

## Gingival Overgrowth: An Enigma to Periodontists

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### ABSTRACT

Gingival overgrowth which is an abnormal growth of the periodontal tissue is mainly associated with dental plaque-related inflammation and drug therapy. Its true incidence in the general population is unknown. Gingival enlargement produces aesthetic changes, pain, gingival bleeding and periodontal disorders. The three classes of drugs namely, anti epileptic, immune-suppressive, produce important changes in fibroblast function, which induce an increase in the extracellular matrix of the gingival connective tissue. In the majority of those patients for whom dosage reduction, or drug discontinuation or substitution is not possible, and for whom prophylactic measures have failed, surgical excision of gingival tissue remains the only treatment of choice. Brunet et al. [1]

**Key words:** Gingival enlargement, gingival hyperplasia, gingival overgrowth, amlodipine, phenytoin, drug induced gingival enlargement.

### INTRODUCTION

An adverse effect caused due to an intake of three types of drugs namely phenytoin, an antiepileptic; cyclosporine A, an immunosuppressant; and calcium channel blockers, such as dihydropyridines (i.e., nifedipine, nitrendipine, and felodipine), diltiazem and Verapamil. The first report of phenytoin induced gingival overgrowth was reported by Kimball in 1939, many clinical and investigative studies have been carried out to determine the etiopathogenesis of this disorder. Even though these studies gave various pathogenetic data, it is still unknown why drugs with such different pharmacological actions induce similar gingival changes. Also the blood drug levels and/or duration of drug intake and the severity of growth,

sex predilection, and effect of local inflammation also affected the severity of the overgrowth.

The capacity of the host to deal metabolically with chronically administered drugs, the responsiveness of gingival tissue to the drugs, and the pre existing gingival condition may differ among different individuals. Moreover, calcium channel blockers are mainly prescribed for post-middle-aged patients to control hypertension, cardiac insufficiencies or myocardial events, whereas phenytoin and cyclosporine A (CsA) are prescribed for a wide range of patients due to their wide spectrum of efficacy. These make it difficult to evaluate the etiology of drug induced gingival overgrowth caused by phenytoin, CsA, and calcium channel blockers and to compare the factors involved. [2]

The influence of plaque on the induction of gingival enlargements by drugs in humans has not been fully elucidated; however, it does appear that the severity of the lesion is influenced by the oral hygiene of the patient. [3]

### Characteristics of drug influenced gingival enlargement

- Variation in inter and intra patient pattern.
- Predilection for anterior gingival.
- Higher prevalence in children.
- Onset within 3 months.
- Change in gingival contour leading to modification of gingival size.
- Enlargement first observed at the interdental papilla.
- Change in gingival color.
- Increased gingival exudates.
- Bleeding upon provocation.

- Found in gingiva with or without bone loss but is not associated with attachment loss.
- Pronounced inflammatory response of gingiva in relation to the plaque present.
- Reductions in dental plaque can limit the severity of the lesion.
- Must be using phenytoin, cyclosporine A or certain calcium channel blockers; the plasma concentrations to induce the lesion have not been clearly defined in humans. [4]

The current term is gingival overgrowth. It had been named gingival hyperplasia/hypertrophy and gingival enlargement. The term hyperplasia is only relevant with the increased number of cells and hypertrophy coincides with increased size.

#### **Classification based on the location and distribution**

- "Localized" - Limited to the gingiva in relation to a single tooth or group of teeth.
- "Generalized"- involving the gingival throughout the mouth.
- "Marginal" - Involvement limited to the gingival margin.
- "Diffuse" - involving the gingival margin and the remainder of the gingival mucosa up to the mucobuccal fold and for a varying distance along the lingual surface.

#### **Classification based on the underlying histopathological changes and etiology**

- Inflammatory
  - A. Chronic Inflammatory Gingival Enlargement
    - 1. Generalized or localized
    - 2. Discrete (Tumor-like)
  - B. Acute Inflammatory Gingival Enlargement (Gingival abscess)
- Non Inflammatory hyperplastic gingival enlargement
  - A. Marginal
  - B. Diffuse
- Combined enlargement
- Conditioned gingival enlargement
  - A. Hormonal

#### **1. Gingival Enlargement of Pregnancy**

##### **2. Gingival Enlargement of Puberty**

##### **B. Leukemic Gingival Enlargement**

##### **C. Gingival Enlargement Associated with Vitamin C Deficiency**

- Neoplasms
- Developmental gingival enlargement [5]

#### **Classification based on scoring the extent of the overgrowth:**

- Grade 0: No signs of gingival enlargement.
- Grade I: Enlargement confined to interdental papilla.
- Grade II: Enlargement involves papilla and marginal gingiva.
- Grade III: Enlargement covers three quarters or more of the crown.

#### **Etiopathogenesis of Gingival Enlargement**

- It was observed that drug induced gingival enlargement occurred in mostly all the individuals who were on the above mentioned three classes of drugs. The overgrowth regressed significantly after cessation of the drug. This concluded that the overgrowth depends upon the blood drug concentration.
- The order of induced gingival overgrowth was phenytoin < nifedipine < Cephalosporins.
- Phenytoin decreases the collagen degradation as a result of the production of an inactive fibroblastic collagenase. [6]
- Males are more susceptible than the female counterparts. This is mainly due to the female hormone progesterone. Progesterone decreases glycosaminoglycan synthesis by human gingival fibroblasts in vitro.
- Younger age predilection was observed for nifedipine and phenytoin drug intake but not for cephalosporin intake.
- Dental plaque as observed in many studies is the primary causative agent and factor for any periodontal disease.

However in drug induced gingival overgrowth, it is an aggravating factor rather than a causative factor. This is because it is difficult to assess the state of oral hygiene prior to drug administration and that overgrowth often produces secondary gingival inflammation. Also if the individual is on a thorough oral hygiene protocol while taking phenytoin, the overgrowth can be minimal but not totally absent. The overgrowth is severe in individuals with poor plaque control

- Epidermal growth factor (EGF) was involved in phenytoin induced gingival overgrowth. It was found that phenytoin increased the level of EGF receptors in human gingival fibroblasts, resulting in increased cellular responsiveness to EGF thereby can stimulating cellular DNA synthesis.
- Phenytoin can stimulate testosterone metabolism, increasing the level of 5 $\alpha$  dihydrotestosterone which is a stimulator of fibroblast growth in the overgrown tissue. [2]

#### **Clinical features of chronic inflammatory enlargement**

- It produces a life preserver– shaped bulge around the involved teeth which can increase in size until it covers part of the crowns.
- The enlargement may be localized or generalized progressing slowly and painlessly, unless it is complicated by acute infection or trauma.
- It presents like a slight ballooning of the interdental papilla and the marginal gingival.
- It can also occur as a discrete sessile or pedunculated mass resembling a tumor which may be interproximal or on the marginal or attached gingiva.
- They may also undergo spontaneous reduction in size, followed by exacerbation and continued enlargement.

- Painful ulcerations sometimes occur in the fold between the mass and the adjacent gingiva.

#### **Clinical features of drug induced gingival overgrowth:**

- The enlargement is generalized but is severe in the maxillary and mandibular anteriors.
- The exhibited growth starts as a painless, beadlike enlargement of the interdental papilla extending to the facial and lingual gingival margins. After time a union of the marginal and papillary enlargements occur and may develop into a massive tissue fold covering a considerable portion of the tooth crowns.
- This might interfere with occlusion.
- When uncomplicated by inflammation, the lesion is mulberry shaped, firm, and pale pink, and resilient, with a minutely lobulated surface and does not bleed on provocation.
- The enlargement projects from beneath the gingival margin, from which it is separated by a linear groove.
- Plaque control is difficult leading to a secondary inflammation complicating this which results to combination of the increase in size caused by the drug and the complicating inflammation caused by bacteria.
- This produces a red or bluish red discoloration, obliterates the lobulated surface demarcations and increased bleeding tendency.
- It is present in the areas where teeth are present but also may exhibit in regions where there is absence of teeth. [7,8]

#### **Clinical features of Idiopathic gingival overgrowth:**

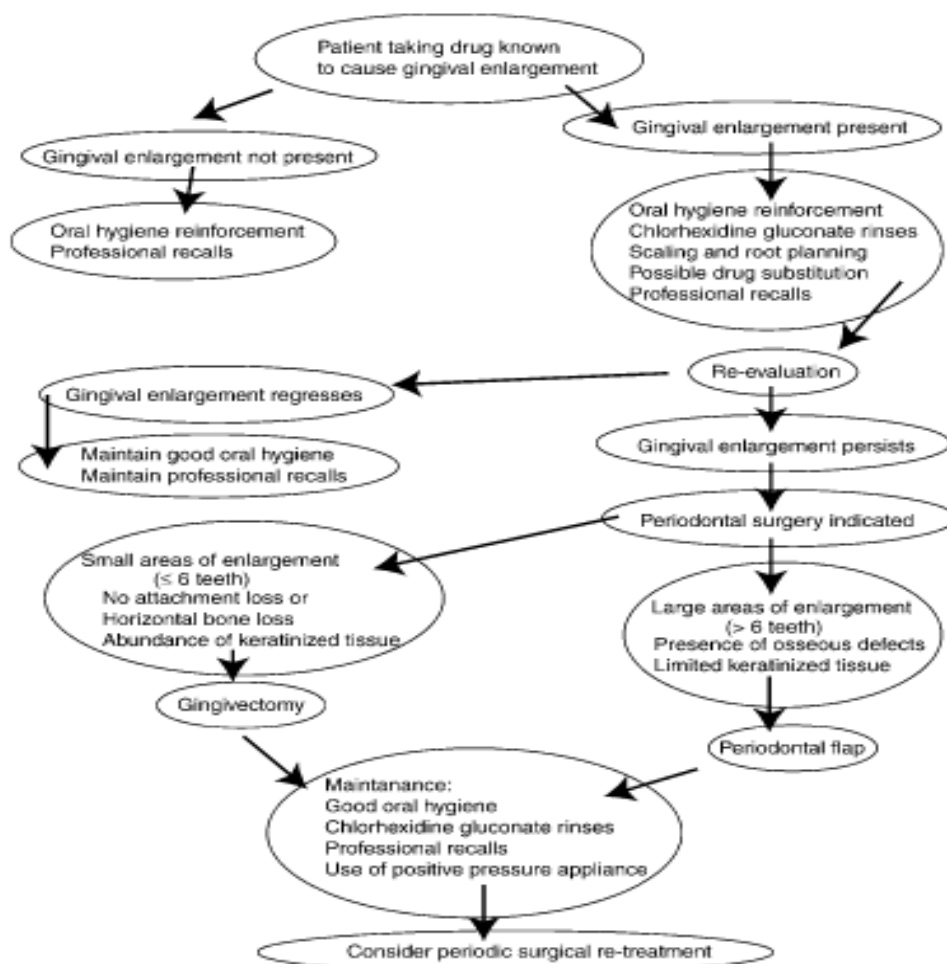
- The enlargement affects the attached gingiva, gingival margin and interdental papillae, in contrast to phenytoin-induced overgrowth, which is only confined to the gingival margin and interdental papillae

- The facial and lingual surfaces of the mandible and maxilla are generally affected, but it is limited to either jaw.
- The enlarged gingiva is pink, firm, and leathery in consistency and has a characteristic minutely pebbled surface.
- In severe cases, the teeth are almost completely covered with the enlargement and it projects into the oral

vestibule. The jaws appear much distorted because of the bulbous enlargement of the gingiva. Secondary inflammatory changes are also seen.

### Treatment of Gingival overgrowth:

The following chart is helpful for decision making in the treatment of gingival overgrowth which is drug induced.



- The first step in the treatment plan for gingival overgrowth is thorough plaque control.
- It should be professionally taken care by the dentist and also at home with various plaque control measures.
- However with the cessation and substitution of the drug and the necessary scaling and root planning, if the overgrowth still continues, it may require surgical intervention.

- Gingivectomy incisions may present with the advantages of technique simplicity and quickness but, unlike the periodontal flap, it will not allow for osseous recon touring and may sacrifice keratinized tissue.
- Also, it results in healing by secondary intention, which causes discomfort and an increased chance of postoperative bleeding.
- It is the clinicians' decision to choose between the two: Gingivectomy or Flap

procedure taking into consideration of the presence of Periodontitis, osseous defects and the availability of keratinized tissue and also of course the esthetics.

#### **Maintenance:**

- There are cases with recurrence seen even in surgically treated individuals.
- Ways to reduce recurrences are meticulous homecare, Chlorhexidine mouth rinses, regular scaling and root planning and bite guard worn at night. [9]

#### **CONCLUSIONS**

Gingival overgrowth may present as an unaesthetic sight to the patient and also to others. It is an absolute enigma to a clinician to diagnose the etiology of the overgrowth. If the enlargement is drug induced, drug substitution should be considered with the plaque control measures. Non surgical methods should be of importance in the early stages of treatment. Failure of the non surgical methods can lead to the surgical intervention of gingivectomies or flap procedures. The success of the treatment of gingival overgrowth not only alone lies in the hands of a periodontist but also in the hands of the patient!

#### **REFERENCES**

1. Drug Safety 1996 Sep; 15 (3): 219-231
2. Seiji Nishikawa, Toshihiko Nagata, Ichijiro Morisaki, Takarni Oka and Hiroshi Ishida. A Review of Drug-Induced Gingival Overgrowth. J Periodontol 1996: 463-471
3. Hassell & Hefti. Drug induced gingival overgrowth: Old problem, New problem Crit Rev Oral Biol Med. 1991; 2(1):103-37.
4. Mariotti A. Dental plaque induced gingival diseases. Ann Periodontol. 1999 Dec;4(1):7-19
5. Irving Glickman. A Basic Classification of "Gingival Enlargement". J Periodontol 1950; 131-139.
6. Hassell TM: Evidence for production of an inactive collagenase by fibroblasts from phenytoin-enlarged human gingiva, J Oral Pathol 11:310, 1982
7. Dallas BM: Hyperplasia of the oral mucosa in an edentulous epileptic. New Zeal Dent J 59:54, 1963.
8. Dreyer WP, Thomas CJ: DPH-induced hyperplasia of the masticatory mucosa in an edentulous epileptic patient, Oral Surg 1945:701, 1978.
9. Paulom. Camargo, Philip R.Melnick, Flavia Q. M. Pirih, Rodrigo Lagos & Henry H. Takei. Periodontology 2000, Vol. 27, 2001, 131-138

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