



Libyan International Medical University
Faculty of Basic Medical Science



2017 - 2018

Gingival Enlargement

Submitted by: Hajer Othman / 1406 / 2nd year dentistry student

Supervisor: Dr. Mahmoud Elmansoury

Date of Submission: 19.04.2018

Abstract :

Gingival enlargement is one of the frequent features of gingival diseases. However due to their varied presentations, the diagnosis of these entities becomes challenging for the clinician. Therefore, the aim of this article is to first define gingival enlargement, and then discuss its classification, causes, and management.

Introduction :

Gingival enlargement, (also termed gingival overgrowth, hypertrophic gingivitis, gingival hyperplasia, or gingival hypertrophy, and sometimes abbreviated to GO), is an increase in the size of the gingiva (gums). It is a common feature of gingival disease.[1] Gingival enlargement can be caused by a number of factors, including inflammatory conditions and the side effects of certain medications. The treatment is based on the cause.[1] A closely related term is epulis, denoting a localized tumor (i.e. lump) on the gingiva.

The terms gingival hyperplasia and gingival hypertrophy have been used to describe this topic in the past.[1] These are not precise descriptions of gingival enlargement because these terms are strictly histologic diagnoses, and such diagnoses require microscopic analysis of a tissue sample. Hyperplasia refers to an increased number of cells, and hypertrophy refers to an increase in the size of individual cells.[2] As these identifications obviously cannot be performed with a clinical examination and evaluation of the tissue,[3] the term gingival enlargement is more properly applied. Gingival enlargement has been classified according to cause into 5 general groups:[1] Inflammatory enlargement, Drug induced enlargement, Enlargement associated with systemic diseases or conditions, Neoplastic enlargement, False enlargement.

Discussion :

Starting with its aetiology, gingival enlargement may be caused by a multitude of causes. The most common is chronic inflammatory gingival enlargement, when the gingivae are soft and discolored. This is caused by tissue edema and infective cellular infiltration caused by prolonged exposure to bacterial plaque, and is treated with conventional periodontal treatment, such as scaling and root planing.[1] Gingivitis and gingival enlargement are often seen in mouth breathers, [4] as a result of irritation brought on by surface dehydration, but the manner in which it is caused has not been demonstrated.[1] The accumulation and retention of plaque is the chief cause of inflammatory gingival enlargement. Risk factors include poor oral hygiene,[5] as well as physical irritation of the gingiva by improper restorative and orthodontic appliances.[1]

Another type of gingival enlargement is sometimes termed "drug induced gingival enlargement" or "drug influenced gingival enlargement",[6] abbreviated to "DIGO".[7] Gingival enlargement may also be associated with the administration of three different classes of drugs, all producing a similar response:[8] Gingival overgrowth is a common side effect of phenytoin, termed "Phenytoin-induced gingival overgrowth" (PIGO).[9] Anticonvulsants (such as phenytoin, phenobarbi-

tal, lamotrigine, vigabatrin, ethosuximide, topiramate and primidone NOT common for valproate)[10], Calcium channel blockers (antihypertensives such as nifedipine, amlodipine, and verapamil). The dihydropyridine derivative isradipidine can replace nifedipine and does not induce gingival overgrowth.[10], and, finally, cyclosporine, an immunosuppressant.[10]

Of all cases of DIGO, about 50% are attributed to phenytoin, 30% to cyclosporins and the remaining 10-20% to calcium channel blockers. Furthermore, drug-induced enlargement has been associated with a patient's genetic predisposition,[11] and its association with inflammation is debated. Some investigators assert that underlying inflammation is necessary for the development of drug-induced enlargement,[12] while others purport that the existing enlargement induced by the drug effect compounds plaque retention, thus furthering the tissue response.[13] Careful attention to oral hygiene may reduce the severity of gingival hyperplasia.[14] In most cases, discontinuing the culprit drug resolves the hyperplasia.[14]

Enlargement may also be associated with systemic factors that may include gingival enlargement, some that are related to conditions and others that are related to disease:[15] Conditioned enlargement, pregnancy, puberty, vitamin C deficiency, nonspecific, such as a pyogenic granuloma, Systemic disease causing enlargement, leukemia, granulomatous diseases, such as granulomatosis with polyangiitis, sarcoidosis, or orofacial granulomatosis.[16], neoplasm, benign neoplasms, such as fibromas, papillomas and giant cell granulomas, malignant neoplasms, such as a carcinoma or malignant melanoma, false gingival enlargements, such as when there is an underlying bony or dental tissue lesion.

As for the management, the first line management of gingival overgrowth is improved oral hygiene, ensuring that the irritative plaque is removed from around the necks of the teeth and gums. Situations in which the chronic inflammatory gingival enlargement include significant fibrotic components that do not respond to and undergo shrinkage when exposed to scaling and root planing are treated with surgical removal of the excess tissue, most often with a procedure known as gingivectomy.[1]

In DIGO, improved oral hygiene and plaque control is still important to help reduce any inflammatory component that may be contributing to the overgrowth. Reversing and preventing gingival enlargement caused by drugs is as easy as ceasing drug therapy or substituting to another drug. However, this is not always an option; in such a situation, alternative drug therapy may be employed, if possible, to avoid this deleterious side effect. In the case of immunosuppression, tacrolimus is an available alternative which results in much less severe gingival overgrowth than cyclosporin, but is similarly as nephrotoxic.[17] The dihydropyridine derivative isradipidine can replace nifedipine for some uses of calcium channel blocking and does not induce gingival overgrowth.[18]

Conclusion:

Gingival enlargement or gingival overgrowth, a common trait of gingival disease, is characterized by an increase in the size of gingiva. Pertinent management depends on precisely diagnosing the origin of enlargement.

References:

1. Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. (2012). *Carranza's clinical periodontology* (11th ed.). St. Louis, Mo.: Elsevier/Saunders. pp. 84–96. ISBN 978-1-4377-0416-7.
2. Merriam-Webster's Medical Desk Dictionary, 2002, ISBN 1-4018-1188-4, page 367-368.
3. Oral Pathology Lecture Series Notes, New Jersey Dental School, 2004-2005, page 24.
4. Lite, Theodore; Dominic J. DiMaio; Louis R. Burman (1955). "Gingival pathosis in mouth breathers: A clinical and histopathologic study and a method of treatment". *Oral Surgery, Oral Medicine, Oral Pathology*. 8 (4): 382–391. doi:10.1016/0030-4220(55)90106-7. ISSN 0030-4220.
5. Hirschfield, I (1932). "Hypertrophic gingivitis; its clinical aspect". *JADA* (19): 799.
6. Lindhe J, Lang NP, Karring T, eds. (2008). *Clinical periodontology and implant dentistry* (5th ed.). Oxford: Blackwell Munksgaard. p. 641. ISBN 9781405160995.
7. Subramani, T; Rathnavelu, V; Yeap, SK; Alitheen, NB (Feb 2013). "Influence of mast cells in drug-induced gingival overgrowth". *Mediators of Inflammation*. 2013: 275172. doi: 10.1155/2013/275172. PMC 3569901 Freely accessible. PMID 23431239.
8. Butler RT, Kalkwarf KL (1987). "Drug-induced gingival hyperplasia: phenytoin, cyclosporine, and nifedipine". *JADA* (114): 56.
9. Arya, R; Gulati, S (March 2012). "Phenytoin-induced gingival overgrowth". *Acta neurologica Scandinavica*. 125 (3): 149–55. doi:10.1111/j.1600-0404.2011.01535.x. PMID 21651505.
10. Bologna, Jean L. (2007). *Dermatology*. St. Louis: Mosby. ISBN 1-4160-2999-0.
11. Hassell, TM; Burtner, A. Paul; McNeal, Donald; Smith, Robert G. (1994). "Hypertrophic Oral problems and genetic aspects of individuals with epilepsy". *Periodontology* 2000. 6 (1): 68–78. doi:10.1111/j.1600-0757.1994.tb00027.x.
12. Ciancio, SG (1972). "Gingival hyperplasia and diphenylhydantoin". *J Perio* (43): 411.
13. Carranza's Clinical Periodontology, 9th Ed. W.B. Saunders 1996 ISBN 0-7216-8331-2, page 282.

14. Brian K. Alldredge; et al., eds. (2013). *Applied therapeutics : the clinical use of drugs* (10th ed.). Baltimore: Wolters Kluwer Health/Lippincott Williams & Wilkins. p. 1403. ISBN 978-1609137137.
15. Carranza's *Clinical Periodontology*, 9th Ed. W.B. Saunders 1996 ISBN 0-7216-8331-2, page 285.
16. Leão, JC; Hodgson, T; Scully, C; Porter, S (Nov 15, 2004). "Review article: orofacial granulomatosis". *Alimentary pharmacology & therapeutics*. 20 (10): 1019–27. doi:10.1111/j.1365-2036.2004.02205.x. PMID 15569103.
17. Spencer, CM; Goa, KL; Gillis, JC (1997). "Tacrolimus: an update of its pharmacology and drug efficacy in the management of organ transplantation". *Drugs*. 54 (6): 925–75. doi: 10.2165/00003495-199754060-00009. PMID 9421697.
18. Westbrook, P (1997). "Regression of nifedipine-induced gingival hyperplasia following switch to a same class calcium channel blocker, isradipine". *J Perio* (68): 645.