Gingival diseases in children

The gingiva is the part of the oral mucous membrane that covers the alveolar processes and the cervical portions of the teeth. It has been traditionally divided into free and attached gingiva.

The free gingiva is the tissue coronal to the bottom of the gingival sulcus.

The attached gingiva extends apically from the free gingival groove to the mucogingival junction. Attached gingiva is necessary to maintain sulcus depth, to resist functional stresses during mastication, and to resist tensional stress by acting as a buffer between the mobile gingival margin and the loosely structured alveolar mucosa. The width of the attached gingiva is narrower in the mandible than in the maxilla, and both widths increase with the transition from the primary to permanent dentition in the child.

The gingival tissues are normally light pink in adults. The gingival colour of the young child may be more reddish due to increased vascularity and less keratinized thinner epithelium. This may be interpreted as mild inflammation. The surface of the gingiva of a child appears less stippled or smoother than that of an adult.

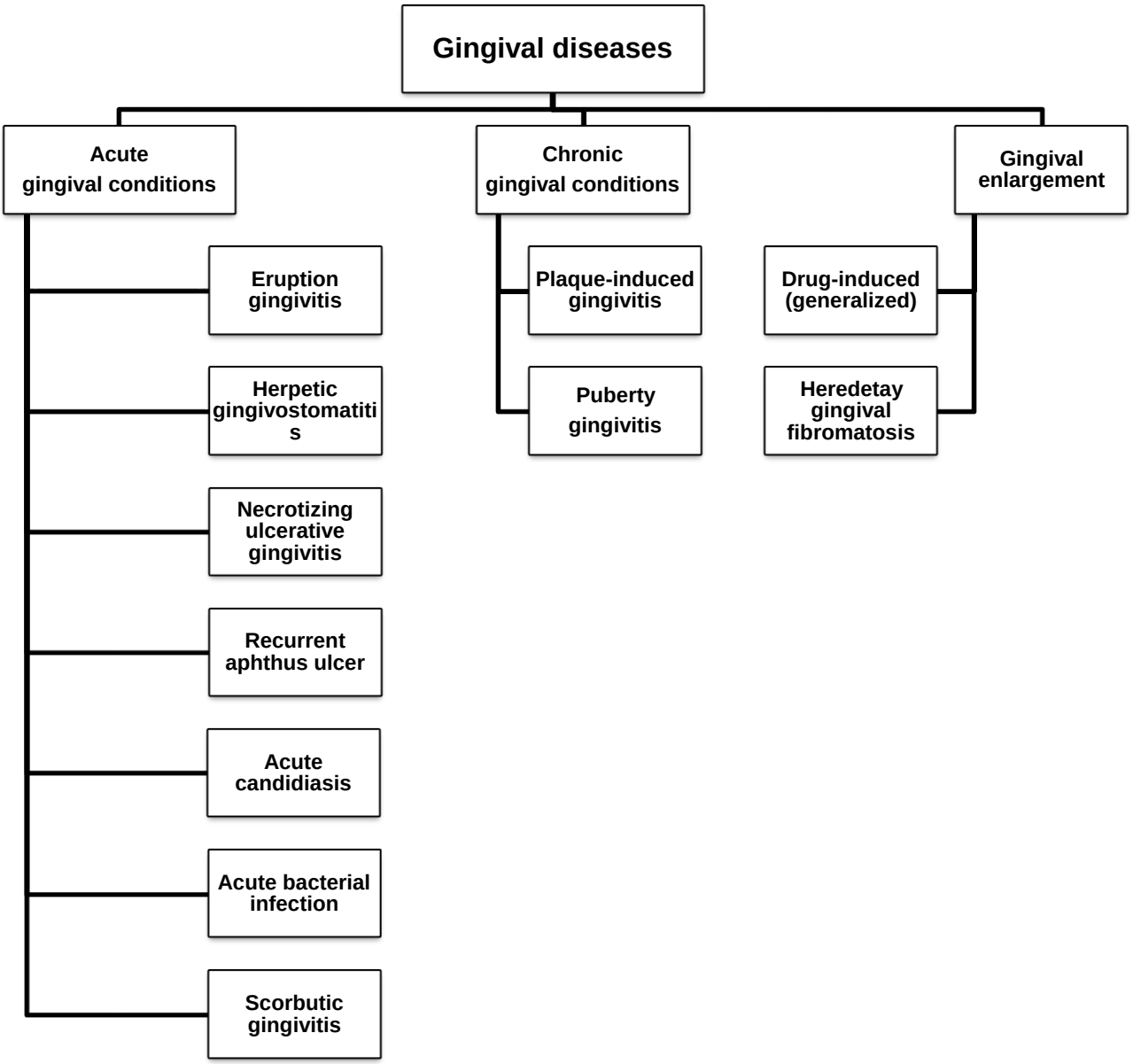
In the healthy adult, the marginal gingiva has a sharp, knifelike edge. During the period of tooth eruption in the child, however, the gingivae are thicker and have rounded margins due to the migration and cervical constriction of the primary teeth. The probing depths around primary teeth is approximately 2 mm, with the facial and lingual probe sites shallower than the proximal sites.

Gingivitis is inflammation involving only the gingival tissues surrounding the tooth without loss of connective tissue attachment.

Numerous surveys have shown that a large portion of the children has mild gingivitis. The major etiologic factors associated with gingivitis and more significant periodontal disease are uncalcified and calcified bacterial plaque.

Bacterial plaque is a complex, metabolically interconnected, and highly organized bacterial system that adhere firmly to the teeth. It consists of dense masses of microorganisms embedded in an intermicrobial matrix (biofilm). In sufficient concentration, it can disturb the host-parasite relationship and cause dental caries and periodontal disease.

❖ Types of gingival diseases in children:



❖ Chronic gingival conditions:

Chronic gingivitis is a nonspecific inflammatory lesion of the marginal gingiva which reflects the bacterial challenge to the host when dental plaque accumulates in the gingival crevice. The development of chronic gingivitis is enhanced when routine oral hygiene practices are impaired. Chronic gingivitis is reversible if effective plaque control measures are introduced. If left untreated, the condition invariably converts to chronic periodontitis which is characterized by resorption of the supporting connective tissue attachment and apical migration of the junctional epithelia

➢ Plaque-induced gingivitis:

The degree of dental cleanliness and the condition of the gingival tissues in children are related. Adequate mouth hygiene and cleanliness of the teeth are related to frequency of brushing and the thoroughness with which bacterial plaque is removed from the teeth. Favourable occlusion and the chewing of coarse, detergent-type foods, such as raw carrots, celery, and apples, have a beneficial effect on oral cleanliness.

Gingivitis associated with poor oral hygiene is usually classified as early (slight), moderate, or advanced. Early gingivitis is quickly reversible and can be treated with good oral prophylactic treatment and instruction in toothbrushing and flossing techniques to keep the teeth free of bacterial plaque. Gingivitis is generally less severe in children than in adults with similar plaque levels.

➢ Puberty gingivitis:

Puberty gingivitis is a distinctive type of gingivitis that occasionally develops in children in the prepubertal and pubertal period. The onset of puberty and the increase in circulating levels of sex hormones is one explanation for the increase in gingivitis seen in 11-year-olds. Oestrogen increases the cellularity of tissues and progesterone increases the permeability of the gingival vasculature.

The enlargement of the gingival tissues in puberty gingivitis is confined to the anterior segment and may be present in only one arch. The lingual gingival tissue generally remains unaffected. Treatment of puberty gingivitis should be directed toward improved oral hygiene, removal of all local irritants, restoration of carious teeth, and dietary changes necessary to ensure an adequate nutritional status.

❖ Acute gingival conditions:

The principal acute gingival conditions that affect children are primary herpetic gingivostomatitis and necrotizing ulcerative gingivitis. The latter is most frequently seen in young adults, but it also affects teenagers.

➢ Eruption gingivitis:

A transitory type of gingivitis that is often observed in young children when the primary teeth are erupting. This gingivitis, often localized and associated with difficult eruption, subsides after the teeth emerge into the oral cavity. The

greatest increase in the incidence of this type of gingivitis in children is often seen in the 6- to 7-year age group, when the permanent teeth begin to erupt. This increase in gingivitis apparently occurs because the gingival margin receives no protection from the coronal contour of the tooth during the early stage of active eruption, and the continual impingement of food on the gingivae exacerbates the inflammatory process.

Food debris, materia alba, and bacterial plaque often collect around and beneath the free gingival tissue, partially cover the crown of the erupting tooth, and cause the development of an inflammatory process. This inflammation is most commonly associated with the eruption of the first and second permanent molars, and the condition can be painful and can develop into pericoronitis or a pericoronal abscess. Mild eruption gingivitis requires no treatment other than improved oral hygiene. Painful pericoronitis may be helped when the area is irrigated with a counterirritant, such as Peroxyl. Pericoronitis accompanied by swelling and lymph node involvement should be treated with antibiotic therapy.

➢ Herpetic ginivostomatitis:

Herpetic gingivostomatitis is an acute infectious disease caused by Herpes Simplex virus (HSV-I). The primary infection is most frequently seen in children between 2 and 5 years of age, although older age groups can be affected. A degree of immunity is transferred to the new born from circulating maternal antibodies, so an infection in the first 12 months of life is rare. It is believed that 99% of all primary infections are of the subclinical type. In some preschool children the primary infection may be characterized by only one or two mild sores on the oral mucous membranes, which may be of little concern to the child or may go unnoticed by the parents. In other children the primary infection may be manifested by acute symptoms (acute herpetic gingivostomatitis).

Transmission of the virus is by droplet infection and the incubation period is about a week. The child develops a febrile illness with a raised temperature of 37.8–38.9°C. Headaches, malaise, oral pain, mild dysphagia, and cervical lymphadenopathy are the common symptoms that accompany the fever and precede the onset of a severe oedematous marginal gingivitis. Characteristic fluid-filled vesicles appear on the gingiva and other areas such as the tongue, lips, and buccal and palatal mucosa. The vesicles, which have a grey membranous covering, rupture spontaneously after a few hours to leave

extremely painful (1-3 mm diameter) yellowish ulcers with red inflamed margins. The clinical episode runs a course of about 10-14 days and the oral lesions heal without scarring. Rare but severe complications of the infection are aseptic meningitis and encephalitis.

Herpetic gingivostomatitis does not respond well to active treatment. Bed rest and a soft diet are recommended during the febrile stage and the child should be kept well hydrated. Pyrexia is reduced using a paracetamol or ibuprofen suspension and secondary infection of ulcers may be prevented using chlorhexidine. A chlorhexidine mouth rinse (0.2%, two to three times daily) can be used in older children who are able to expectorate, but in younger children (under 6 years of age) a chlorhexidine spray can be used (twice daily) or the solution applied using a sponge swab. To help the child eat, mild topical anaesthetic agents can be used before mealtime

In severe cases of herpes simplex infection, systemic aciclovir can be prescribed as a suspension (200mg) swallowed five times daily for 5 to 10 days. In children under 2 years the dose is halved. Aciclovir is active against the herpesvirus but is unable to eradicate it completely. The drug is most effective when given at the onset of the infection.

After the initial primary attack during early childhood, the herpes simplex virus becomes inactive and resides in the host's epithelial cells. Reactivation of the latent virus or re-infection in subjects with acquired immunity occurs in children and adults. Recurrent disease presents as an attenuated intra-oral form of the primary infection or as herpes labialis, i.e. the common 'cold sore' on the mucocutaneous border of the lips. Cold sores are treated by applying aciclovir cream (5%) five times daily for about 5 days. To prevent auto-inoculation and spread of the lesions onto hands and the face, children should be discouraged from touching the vesicles.

➢ Necrotizing ulcerative gingivitis (Vincent infection):

Necrotizing ulcerative gingivitis (NUG) is one of the most common acute diseases of the gingiva. The condition occurs occasionally in children from 6 to 12 years old, and is more commonly seen in young adults and rarely seen among preschool children.

NUG is characterized by necrosis and ulceration, which first affect the

interdental papillae and then spread to the labial and lingual marginal gingiva. The ulcers are 'punched out', covered by a yellowish-grey pseudomembranous slough, and extremely painful to the touch. The acute exacerbation is often superimposed upon a pre-existing gingivitis, and the tissues bleed profusely on gentle probing. The standard of oral hygiene is usually very poor. A distinctive halitosis is common in established cases of NUG, although fever and lymphadenopathy are less common than in herpetic gingivostomatitis. The clinical course of NUG is such that the acute stage enters a chronic phase of remission after 5–7 days. However, recurrence of the acute condition is inevitable, and if this acute–chronic cycle is allowed to continue the marginal tissues lose their contour and appear rounded. Eventually, the inflammation and necrosis involve the alveolar crest and the subsequent necrotizing periodontitis leads to rapid bone resorption and gingival recession. Progressive changes are also a consequence of inadequate or incomplete treatment.

✓ Treatment:

It is important that the patient is informed at the outset of the nature of NUG and the likelihood of recurrence of the condition if the treatment is not completed. The treatment should involve:

1- Intense oral hygiene: A soft multi-tufted brush is recommended when a medium-textured brush is too painful. Mouth rinses may be recommended but only for short-term use (7–10 days). Rinsing with chlorhexidine (0.2%) for about a minute reduces plaque formation, and the use of a hydrogen peroxide mouth rinse oxygenates and cleanses the necrotic tissues.

2- Mechanical debridement should be undertaken at the initial visit. An ultrasonic scaler with its accompanying water spray can be effective with minimal discomfort for the patient. Further, if NUG is localized to one part of the mouth, local anaesthesia of the soft tissues can allow some sub-gingival scaling to be undertaken.

3- Antibiotic: In severe cases of NUG, a 3-day course of metronidazole (for children over 10 years of age: 200mg three times daily) alleviates the symptoms, but the patients must be informed that they are required to re-attend for further treatment.

Occasionally, it is necessary to surgically recontour the gingival margin (gingivoplasty) to improve tissue architecture and facilitate subgingival cleaning.

➢ Recurrent aphthus ulcer:

The recurrent aphthous ulcer (RAU)—also referred to as recurrent aphthous stomatitis (RAS)—is a painful ulceration on the unattached mucous membrane that occurs in school-aged children and adults. The peak age for RAU is between 10 and 19 years of age. It has been reported to be the most common mucosal disorder in people of all ages and races in the world.

This disorder is characterized by recurrent ulcerations on the moist mucous membranes of the mouth, in which both discrete and confluent lesions form rapidly in certain sites and feature a round to oval crateriform base, raised reddened margins, and pain. They may appear as attacks of minor or single, major or multiple, or herpetiform lesions. They may or may not be associated with ulcerative lesions elsewhere.

Lesions persist for 4 to 12 days and heal uneventfully, leaving scars only rarely and only in cases of unusually large lesions. The description of RAU frequently includes the term canker sores.

The cause of RAU is unknown. Local and systemic conditions along with a genetic predisposition and immunologic and infectious microbial factors have been identified as potential causes. It is also possible that the lesions are caused by an autoimmune reaction of the oral epithelium.

Local factors include trauma, allergy to toothpaste constituents (sodium lauryl sulfate), and salivary gland dysfunction. Injuries caused by cheek biting and minor facial irritations are probably the most common precipitating factors. Nutritional deficiencies are found in 20% of persons with aphthous ulcers. The clinically detectable deficiencies include deficiencies of iron, vitamin B12, and folic acid. Stress may prove to be an important precipitating factor, particularly in stress-prone groups, such as students in professional schools and military personnel.

Current treatment is focused on promoting ulcer healing, reducing ulcer duration and patient pain, maintaining the patient’s nutritional intake, and preventing or reducing the frequency of recurrence of the disease. Numerous

treatments have been recommended for RAU, but a completely successful therapy has not been found. Topical anti-inflammatory and analgesic medicines and/or systemic immunomodulatory and immunosuppressive agents have been used for RAU. The primary line of treatment uses topical gels, creams, and ointments as anti-inflammatory agents. Currently, a topical corticosteroid (e.g., 0.5% fluocinonide) is applied to the area with a mucosal adherent (e.g., Orabase). For example, the application of triamcinolone acetonide (Kenalog in Orabase) to the surfaces of the lesions before meals and before sleeping may also be helpful.

In severe cases oral prednisone has been prescribed. Topical rinses have also been helpful for the relief of RAU. The topical application of tetracycline to the ulcers is often helpful in reducing the pain and in shortening the course of the disease. A mouthwash containing suspension of one of the tetracyclines has been helpful to some, but the mouthwash should not be swallowed.

➢ Acute candidiasis (thrush, candidosis, moniliasis):

Candida (Monilia) albicans is a common inhabitant of the oral cavity but may multiply rapidly and cause a pathogenic state when host resistance is lowered. Young children sometimes develop thrush after local antibiotic therapy, which allows the fungus to proliferate.

The lesions of oral candidiasis appear as raised, furry white patches, which can be removed easily to produce a bleeding underlying surface.

Antifungal antibiotics control thrush. For infants and very young children, a suspension of 1 mL (100,000 units) of nystatin (Mycostatin) may be dropped into the mouth for local action 4 times a day. Nystatin is non-irritating and nontoxic. Clotrimazole suspension (10 mg/mL), 1 to 2 mL applied to affected areas 4 times daily, is an effective antifungal medication. Systemic fluconazole suspension (10 mg/mL) is safe to use in infants at a total dosage of up to 6 mg/kg/day. For children old enough to manage solid medications allowed to dissolve in the mouth, clotrimazole troches or nystatin pastilles are recommended because the therapeutic agent remains in the saliva longer than with the liquid medication. For children old enough to swallow, systemic fluconazole (100 mg tablets) in a 14 day course may be prescribed when the infection has not responded to topical antifungal agents.

➢ Acute bacterial infection:

The prevalence of acute bacterial infection in the oral cavity is unknown. Acute streptococcal gingivitis is an example of this type of infection. It is characterized by enlarged papilla, gingival abscesses, and painful, erythematous gingiva that bleed easily. Cultures show a predominance of hemolytic streptococci. Acute infections of this type may be more common than was previously realized. The diagnosis is difficult to make, however, without extensive laboratory tests. Broadspectrum antibiotics are recommended if the infection is believed to be bacterial in origin. Improved oral hygiene is important in treating the infection. As with any acute microbial oral infection, CH mouthrinses are also appropriate.

➢ Ascorbic acid-deficiency gingivitis (Scorbutic gingivitis):

Scorbutic gingivitis is associated with vitamin C deficiency and differs from the type of gingivitis related to poor oral hygiene. The involvement is usually limited to marginal tissues and papillae. The child with scorbutic gingivitis may complain of severe pain. Spontaneous hemorrhage may be evident.

Aside from dietary deficiencies, ascorbic acid deficiency gingivitis can be seen in both pediatric and adult cancer patients undergoing radiotherapy and/ or chemotherapy where the mucosal linings of the intestinal walls are affected and the absorption of nutrients is impaired.

Severe clinical scorbutic gingivitis is rare in children. However, it may occur in children allergic to fruit juices when provision of an adequate dietary supplement of vitamin C is neglected. When blood studies indicate a vitamin C deficiency and exclude other possible systemic conditions, the gingivitis responds dramatically to the daily administration of 250 to 500 mg of ascorbic acid. Older children and adults may require 1 g of vitamin C for 2 weeks to speed recovery.

A less severe type of gingivitis resulting from vitamin C deficiency is probably much more common than most dentists realize. Inflammation and enlargement of the marginal gingival tissue and papillae in the absence of local predisposing factors are possible evidence of scorbutic gingivitis. Questioning the child and parents regarding eating habits and using the 7-day diet survey frequently reveal

that the child is receiving inadequate amounts of foods containing vitamin C.

Complete dental care, improved oral hygiene, and supplementation with vitamin C and other water-soluble vitamins will greatly improve the gingival condition.

❖ Gingival enlargement:

Enlargement of the gingiva is a well-recognized increase in the size of the gingiva. It can be caused by a number of factors such as inflammatory conditions or as a side effect of certain medications.

➢ Drug-induced gingival enlargement (generalized):

The medications most frequently associated with gingival enlargement are phenytoin, cyclosporine, and nifedipine.

- **Phenytoin**

Phenytoin is an anticonvulsant used in the management of epilepsy. Gingival enlargement occurs in about 50% of dentate subjects who are taking the drug, and is most severe in teenagers and those who are cared for in institutions. The exact mechanism by which phenytoin induces enlargement is unclear. The gingival enlargement reflects an overproduction of collagen (rather than a decrease in degradation). Phenytoin-induced enlargement has been associated with a deficiency of folic acid, which may lead to impaired maturation of oral epithelia.

- **Cyclosporine**

Cyclosporine is an immunosuppressant drug that is used widely in organ transplant patients to prevent graft rejection. Approximately 30% of patients taking the drug demonstrate gingival enlargement, with children being more susceptible than adults. The exact mechanism of the drug in causing enlargement is unknown. There is evidence to suggest a stimulatory effect on fibroblast proliferation and collagen production as well as an inhibitory effect on collagen breakdown by the enzyme collagenase.

- **Nifedipine**

Nifedipine is a calcium-channel blocker that is used in adults for the control of cardiovascular problems. It is also given to post-transplant patients to reduce the

nephrotoxic effects of cyclosporine. The incidence of gingival enlargement in dentate subjects taking nifedipine is 10–15%. The drug blocks the calcium channels in cell membranes—intracellular calcium ions are a prerequisite for the production of collagenases by fibroblasts. The lack of these enzymes could be responsible for the accumulation of collagen in the gingiva.

Clinical features of gingival enlargement:

The clinical changes seen in drug-induced enlargement are very similar irrespective of the drug involved. The first signs of change are seen after 3–4 months of drug administration. The interdental papillae become nodular before enlarging more diffusely to encroach upon the labial tissues. The anterior part of the mouth is most severely and frequently involved, so that the patient's appearance is compromised. The tissues can become so abundant that oral functions, particularly eating and speaking, are impaired. Enlarged gingiva is pink, firm, and stippled in subjects with a good standard of oral hygiene. When there is a pre-existing gingivitis, the enlarged tissues compromise an already poor standard of plaque control. The gingiva then exhibit the classical signs of gingivitis.

Management of gingival enlargement:

A strict programme of oral hygiene instruction, scaling, and polishing must be implemented. Severe cases of gingival enlargement inevitably need to be surgically excised (gingivectomy) and then recontoured (gingivoplasty) to produce an architecture that allows adequate access for cleaning.

Follow-up programme is essential to ensure a high standard of plaque control and to detect any recurrence of the enlargement. As the causative drugs need to be taken on a long-term basis, recurrence is common. When a phenytoin-induced enlargement is refractory to long-term treatment, the patient's physician may be requested to modify or change the anticonvulsant therapy to drugs such as sodium valproate or carbamazepine, which do not cause gingival problems.

➢ Hereditary gingival fibromatosis:

Hereditary gingival fibromatosis (HGF) is characterized by a slow, progressive, benign enlargement of the gingiva. Clinically, the HGF-gingival enlargement is characterized as being of normal color, firm consistency, non-hemorrhagic, and

asymptomatic with an equal gender predilection.

HGF, which is the most common genetic form of gingival enlargement, usually has an autosomal-dominant mode of inheritance. This rare type of gingivitis has been referred to as elephantiasis gingivae or hereditary hyperplasia of the gums. The gingival tissues appear normal at birth but begin to enlarge with the eruption of the primary teeth. Although mild cases are observed, the gingival tissues usually continue to enlarge with eruption of the permanent teeth until the tissues essentially cover the clinical crowns of the teeth. The dense fibrous tissue often causes displacement of the teeth and malocclusion. The condition is not painful until the tissue enlarges to the extent that it partially covers the occlusal surfaces of the molars and becomes traumatized during mastication.

Surgical removal of the hyperplastic tissue achieves a more favourable oral and facial appearance. However, hyperplasia can recur within a few months and can return to the original condition within a few years. Although the tissue usually appears pale and firm, the surgical procedure is accompanied by excessive hemorrhage. Therefore quadrant surgery is usually recommended. CO2 laser evaporation were used to reduce the gingival tissue. The importance of excellent plaque control should be stressed to the patient because this delays the recurrence of the gingival overgrowth.

❖ References:

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