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Treatment of Gingival Enlargement

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Abstract

Gingival enlargement or overgrowth is a common disease of gingiva. The causative factors may range from inflammation due to local factors to conditioned enlargement and neoplastic enlargements. They commonly present as bulbous interdental gingival, diffuse swelling of gingival. Due to the unaesthetic appearance of the overgrown gingiva, treatment becomes inevitable. This results in excision of overgrowth known as gingivectomy. The first gingivectomy procedure was explained by Robicsek in 1884 and later by Zentler (1918). Grant (1979) defined gingivectomy as excision of soft tissue wall of pathologic periodontal pocket. Gingivectomy procedures can be done by means of scalpel, laser, electrosurgery and chemosurgery. The ultimate result remains the same indifferent of the method used. However the amount of remaining keratinized gingival and esthetic appearance is of supreme importance.

Keywords: gingival enlargement, gingival overgrowth, gingival hyperplasia, gingivectomy, gingival diseases, anticonvulsants, abscess

1. Introduction

Gingival enlargement is a common clinical problem, usually associated with specific conditions. This condition finds a unique place in literature, because it has been associated with a variety of local and systemic factors. Enlargement of any part, tissue or organ in the body may be attributable to one or more of the following pathological processes [1]:

Cellular hypertrophy: defined as an increase in the size of a part due to an increase in the size of the individual cells comprising that part.

Cellular hyperplasia: increase in size due to an increase in the absolute number of cells, though cell size is not altered.

Fibrosis: an accumulation of collagenous connective tissue which is classically characterized by relative acellularity.

Edema: it is nothing more than the presence of abnormally large amounts of fluid in the intercellular spaces.

The events leading to gingival enlargement are complex.

2. Classification

1. According to etiologic factors and pathologic changes [2]:

- Inflammatory enlargements:

- Chronic
- Acute
- Drug induced enlargements:
 - Anticonvulsants
 - Antihypertensive calcium antagonists
 - Immunosuppressant
- Idiopathic enlargement
- Enlargements associated with systemic diseases:

Conditioned enlargements

- Pregnancy
- Puberty
- Vitamin C deficiency
- Plasma cell gingivitis
- Non-specific conditioned enlargement (pyogenic granuloma)

Systemic diseases causing gingival enlargement:

- Leukemia
- Granulomatous diseases (Wegener's granulomatosis, sarcoidosis, etc.)
- Neoplastic enlargements: (gingival tumors)
 - Benign tumors
 - Malignant tumors
- False enlargements

2. According to location and distribution:

- Localized: limited to the gingiva of a single or group of teeth.
- Generalized: involving gingiva throughout the mouth.
- Marginal: confined to the gingival margin.
- Papillary: confined to the interdental papilla.
- Diffuse: involving marginal and attached gingiva and papilla.
- Discrete: an isolated sessile or pedunculated or tumor like enlargement.

Scoring of gingival enlargement:

- 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.

3. Clinical features

3.1 Inflammatory enlargement

It may be chronic or acute. This usually results from accumulation of local deposits. Factors resulting in plaque accumulation predisposes to inflammatory enlargement. Chronic inflammatory enlargement originates as slight ballooning of interdental papilla and marginal gingival. A life preserver shaped bulge appears around the involved teeth. This can increase in size until it covers the crown. It is usually painless until trauma or acute infection is superimposed [2].

Inflammatory enlargement may be localized or generalized. Localized enlargement may appear as tumor like mass or nodule, sessile or pedunculated. It may involve interdental papilla, marginal gingival or attached gingiva. They may undergo spontaneous reduction in size, followed by exacerbation and continued enlargement. Painful ulceration sometimes occurs in the fold between the mass and the adjacent gingiva [2]. Chronic inflammatory enlargement may also occur because of presence of mouth breathing habits. Anterior region predominantly papilla is involved. The mouth breathing habit results in dryness of the mucosa. There is a clear demarcation between normal and involved gingival [2] (**Figure 1**).

3.2 Acute inflammatory enlargement

Acute form of gingival enlargement includes abscesses of the periodontium. They results in a localized painful area of purulent material which needs to be drained. Lindhe et al. [3] classified as (a) periodontitis-related abscess, infection caused by the bacteria present at the subgingival biofilm in a deepened periodontal pocket, (b) non-periodontitis-related abscess, infection caused by the bacteria originating from another local source, such as a foreign body impaction or from alterations in the integrity of the root leading to bacteria colonization. Meng [4] classified as gingival abscesses (in previously healthy sites and caused by impaction of foreign bodies), periodontal abscesses (either acute or chronic, in relation to a periodontal pocket), and pericoronal abscesses (at incompletely erupted teeth). The gingival abscess involves the marginal gingival and interdental tissues. The periodontal abscess is an acute destructive process in the periodontium, resulting in the localized collection of pus, communicating with the oral cavity through gingival sulcus or other periodontal sites and not arising from tooth pulp. The pericoronal abscess is associated with the crown of a partially erupted tooth [4, 5].

Periodontal abscess (**Figure 2**) formation may occur in the following ways [6]:

1. Extension of infection from a periodontal pocket deeply into the supporting periodontal tissues, and localization of the suppurative inflammatory process along the lateral aspect of the root.
2. Lateral extension of inflammation from the inner surface of a periodontal pocket into the connective tissue of the pocket wall. Localization of the abscess results when drainage into the pocket space is impaired.
3. Formation in a pocket with a tortuous course around the root. A periodontal abscess may form in the cul-de-sac, the deep end of which is shut off from the surface.
4. Incomplete removal of calculus during treatment of a periodontal pocket. The gingival wall shrinks, occluding the pocket orifice, and a periodontal abscess occurs in the sealed-off portion of the pocket.



Figure 1.
Chronic inflammatory enlargement.

3.3 Drug-induced gingival enlargement

The growth starts as painless bead like enlargement in the papillary region which extends on the facial and lingual region. As the overgrowth increases the massive fold of tissue can be observed covering considerable or entire portion of crown. This will result in one or more problems like difficulty in oral hygiene maintenance and mastication, may alter tooth eruption, interference of speech and esthetic issues [7, 8].

The clinical appearance reveals firm and fibrotic component unless superimposed by secondary infection because of lack of oral hygiene and biofilm accumulation. These secondary changes include change in color of gingiva, increased bleeding tendency and superimposed inflammation obliterates the demarcation of the lobules [6].

Phenytoin induced gingival overgrowth (**Figure 3**) is characterized by granular or pebbly surface with enlargement of interdental papilla to an extent they result in pseudoclefts. The growth diminishes as they reach to the mucogingival junction but continue to grow in coronal direction resulting in partial or complete obscure of teeth [9]. The systemic administration of phenytoin accelerates the healing of gingival wounds in nonepileptic humans and increases the tensile strength of healing abdominal wounds in rats [10]. Cyclosporin induced enlargement appears to be more prominent on the labial surface, hyperemic, soft, friable and has high bleeding tendency. Calcium channel blocker (**Figure 4**) affects papillary region initially resulting into a lobular and nodular morphology extending to attached and marginal gingival. The inflammatory changes are associated leading to poor plaque control and esthetic concerns [11]. Gingival enlargement is greater in patients



Figure 2.
Periodontal abscess.



Figure 3.
Phenytoin induced gingival enlargement.

who are medicated with both cyclosporine and calcium channel blockers [12]. The microscopic finding of many plasma cells plus the presence of an abundant amorphous extracellular substance has suggested that the enlargement is a hypersensitivity response to the cyclosporine [13].

The probable mechanism include role of fibroblasts, inflammatory cytokines and matrix metalloproteinases (MMP). It has been seen that not all patients treated with these drugs show alteration in size of gingival rather some have very pronounced effect. It has been hypothesized that these individuals have fibroblasts with an abnormal susceptibility to the drug. Fibroblasts from overgrown gingiva in phenytoin-treated patients are characterized by elevated levels of protein synthesis, most of which is collagen. The susceptibility to enlargement is governed by presence of fibroblast subsets which are reactive to these medications [14].

A synergistic enhancement of collagenous protein synthesis by human gingival fibroblasts was found when these cells were simultaneously exposed to nifedipine and interleukin-1 β (IL-1 β), a proinflammatory cytokine that is elevated in inflamed gingival tissues. In addition to IL-1 β , IL-6 may play a role in the fibrogenic responses of the gingiva to these medications. A reported histologic feature of cyclosporin-induced gingival lesions is a elevation in the expression of IL-6 by cells within the gingival connective tissue. IL-6 appears to target connective tissue cells such as fibroblasts both by enhancing proliferation and by exerting a positive regulation on collagen and glycosaminoglycan synthesis [14].

It was also assumed that these drugs may interfere with the synthesis and function of collagenases. In support of this hypothesis, a recent in vitro study has shown that human gingival fibroblasts treated with clinically relevant cyclosporin doses



Figure 4.
Nifedipine induced gingival enlargement.

exhibit significantly reduced levels of MMP-1 and MMP-3 secretion; these reduced levels may contribute to the accumulation of extracellular matrix components [15].

3.4 Idiopathic gingival enlargement

It is an uncommon benign hereditary condition with no specific cause. It is characterized by slow progressive firm and fibrous enlargement of gingiva. Synonyms are hereditary gingival fibromatosis, elephantiasis gingivae, congenital hypertrophy of gingiva, fibromatosis gingivae, congenital macrogingivae and hypertrophic gingiva. The color of the tissue appears pale pink, has characteristic leathery consistency and pebbled surface (**Figures 5 and 6**). Exaggerated stippling is observed. The enlargement poses esthetic and functional irregularities. It may also lead to displacement of teeth. It affects attached, marginal and interdental gingiva. The genetic mechanisms are not well understood [16]. The gingival enlargement usually begins at the time of eruption of the permanent dentition but can develop with the eruption of the deciduous dentition and rarely is present at birth [17].

3.5 Conditioned enlargement

It occurs when the patient response to plaque accumulation is magnified because of the systemic condition of the patient.

Enlargement in pregnancy: it occurs as single or multiple tumor like masses in the marginal or attached gingiva. The hormonal change in the pregnancy leads to increased vascular permeability, gingival edema and increased response to dental plaque. The lesion appears as mushroom like, bleed easily, sessile or pedunculated, protruding from the margin or interproximal area. The lesion does not invade the bone and has pinpoint markings on the surface (**Figure 7**). The lesion usually grows till third trimester after which it may regress spontaneously [2]. The hormonal changes induce changes in vascular permeability, which leads to gingival edema and an increased inflammatory response to dental plaque. The subgingival microbiota may also undergo changes, including an increase in *Prevotella intermedia* [18].

Enlargement in puberty: Due to change in hormones during adolescences leads to aggravated response during puberty in areas of plaque accumulation. It is manifested as bulbous enlargement in the papillary region leading to enlargement in facial region, lingual region is relatively unaffected. The tendency for recurrence into massive enlargement in presence of scanty deposits differentiates it from chronic inflammatory enlargement [2].

A longitudinal study of 127 children between the ages of 11 and 17 years demonstrated a high initial prevalence of gingival enlargement that tended to decline with



Figure 5.
Idiopathic gingival enlargement (left lateral).



Figure 6.
Idiopathic gingival enlargement (right lateral).

age [19]. Studies have reported that hormonal changes coincide with an increase in the proportion of *Prevotella intermedia* and *Prevotella nigrescens* [20, 21].

Plasma cell gingivitis: synonyms are atypical gingivitis and plasma cell gingivostomatitis, it exhibits enlargement in marginal gingiva extending onto attached gingiva. Gingiva appears reddish, soft, friable, and sometimes granular and has high bleeding tendency; no loss of attachment is seen (if not periodontally involved). It is located in the facial aspect of the attached gingiva and thus distinguished from plaque-induced gingivitis [6]. In rare instances, marked inflammatory gingival enlargements with a predominance of plasma cells can appear; these are associated with rapidly progressive periodontitis [22].

3.6 Nonspecific conditioned enlargement

Pyogenic granuloma: it is a tumor-like enlargement of gingiva which is considered to be exaggerated conditioned response to minor trauma. The exact nature of the systemic conditioning factor has not been identified [23]. The lesion varies from a discrete spherical, tumor-like mass with a pedunculated attachment to a flattened, keloid-like enlargement with a broad base. It is bright red or purple and either friable or firm, depending on its duration; in the majority of cases it presents with surface ulceration and purulent exudation (**Figure 8**). The lesion tends to involute spontaneously to become a fibroepithelial papilloma, or it may persist relatively unchanged for years [6].

3.7 Systemic diseases that cause gingival enlargement

Leukemia: leukemic enlargement is prominently because of accumulation of leukemic cells in the gingival. It manifests as diffuse or solitary and localized or



Figure 7.
Enlargement in pregnancy.



Figure 8.
Pyogenic granuloma.

generalized. It may appear as either a diffuse enlargement of the gingival mucosa, an oversized extension of the marginal gingival or a discrete tumor-like inter-proximal mass. In leukemic enlargement the gingiva is usually bluish red with a shiny surface. The consistency is moderately firm, but presents with a tendency towards friability and hemorrhage, occurring either spontaneously or on slight irritation [2]. Dreizen et al. found that cases with acute monocytic leukemia had the highest incidence of gingival infiltrates (M5) (66.7%) followed by acute myelomonocytic leukemia (M4) (18.5%) and acute myeloblastic leukemia (M1, M2) (3.7%) [24].

Wegener's granulomatosis: it is a rare disease characterized by acute granulomatous necrotizing lesions of the respiratory tract, including nasal and oral defects. It includes oral mucosal ulceration, gingival enlargement, abnormal tooth mobility, exfoliation of teeth, and delayed healing response [6]. The initial manifestations of Wegener's granulomatosis may involve the orofacial region and include oral mucosal ulceration, gingival enlargement, abnormal tooth mobility, exfoliation of teeth, and delayed healing response [25].

4. Neoplastic enlargement (gingival tumors)

4.1 Benign tumors of the gingiva

Epulis: it is a generic term used clinically to designate all discrete tumors and tumor-like masses of the gingiva [2]. The term is used to explain the location of the tumor mass not to portray it. Most lesions referred to as "epulis" are inflammatory rather than neoplastic (**Figure 9**).



Figure 9.
Epulis.

Fibroma: fibromas of gingiva arise either from connective tissue of gingiva or from periodontal ligament. Fibromas are slow-growing, spherical tumors that tend to be firm and nodular but may be soft and vascular. Fibromas are usually pedunculated. Hard fibromas of the gingiva are rare; most of the lesions diagnosed clinically as “fibromas” are inflammatory enlargements (**Figure 10**).

Papilloma: they are benign proliferations of surface epithelium associated with the human papillomavirus (HPV). Gingival papillomas appear as solitary, wart-like or cauliflower-like protuberances. They may be small and discrete or broad, hard elevations with minutely irregular surfaces.

Peripheral giant cell granuloma: giant cell lesions of the gingiva arise interdentially or from the gingival margin, occur most frequently on the labial surface, and may be sessile or pedunculated. They vary in appearance from smooth, regularly outlined masses to irregularly shaped, multilobulated protuberances with surface indentations. Ulceration of the margin is occasionally seen. The lesions are painless, vary in size, and may cover several teeth.

Central giant cell granuloma: these lesions arise within the jaws and produce central cavitation. They occasionally create a deformity of the jaw that makes the gingiva appear enlarged.

4.2 Malignant tumors of the gingiva

Squamous cell carcinoma is the most common malignant tumor of the gingiva. It may be exophytic, presenting as an irregular outgrowth, or ulcerative, appearing as flat, erosive lesions. Malignant melanoma is a rare oral tumor that tends to occur in the hard palate and maxillary gingiva of older persons. It is usually darkly pigmented and is often preceded by localized pigmentation. Fibrosarcoma, lymphosarcoma, and reticulum cell sarcoma of the gingiva are rare; only isolated cases have been described in the literature [6].

4.3 False enlargement

False enlargements are not true enlargements of the gingival tissues but may appear as such as a result of increases in size of the underlying osseous or dental tissues. The gingiva usually presents with no abnormal clinical features except the massive increase in size of the area. It may be caused by increased underlying bone tissue or presence of normal underlying dental tissue [2].

4.4 Treatment

The treatment of gingival enlargement is based on the understanding of the cause and underlying pathology. The treatment differs for each type of enlargement



Figure 10.
Fibroma.

based on the clinical and pathological signs and symptoms. The phase I therapy should be instituted before any surgical therapy.

4.5 Treatment protocol

The treatment protocol varies with each type of enlargement. Combinations of surgical and nonsurgical therapy are prevalent; used according to the need of the patient. The functional and esthetic demands should also be kept in mind.

4.5.1 Chronic inflammatory enlargement

These are presented as soft and edematous gingival tissues. The color changes are prominent with visibly reddish hue of the tissue. Bleeding is spontaneous. The therapy consists of thorough scaling and root planing and complete debridement of deposits [26]. This leads to shrinkage of tissue, slight if not complete.

Chronic gingival enlargement may also show fibrotic components; hence complete shrinkage of the tissue does not happen in such cases. Once the Phase I therapy has been instituted and gingival tissue does not return back to normal stage, surgical therapy should be considered. The surgical therapy consists of either gingivectomy procedures or/and flap operation. If the tissues are soft and edematous gingivectomy procedures are preferred. If the tissues are firm and fibrotic preferred treatment options is flap operation. The conservation of the keratinized, attached gingiva must be considered along with removal of the excessive gingival tissue [26].

During surgical procedure, the tissue is separated from the mucosa at its base by using a surgical blade. If the lesion extends interproximally, the interdental gingiva is included in the incision to ensure the exposure of deposits and form scalloped contour of gingiva. After complete removal of enlarged tissue and adequate accessibility, the root surfaces are scaled and planed, and the area is irrigated. A periodontal dressing is applied which is removed after a week. Depending on the extent of the surgery, the postoperative appointment may have to be scheduled in 2 weeks to allow for further healing. The healing is through secondary intention in gingivectomy. In case of flap operation healing is through primary intention. After removal of excess tissue and elevation of mucoperiosteal flap, roots surface are debrided and sutures are given [6].

4.5.2 Periodontal and gingival abscesses

Periodontal and gingival abscesses results in acute enlargement of gingival which is usually localized around the area of the lesion, and the content of the enlarged area is purulent material, which must be drained and the area curetted. Drainage should be either from periodontal pocket or external incision [6].

4.5.3 Drainage through the periodontal pocket

The peripheral area around the abscess is adequately anesthetized. The pocket wall is gently retracted using a periodontal probe or curette in an attempt to initiate drainage through the pocket entrance. Gentle digital pressure and irrigation may be used to express the exudate and drain the pocket. Curette is inserted into the pocket entrance to establish drainage. Thorough scaling and root planing is done. If the lesion is large and drainage cannot be established, root debridement by scaling and root planing or surgical access should be delayed until the major clinical signs have abated. Prophylactic antibiotics should be given. Antibiotic therapy alone without subsequent drainage and subgingival scaling is contraindicated and avoided [26].

4.5.4 Drainage through an external incision

The lesion is isolated and anesthetized. A vertical incision through the most fluctuant center of the abscess is made with a no. 15 surgical blade. The tissue adjacent to incision can be separated using a curette or periosteal elevator. The fluctuant matter is expressed, and the wound edges are approximated using mild digital pressure with a help of moist gauze pad. In abscesses manifesting with severe swelling and inflammation, aggressive mechanical instrumentation should be delayed in favor of antibiotic therapy to avoid damage to surrounding healthy periodontal tissues. Patient is dismissed after bleeding and suppuration are controlled. Patients are advised for post-treatment plaque control measures [6].

4.5.5 Chronic abscess

The chronic abscess is treated with scaling and root planing and, if indicated, surgical therapy. Surgical treatment is considered when deep vertical pocket or furcation defects are observed that cannot be treated with mere nonsurgical instrumentation. Access to subgingival calculus is mandatory in areas of deep pockets [6].

4.5.6 Gingival abscess

Treatment of the gingival abscess is done to reverse the acute phase and immediate removal of the cause. As it is often seen that the lesion gets fluctuant, exudate is expressed and becomes symptomless and the cycle is repeated, the offending agent is to be removed. Topical or local anesthesia by infiltration is administered. When possible, scaling and root planing are completed to establish drainage and remove microbial deposits. In more acute situations, the fluctuant area is incised with a no. 15 scalpel blade, and exudate may be expressed by gentle digital pressure. Any foreign material (offending agent e.g., dental floss, impression material) is removed. The area is irrigated with normal saline and covered with moist gauze under light pressure. Once bleeding is controlled, the patient is dismissed with post treatment instructions. The area is to be reassessed after 24 hours and if resolution is sufficient, scaling not previously completed is done. If the residual lesion is large or poorly accessible, surgical access may be required [26].

4.6 Drug-induced gingival enlargement

The examination of drug-induced gingival enlargement patient shows two components of the overgrown tissues which are either fibrotic, due to action of the drug on the physiologic gingival collagen turnover; or inflammatory, because of the presence of bacterial biofilm. Though the fibrotic and inflammatory changes present in the enlarged gingiva are the consequences of distinct pathologic processes, they almost always are observed as gingival enlargement induced by the combination of drugs and biofilm [6].

The role of bacterial biofilm in the overall pathogenesis of drug-induced gingival enlargement is not clear. Some studies indicate that biofilm is a prerequisite for gingival enlargement, whereas others suggest that the presence of biofilm is a consequence of biofilm accumulation caused by the enlarged gingiva [27].

The treatment of drug-induced gingival enlargement should be undertaken in consideration the medication used by the patient and the clinical features of the case. First, discontinuation of the drug or alternate medication should be considered. Consultation with the patient's physician is warranted for any such possibilities. It is not practically possible to completely discontinue the offending drug,

but alternate substitute of the drug may be an option. If any drug substitution is attempted, a time period of 6- to 12-month should be stalled between discontinuation of the offending drug and substitution with an alternative drug [2].

Along with this, oral hygiene instructions, scaling, and root planing should always be instituted. Reevaluation of the gingival enlargement after the alteration of drug therapy and planning of surgical treatment should be done. Alternative medications to the anticonvulsant phenytoin include carbamazepine and valproic acid, both of which have been reported to induce gingival enlargement to a lesser degree. A murine study suggested that lovastatin may attenuate the onset of gingival enlargement induced by phenytoin [28]. Further research is necessary to confirm the therapeutic value of lovastatin. For patients who are taking nifedipine, which has a reported prevalence of gingival enlargement of up to 86%, other calcium channel blockers such as diltiazem or verapamil may be viable alternatives. The reported prevalence of inducing gingival enlargement is 20% for diltiazem and 4% for verapamil [29]. In addition, consideration should be given to the use of another class of antihypertensive medications rather than calcium channel blockers. None of these drugs are known to induce gingival enlargement. Drug substitutions for cyclosporine are more limited.

Tacrolimus is another immunosuppressant that is used in organ transplant recipients [30]. The incidence of gingival enlargement in patients receiving tacrolimus therapy is approximately 65% lower than that in individuals who are receiving cyclosporine [30]. Clinical trials have also shown that the substitution of cyclosporine with tacrolimus results in a significant decrease in the severity of gingival enlargement as compared with patients who are kept on cyclosporine therapy [31]. The use of azithromycin to decrease cyclosporine-induced gingival enlargement resulted in significantly greater changes than those observed with an improvement in oral hygiene. The topical administration of azithromycin in the form of a toothpaste also decreased the severity of cyclosporine-induced gingival enlargement [32, 33].

Secondly, biofilm control is a mandatory step and hence should be prioritize by the clinician in the treatment of drug-induced gingival enlargement. Although the exact role played by bacterial biofilm is not fully understood, evidence suggests that good oral hygiene, chemotherapeutic agents, and the frequent professional removal of biofilm decrease the degree of gingival enlargement and improve overall gingival health [27, 34]. Due to the presence enlarged gingival tissue, it is associated with pseudo-pocket formation and abundant biofilm accumulation, which may lead to the development of periodontitis. Hence, meticulous biofilm control helps to maintain attachment levels. In addition, adequate biofilm control may help to prevent the recurrence of gingival enlargement in surgically treated cases. Still in many patients, gingival enlargement persists after careful consideration of the previous two approaches. With these patients, surgical removal of the enlarged gingiva must be considered [6].

The recurrence of drug-induced gingival enlargement is a reality in surgically treated cases. The major cause of the recurrence of gingival enlargement is the difficulty with postsurgical oral hygiene. Meticulous home care, with a soft, postsurgical brush and chlorhexidine gluconate rinses, is indicated. Frequent professional cleanings can also help reduce the degree of recurrence [35].

4.7 Leukemic gingival enlargement

Leukemic enlargement occurs with acute or subacute leukemia, and it is uncommon among patients in the chronic leukemic state. The patient's blood profile including bleeding and clotting times and platelet count should be checked before treatment, and the hematologist should be consulted before periodontal treatment

is instituted. Gingival bleeding, sometimes spontaneous, is often associated with leukemic gingival enlargement. After subsiding of acute symptoms, attention is directed to correction of the gingival enlargement. Removal of local irritating factors helps in controlling the inflammatory component of the enlargement. Scaling and root planning is done to achieve it. The initial treatment steps consist of gently removing all loose debris with cotton pellets, performing superficial scaling, and instructing the patient in oral hygiene for biofilm control. This hygiene should include the daily use of chlorhexidine mouthrinses. Oral hygiene procedures are of supreme importance for these patients. Definitive scaling and root planing are carried out at subsequent visits using local anesthesia (if required). Treatment sessions are confined to a small area of the mouth if hemostasis poses a challenge. Antibiotics are administered systemically the evening before and for a week after each treatment to reduce the risk of infection [6].

4.8 Gingival enlargement during pregnancy

The elimination of all local irritants that may be responsible for precipitating the gingival changes that occur during pregnancy should be done. This elimination is a preventive procedure to avoid any unfavorable situation as well as the treatment of gingival enlargement after it occurs. Marginal and interdental gingival inflammation and enlargement are treated by scaling and root planing. Treatment of tumor-like gingival enlargements consists of surgical excision, as well as the scaling and root planing of the tooth surfaces adjacent to the lesion. The enlargement may recur unless all irritants are removed. Food impaction is frequently an inciting factor. Gingival lesions during pregnancy should be treated as soon as they are detected, although not necessarily by surgical means. Scaling and root planing procedures and adequate oral hygiene measures may reduce the extent of the enlargement. Gingival enlargements do shrink after pregnancy, but they usually do not disappear. After pregnancy, the entire periodontal status of the patient should be reevaluated, and comprehensive treatment should be undertaken. Lesions should be removed surgically during pregnancy if they interfere with mastication or produce an esthetic disfigurement that bothers the patient. During pregnancy, the emphasis should be on (1) preventing gingival disease before it occurs and (2) treating existing gingival disease before it worsens [14].

4.9 Gingival enlargement during puberty

Gingival enlargement during puberty should be treated by phase I therapy, removal of all local irritant factors, controlling and removing the biofilm. Surgical removal is considered in severe cases and after instituting scaling and root planing. Oral hygiene measures are reinforced as high chances of recurrence is anticipated otherwise. Maintenance therapy is recommended [6].

4.10 Idiopathic enlargement

It usually requires surgical correction. Phase I therapy is undertaken to remove any source of irritants. Inflammatory component if present should be controlled. Functional and esthetic correction using a surgical therapy is undertaken depending on presence or absence of loss of attachment.

According to several authors, the best time is when all of the permanent dentition has erupted, because the risk of recurrence is higher before it [36]. Emerson demonstrated that the degree of enlargement did not appear to be related to the oral hygiene or to the amount of calculus present and that a correct physiologic contour of the marginal gingiva is more important to prevent recurrence [37] (**Table 1**).

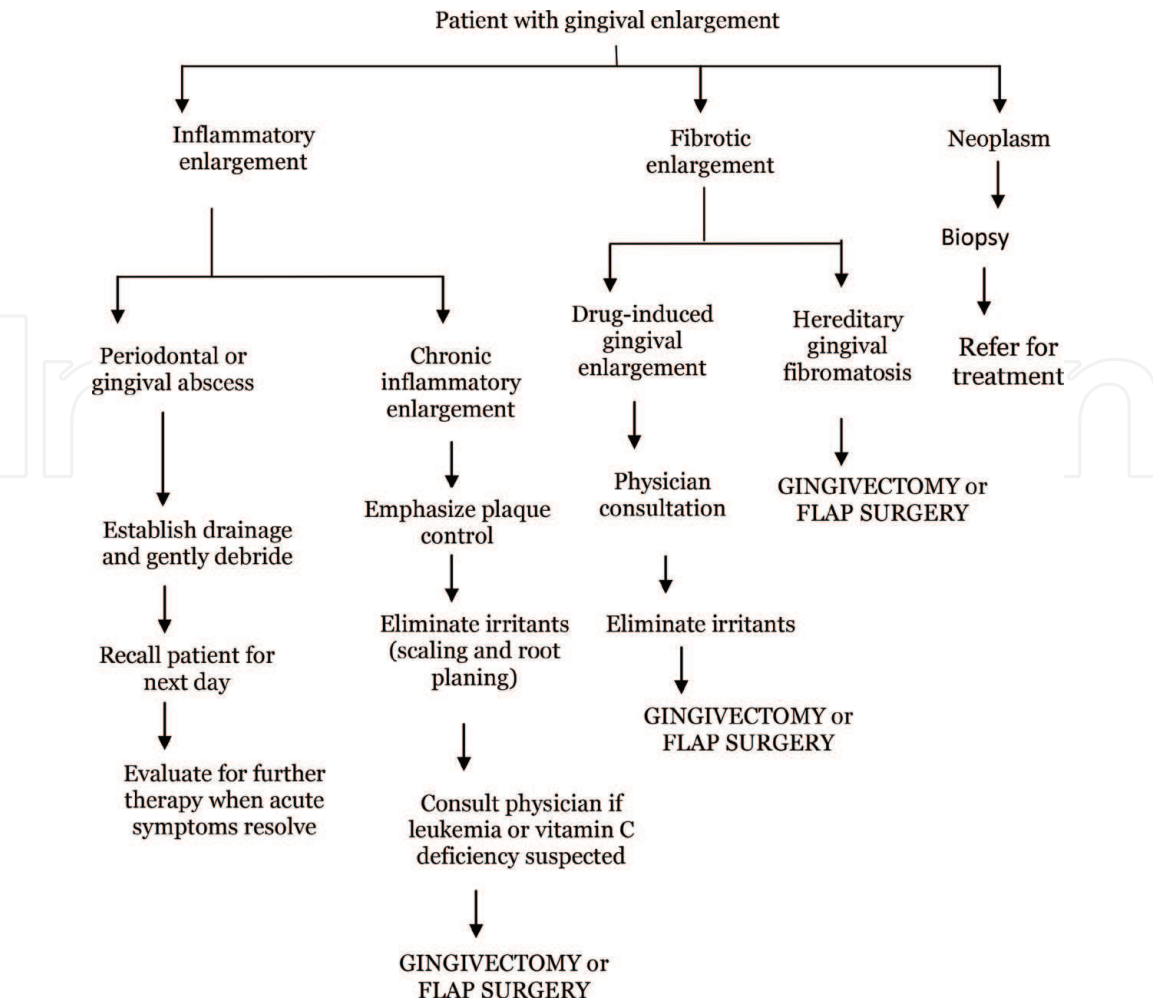


Table 1.
Treatment plan for gingival enlargement [44].

5. Surgical techniques for correction of gingival enlargement

5.1 Gingivectomy

Gingivectomy implies to the excision of gingival. The pocket wall (or enlarged tissue) is removed for accessibility.

Indications [38]: (1) elimination of suprabony pockets if the pocket wall is fibrous and firm, (2) elimination of gingival enlargements and (3) elimination of suprabony periodontal abscesses.

Contraindications to gingivectomy include the following: (1) access to bone required (2) narrow zone of keratinized tissue (3) esthetics particularly in the anterior maxilla (4) patients with high postoperative risk of bleeding (5) situations in which the bottom of the pocket is apical to the mucogingival junction.

Advantages: ease and simplicity of the procedure.

Disadvantages: more postoperative discomfort, increased chance of postoperative bleeding, sacrifices keratinized tissue and does not allow for osseous recontouring [3].

5.1.1 Gingivectomy procedures

In the latter part of the nineteenth century Robicsek (1884) pioneered gingivectomy procedure. Grant (1979) defined gingivectomy as being “the excision of the soft tissue wall of a pathologic periodontal pocket”. The surgical procedure included

elimination of pocket as well as osseous recontouring. Zentler (1918) later described a different technique [3].

Robicsek described a straight line incision to resect the gingival tissue while Zentler advocated a scalloped incision, first on the labial and then on the lingual surface of each tooth, the diseased tissue should be loosened and lifted out by means of a hook-shaped instrument. The soft tissues are removed and alveolar bone is exposed. The bone is scraped and debrided. The wound is then covered with some kind of anti-bacterial gauze or be painted with disinfecting solutions. Eradication of the deepened periodontal pocket and an area which can be easily maintained is expected [3].

5.2 Technique

The gingivectomy procedure as it is employed today was described in 1951 by Goldman (**Figures 11–16**) [3]

- After anesthesia of the affected area, the depths of pathological pockets are assessed using a periodontal probe. Bottom of the pocket is assessed and bleeding points are marked with a probe. Alternatively a pocket marker is used and bottom of the pocket is marked using the toothed end. The calibrated end is inserted into and measures the pocket. The bleeding points are used to guide the incision and to determine the depth of the tissue to be resected.
- The incision is given using scalpel or a Kirkland knife No. 15/16, maintaining the scalloping and festooning of the gingiva. The area with more bulky tissue will have more apically placed incision. In areas of thin gingival a less accentuated bevel is needed. The angulation of the incision is eternal bevel (45 degree towards the coronal portion). The incision is directed towards the base of the pocket and crest of the bone. Care should be observed to avoid exposure of bone. Physiologic contour of gingival should be established. Incision may be continuous or interrupted.
- After completing the incision, the interdental soft tissue is separated by a secondary incision using an Orban knife (No. 1 or 2) or a Waerhaug knife (No. 1 or 2; a saw-toothed modification of the Orban knife). Tissues are then separated using a curette. Tissue nippers are used to remove tissue tags and obtain smooth margins. Scaling and root planning is done to remove plaque and calculus.
- Probing should be done to assess for any remaining pockets if present. Rotatory instruments may be used to correct gingival contour, if necessary.
- Periodontal dressing is applied for protection of the surgical area. The dressing should be closely adapted to the buccal and lingual wound surfaces as well as to the interproximal spaces.
- Dressing should be given for 7 days. If necessary (depending on the healing and area of wound) dressing should remain in position for 10–14 days. Postoperative antibiotics and analgesics should be advised. Chlorhexidine gluconate (0.2%) mouthwash should be prescribed for oral hygiene.

5.2.1 Gingivectomy by electrosurgery

Gingivectomy can be done using electrosurgical unit. It provides hemostasis and proper contouring of the tissue. Use of electrosurgery also facilitates easy tissue incision accompanied with a strong hemostatic effect [39]. However, it is contraindicated in patients with cardiac pacemaker. Any contact to bone or cementum has to

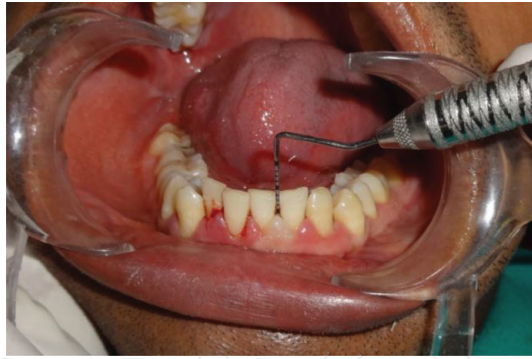


Figure 11.
Gingivectomy: measuring the pockets using periodontal probe.



Figure 12.
Gingivectomy: marking the pockets using pocket marker.



Figure 13.
Gingivectomy: incision given using Kirkland knife.



Figure 14.
Gingivectomy: after removal of excess tissue.



Figure 15.
Gingivectomy: periodontal dressing given.



Figure 16.
Gingivectomy: postoperative view after 1 month.

be avoided as irreparable damage is caused. Needle electrode is used for removal of enlarged tissue. Festooning and shaping can be done using ovoid or diamond shaped electrode. Electrode is activated in concise shaving motion making brief contact with the tissues in cut phase. Prolonged contact will result in charring of tissue. A ball electrode is used for control hemorrhage in coagulation phase (**Figures 17–19**).

5.2.2 Gingivectomy by laser

Soft tissue lasers are used for treatment of gingival enlargement. Commonly used lasers are carbon dioxide (CO₂) and the neodymium:yttrium-aluminum-garnet (Nd:YAG), which have wavelengths of 10,600 and 1064 nm, respectively. Proper protection should be observed along with eyewear and avoidance of any reflective surfaces. The procedure is similar to that of electrosurgery. Laser tip is used instead



Figure 17.
Gingivectomy by electrosurgery: preoperative view.



Figure 18.
Gingivectomy by electrosurgery: incision with electrode tip.



Figure 19.
Gingivectomy by electrosurgery: postoperative view after 1 month.

of electrodes for cutting and coagulation. Compared with the use of a conventional scalpel, lasers can cut, ablate and reshape the oral soft tissue more easily, with no or minimal bleeding and little pain as well as no or only a few sutures. Laser surgery occasionally requires no local anesthetic, or only a topical anesthetic [40].

5.2.3 Gingivectomy by chemosurgery

Chemicals can be used to remove gingival tissue. About 5% paraformaldehyde [41] or potassium hydroxide [42] has been used in the past. Epithelialization and reformation of the junctional epithelium and reestablishment of the alveolar crest fiber system occur more slowly in chemically treated gingival wounds than in those produced by a scalpel [43]. However due to inability in controlling depth of action and delayed healing response, it is not used anymore.

The gingivectomy procedures cannot be used in cases of attachment loss or if the bone exposure is required. Thus flap surgery is undertaken in such cases.

5.3 Flap surgery

The flap surgical technique is as follows [14]:

1. After adequate anesthesia, bone sounding is performed with periodontal probe to determine the presence and extent of bone deformities. Depths of periodontal/ pathological pockets are also assessed.
2. Incision is given on buccal and lingual aspects using a #15 surgical blade. The initial scalloped internal bevel incision is made at least 3 mm coronal to the mucogingival junction, which includes the creation of new surgical interdental papillae in each interproximal space.

3. Using the same blade gingival tissues are thinned in the buccolingual direction to the mucogingival junction. The blade establishes contact with the alveolar bone, and a full-thickness or split-thickness flap is elevated.
4. A similar scalloped internal bevel incision is given on the palatal aspect at a point where postoperative gingival margin is intended (at cementoenamel junction or at the bone crest in case of attachment loss). Thinning of palatal flap is done till the apical extent of the flap. The mucoperiosteal flap is then elevated.
5. Interdental incision is given with the help of an Orban knife, the base of each papilla connected to facial and lingual incisions is released.
6. Crevicular incisions are made on buccal, lingual and palatal areas to detach the collar of tissue. The collar of tissue is removed using curettes.
7. Tissues tags are removed using tissue scissors. The roots surfaces are thoroughly debrided.
8. Flap is replaced on to tooth bone junction and secured using interrupted or continuous mattress suture. Periodontal dressing is placed.
9. Sutures and dressing is removed after 1–2 weeks depending on the healing of the surgical area.
10. Postoperative antibiotics and analgesics are prescribed. Chlorhexidine gluconate mouthwash (0.2%) is given for plaque control.

6. Conclusion

Gingival enlargement is of prime concern to the patient as it impairs both function and esthetics. In excessive enlargement cases, a properly timed surgical procedure to reduce the tissue to a normal contour to reduce the tissues to a normal contour will yield maximum benefit to the patient, reducing the number of clinical visits needed and improving the patients' quality of life.

Conflict of interest

Author reports no conflict of interest.

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