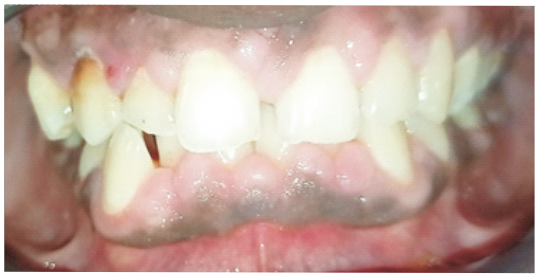

Figure 1: Baseline facial view

Treatment Plan:

With the consent of the patient and his physician, complete oral prophylaxis was performed and 0.2% chlorhexidine mouthwash (10 mL bid for 7 days) was prescribed to the patient. The patient was instructed to maintain good oral hygiene, and proper brushing techniques were explained to him. The patient was advised with the physician consultation for alternate drug regimen. General physician changed the drug to Levetiracetam 500mg twice daily (*Levipil* 500).

At the following visit, surgical intervention, that is, gingivectomy, was performed to eliminate excessive gingival tissue.(Figure 2-4)The sliced tissue was sent for biopsy. 2 months postoperative visit showed good result with complete resolution of enlargement. (Figure 5)

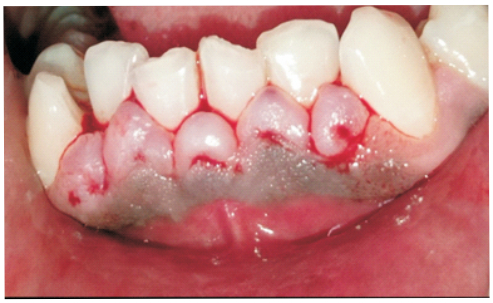

Figure 2: Bleeding Points denotes base of pocket

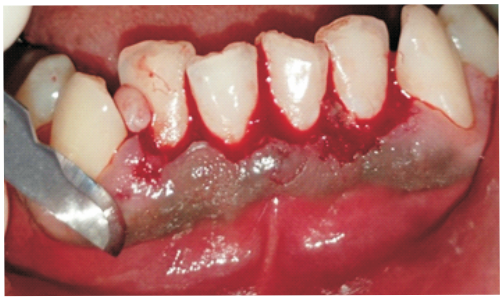

Figure 3: Depigmentation by slicing

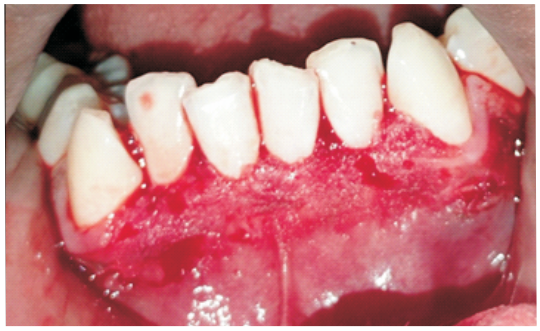

Figure 4: Immediate Post-Operative

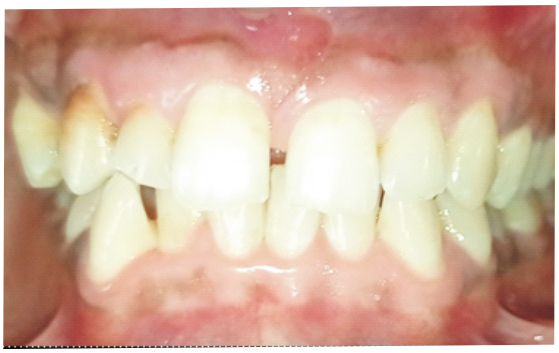

Figure 5: Two Months Post-Operative

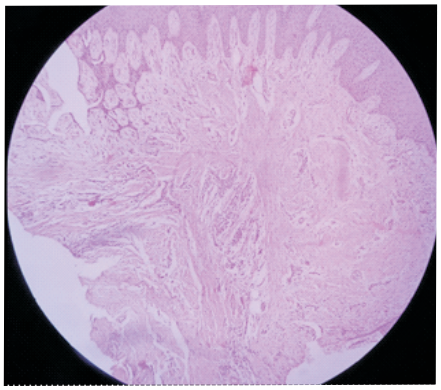

Figure 6: Histological Picture

Microscopic analysis of the histological sections(Figure 6) revealed gingival tissue fragments with numerous and elongated projections (rete pegs) that protruded into the underlying connective tissue. In the underlying lamina propria, there was a dense fibrous connective tissue with an increased amount of collagen fibers, and numerous spindle-shaped fibroblasts. Blood vessels of different calibers were also observed with some congested areas of intense and predominantly mononuclear inflammatory infiltrate.

Discussion:

The most common forms of gingival overgrowth (GO) are those induced by systemic drugs (DIGO)[6]; followed by the inherited HGF or idiopathic IGF conditions[7]. The relationship between the drugs and gingival tissues was influenced by various factors including age, genetic predisposition, and alteration in gingival connective tissue homeostasis, histopathology, ultrastructural factors, inflammatory changes, and action of drugs on growth factors.[8]

The first case of phenytoin-induced gingival enlargement was reported in 1939 by Kimball.[9] Phenytoin is a commonly prescribed medication for the treatment of patients with epilepsy due to its cost and familiarity and is not often substituted by other antiepileptic drugs.[10] Kamali et al. were unable to determine whether concomitant medication of phenytoin with other anticonvulsants could lead to an increase in DIGO incidence during long-term therapy with phenytoin in epileptic patients.[11] Moffitt et al. suggested that other factors should be considered and investigated such as age, plaque control, pharmacokinetic variables, dosage, and duration of drug intake could be involved in the mechanism of gingival overgrowth.[12]

Costa et al. have reported that appropriate plaque control and early detection of periodontal disease is difficult, and that care and periodontal disease tend to worsen easily in patients with refractory epilepsy.[13] Early monitoring of the gingival tissue following the observation of gingival hyperplasia in patients administered anticonvulsant agents is required to prevent aggravation of the condition.[14] For an effective control of this problem, proper treatment protocol would be necessary, which includes drug substitution and control of local inflammatory factors. However, surgical intervention is required when this sequence of treatment fails to resolve the problem.

Gingival Fibromatosis is a condition characterized by the pathological growth of gingival tissue. It is also described as “gingival enlargement”, which comprises gingival hyperplasia and hypertrophy. Gingival Fibromatosis can present, as drug-induced gingival overgrowth (DIGO) characterized by pathological, diffuse or local growth of gingiva.[15] Unlike DIGO which usually occurs as a generalized diffuse enlargement, HGF is characterized by a slow, progressive growth of the gingival tissue. Gingival fibromatosis may be familial or idiopathic[6]. Autosomal dominant forms of gingival fibromatosis, which are usually non-syndromic, have been genetically linked to the

chromosome 2p21-p22 and 5q13-q22.[16]

When hereditary gingival fibromatosis (HGF) is suspected, the first step is to rule out medication or a medical or systemic history that could explain the gingival enlargement. A similar history in other members of the family points to a hereditary etiology but is not an exclusive diagnostic criterion for HGF, so along with anatomy of growth, medical or systemic history, hereditary etiology, it is advised to histopathologic analysis for establishing this diagnosis.

In this case report, the histological images confirmed the features that characterize HGF. Nowadays, owing to high genetic heterogeneity, genetic testing to confirm the diagnosis is not justified.[17] HGF cannot be definitively treated but may be managed with varying and unpredictable degrees of success. The treatment of choice continues to be internal or external bevel gingivectomy in association with gingivoplasty, an apically positioned flap, electrocautery, and CO_2 laser.

References:

1. Goel S, Chhina S, Arora SA. Drug-induced gingival enlargement: A case report. *Journal ofDental Lasers*. 2019 Jul 1;13(2):49.
2. American Academy of Periodontology. Glossary of Periodontal Terms. 4th ed. Chicago, IL: *American Academy of Periodontology*; 2001.
3. Gurgel BC, Morais CR, Rocha-Neto PC, Dantas EM, Pinto LP, Costa AD. Phenytoin-inducedgingival overgrowth management with periodontal treatment. *Brazilian dental journal.* 2015Jan;26:39-43.
4. Bharti V, Bansal C. Drug-induced gingival overgrowth: The nemesis of gingiva unravelled. *Journal of Indian Society of Periodontology*. 2013 Mar;17(2):182.
5. Gawron K, Bartyzel KL, Potempa J and Gajewska M. Gingival fibromatosis: clinical, molecular and therapeutic issues. *Orphanet Journal of Rare Diseases* (2016) 11:9
6. Vipin B, Chhaya B Drug-induced gingival over growth: The nemesis of gingiva unraveled. *J Indian Soc Periodontol.* 2013; 17: 182-187.
7. Bhowmick SK, Gidvani VK, Retting KR. Hereditary gingival fibromatosis and growth retardation. *EndocrPract.* 2001;7: 383-387
8. Seymour RA, Thomason JM, Ellis JS. The pathogenesis of drug-induced gingival overgrowth. *J Clin Periodontol* .1996;23:165-75.
9. Kimball OP. The treatment of epilepsy with sodium diphenyl hydantoinate. *JAMA*1939;112:1244-5.
10. Majola MP, McFadyen ML, Connolly C, Nair YP, Govender M, Laher MH. Factors influencing phenytoin-induced gingival enlargement. *J Clin Periodontol2000*;27:506-512.
11. Kamali F, McLaughlin WS, Ball DE, Seymour RA. The effect of multiple anticonvulsant therapy on the expression of phenytoin-induced gingival overgrowth. *J ClinPeriodontol.* 1999;26:802-805.
12. Moffitt ML, Bencivenni D, Cohen RE. Drug-induced gingival enlargement: an overview.*Compend Contin Educ Dent*. 2013;34:330-336.
13. Costa AL, Yasuda CL, Shibasaki W, Nahás-Scocate AC, de Freitas CF, Carvalho PE et al. The association between periodontal disease and seizure severity in refractory epilepsy patients. *Seizure.* 2014 Mar 1;23(3):227-30.
14. Hatahira H, Abe J, Hane Y, Matsui T, Sasaoka S, Motooka Y et al. Drug-induced gingival hyperplasia: a retrospective study using spontaneous reporting system databases. *Journal of pharmaceutical health care and sciences.* 2017 Dec;3(1):1-1.
15. Gawron K, Łazarz-Bartyzel K, Potempa J, Chomyszyn-Gajewska M. Gingival fibromatosis: clinical, molecular and therapeutic issues. *Orphanet journal of rare diseases.* 2016 Dec;11(1):1-4.
16. Bansal A, Narang S, Sowmya K, Sehgal N. Treatment and two yearfollow up of a patient with hereditary gingival fibromatosis. *J Indian SocPeriodontol.*2011; 15: 406-409
17. Ye X, Shi L, Yin W, Meng L, Wang QK, Bian Z. Further evidence of genetic heterogeneity segregating with hereditary gingival fibromatosis. *J Clin Periodontol.*2009;36:627-33.