

Gingival Diseases in Children and Adolescents

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Abstract

Gingival diseases are prevalent in both children and adolescents. These diseases may or may not be associated with plaques, maybe familial in some cases, or may coexist with systemic illness. However, gingiva and periodontium receive scant attention as the primary dentition does not last for a considerable duration. As gingival diseases result in the marked breakdown of periodontal tissue, and premature tooth loss affecting the nutrition and global development of a child/adolescent, precise identification and management of gingival diseases is of paramount importance. This article comprehensively discusses the nature, spectrum, and management of gingival diseases.

Keywords: *Gingival diseases; children and adolescents; spectrum, and management.*

Introduction

Children are more susceptible to several gingival diseases, paralleling to those observed in adults, though vary in numerous aspects. Occasionally, natural variations in the gingiva can masquerade as genuine pathology.¹ On the contrary, a manifestation of a life-threatening underlying condition is misdiagnosed as normal gingiva. Hence, it is crucial for dental professionals to acquire expertise and diagnostic skills to identify the diseases in childhood before it turns out to be a catastrophe.²

Anatomical Consideration: Gingiva of children and adults differ anatomically. Gingiva of the primary dentition usually appears pale pink, but paler than that of adults. The marginal gingiva has rich vascularity but comprises fewer connective tissues. The thinner,

reddish epithelium with mild keratinization may be misdiagnosed as inflammation. Lesser variability in the width of the attached gingiva in the primary dentition results in fewer mucogingival problems. The interdental papilla is broad buccolingual, and narrow mesiodistally. The junctional epithelium associated with the deciduous dentition is thicker than the permanent dentition. Gingival sulcular depth in children varies from 1 to 2 mm, which is shallower than that in adults.³

Epidemiology: Previous investigations have revealed the frequent occurrence of gingivitis of various intensity in children and adolescents. These investigations illustrate that the universality of severe forms of periodontal pathologies is fewer in the younger population as compared to the adults. Al Bandar and colleagues in 1996 evaluated a substantial number of adolescents in the United States and reported the existence of gingivitis in 82.1% of the participants. Several other studies from other parts of the world reported comparable results. Epidemiological studies indicate that loss of periodontal attachment and supporting bone at different locations can be recognized in 1% to 9% of 5 to 11-year-old, 1% to 46% of 12 to 15-year-olds (juvenile periodontitis) and as low as 2.75% of 16-17-year-olds (chronic periodontitis). When a broad mixture of the population was taken into consideration, reports revealed

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a fewer universality of periodontitis among pediatric and adult patients. Many investigators have proclaimed that higher plaque quantity and fewer inflammation in children as compared to young individuals. However, physicians recognize that the tissue destruction related to the periodontal pathologies in children and adolescents are generally self-healing and less prevalent compared to that in the adults.⁴⁻⁷

Classification of Gingival Diseases:

A. Gingival Disease Associated With Plaque

1. Without Local Contributing Factor

Plaque Induced Gingivitis: The presence of plaques is the prime mover of this pathological entity. Dental plaque usually builds up more briskly in the 8-12 years age group compared to the adults. However, gingivitis in the pediatric population is typically less severe than in adults with the same plaque level. Inflammation is generally restricted to the marginal gingiva with insignificant depletion of bone or connective tissue attachment in the majority of the cases. Red, linear inflammation accompanied by underlying chronic changes including swelling, augmented vascularity, and hyperplasia. Bleeding and deep pockets are not common findings in children. The greatest intensity of gingival inflammation exists in patients aged 14-16-year. Aggressive plaque control is recommended along with gingivectomy or gingivoplasty as per the condition.^{8,9}

2. With Local Contributing Factor

(a) Eruption cyst and hematoma: Erupting teeth are frequently related to a variety of dentigerous cyst known as eruption cyst. These cysts have circumscribed swellings and are usually translucent and fluctuant. The presence of blood in the cystic cavities imparts a purple/deep-blue discoloration to the circumscribed swelling, described as eruption hematoma.¹⁰

(b) Eruption gingivitis: Emergence of primary teeth into the oral cavity is often associated with gingival inflammation. Malalignment of the erupting teeth and/or neglected oral hygiene may exaggerate the inflammation. However, optimal occlusion of the tooth subsides the inflammation. It may reappear in the 6-7 years of age group when the permanent teeth set to erupt. This develops, as the coronal contour of the tooth provides limited protection to the gingival margin during the initial stage of an active eruption. Additionally, frequent gingival food impingement is

also contributory. Hence, plaque control should be taken seriously before it culminates in pericoronal abscess or leads to pericoronitis.¹¹

(c) Gingivitis secondary to the orthodontic appliance:

Effective brushing of the interproximal tooth is hampered during fixed appliance therapy. Accumulated plaques have a subgingival shift generated by the tipping movement which is virtually out of reach of the bristles. The gingival changes occurring in the first two months of appliance placement is temporary and largely reversible.¹²

(d) Gingivitis associated with mouth-breathing:

Oral tissue undergoes dehydration as an effect of frequent mouth-breathing, resulting in inflammation of the gingiva and halitosis. This should be addressed with appropriate oral hygiene, moisturizing the tissues, use of the oral screen to protect the tissue during sleep, planning a comprehensive treatment protocol with orthodontics, and otolaryngology consultations.¹³

(e) Other elements:

Excessive overjet and overbite, nasal blockage, partially exfoliated, loose deciduous, malaligned tooth, chipped off sharp edges of partly cavitated and decayed teeth can usually lead to gingival inflammation.¹⁴

B. Gingival Diseases and Systemic Factors:

1. Effect of Endocrine Alterations

(a) Puberty gingivitis: Pubertal gingivitis or steroid hormone-associated gingivitis is described as aggravation of gingivitis by pre-pubertal and pubertal alterations in sex hormone levels. This can be justified by estrogen and progesterone upsurge in the gingival tissues leading to vasodilatation and proliferation and increased susceptibility to inflammation. The interrelationship between elevated levels of the gonadotrophic hormones and pubertal gingivitis is noticeable from the fact that gingival inflammation peaks prior in girls (10-13 years) than in boys (13-15 years). According to in vivo evidence, *P. intermedius* is associated with the elevated levels of plasma estrogen and progesterone, suggesting that *P. intermedius* thrives on these hormones for nutrients. It is markedly distinguished by conspicuous inflammation, bluish-red appearances, swelling, and overgrowth, prominent bulbous interproximal papillae of anterior segments which are precipitated from local irritating factors

that would normally evoke a relatively benign gingival reaction. Management of puberty gingivitis should be targeted towards professional prophylaxis, elimination of all local irritants, restoration of carious teeth, and nutritional monitoring to ensure a sufficient nutritional status. Occasionally, the fibrotic transformation of the gingival swelling warrants timely surgical debridement.¹⁵

2. Gingivitis Associated with Leukemia: Leukemias are malignant diseases resulted from the proliferation of the WBC-precursors. Leukemia may have an acute or a chronic presentation and may be associated with one of the WBC precursors such as granulocytes (myeloid), lymphocytes, or monocytes. Acute leukemias are prevalent under the age group of 19-years. Acute lymphoblastic leukemia is common in the first decade of life. Factors that have been suggested in the etiology are radioactive rays and chemical damage, inherited risk factors (for example, Down's syndrome), immune-compromised state, and viral ailment. Gingiva appears as swollen, glazed, and purplish-deep-red in color accompanying hemorrhage. The swelling may emerge as a scattered expansion of the gingival mucosa, and overgrown augmentation of the marginal gingiva, or a distinct lump like interdental papillary mass. The texture is reasonably firm, but there is a propensity towards friability and bleeding, either involuntarily or on trivial provocation. Indolence, restlessness, painful throat, pyrexia, recurrent dermatological manifestations, purpura, cervical lymphadenopathy, splenomegaly, hepatomegaly, and petechiae are common signs.¹⁶⁻¹⁸

3. Associated with Nutritional Deficiency

(a) Gingivitis associated with nutritional deficiency: Several lines of evidence indicate an association of hypervitaminosis and mineral deficiencies with periodontal diseases. Scurvy, which emerges due to hypovitaminosis-C culminates in the reduction of collagen synthesis or the production of abnormal ones. Oral scurvy manifests with painful gingival swelling, edematous and spongy gingiva, and hemorrhage, either spontaneously or with a slight provocation. A combination of vitamin C deficiency and poor oral hygiene contributes to a painful version of gingivitis known as scorbutic gingivitis. It has additional signs that include ulcerative gingivitis, malodor, the fast formation of the periodontal pocket, and tooth damage.

C. Effect of Medications: Drug-induced gingival overgrowth: Gingival hyperplasia is an established side effect of several medications which include phenytoin (anti-convulsant), cyclosporine (immunodepressant), nifedipine, verapamil, and amlodipine (calcium channel blocker and anti-hypertensive). Gingival hyperplasia was described in 30% of patients receiving cyclosporin (Seymour & Heasman 1992), 50% of patients receiving phenytoin (Hassell 1981), and 15% of patients receiving the above-mentioned calcium channel blockers (Lederman et al. 1984, Pernu et al. 1989, Seymour et al. 1994). The gums look normal during the neonatal period but start to enlarge with the eruption of the primary teeth. This pattern of gingival hyperplasia typically starts as a nodular bulge in the interdental papillae before growing extensively to intrude the marginal gingival and labial tissues. The mouth is predominantly affected in the anterior side, but occasionally it affects the incisal and occlusal surfaces of the teeth. Histological hallmarks of this condition include epithelial acanthosis, higher fibroblast quantity, and enhanced collagen synthesis. Mechanical and chemical plaque control method are essential for the improvement of oral hygiene. Besides, professional scaling and root planning are warranted to eliminate the local contributing factors. On occasions, gingivectomy and gingivoplasty are recommended so that the patient can be esthetically sound which ultimately enhances the hygiene. Patients' physicians should be consulted to discuss the drug side effects so that other safe therapeutic avenues can be explored at the earliest.¹⁷

B. Non-Plaque Induced Gingival Diseases

Bacterial

(a) Acute necrotizing ulcerative gingivitis (ANUG): ANUG is also recognized as Trench mouth or Vincent's disease and is caused by *Borrelia vincentii*. ANUG has a greater prevalence in developing countries than in developed countries, and children are more frequently affected compared to adults. India has up to 2/3rd of the cases affecting the children in the first decade of life. ANUG is described by punched-out crater-like ulcers in interproximal gingiva coated with a grayish-white pseudo-membrane, which may expand to encompass marginal gingiva. It sometimes presents with deep pockets as a result of extensive gingival necrosis and crestal alveolar bone destruction. Patients

commonly report excruciating pain, a distinctive metallic taste, spontaneous gingival bleeding, and halitosis. Low-grade fever, swollen lymph nodes, restlessness, myalgia, loss of appetite are the frequent accompanying symptoms. Simultaneous local and systemic therapy is required to manage ANUG. The initial step should be professional scaling and root planing along with the debridement of necrotic tissue. Patients are encouraged to maintain a proper diet and oral hygiene. Oxidizing mouthwash (e.g., chlorhexidine) may serve to improve microbial stability. Five-day therapy with penicillin or erythromycin is usually recommended. Metronidazole can be administered for the speedy recovery of symptoms.¹²⁻¹⁵

Viral

- (a) **Acute herpetic gingivostomatitis:** This self-limiting disease occurs due to Herpes simplex virus (HSV-1) infection. Pediatric patients younger than 6-7 years are chiefly affected. It is characterized by a scattered, reddish, and glazed appearance of the gums and the neighboring tissues. Varying grades of swelling, spontaneous gingival bleeding, disjunct spheroidal gray vesicles that eventually burst and develop into ulcers with an erythematous, raised, nimbus like edge and a depressed yellowish or grayish-white central part are also recognized.¹⁸

Fungal

- (a) **Acute candidiasis (Thrush, Candidiasis):** It arises due to the overgrowth of the fungus *Candida albicans*. Children frequently develop acute candidiasis after prolonged local antibiotic therapy. The lesions appear as elevated, furry, white patches, which can be taken out easily to produce bleeding of the underlying surface. For infants and very young children, daily administration of Nystatin suspension (1 ml, 4 times/d) is recommended. Clotrimazole suspension is also highly effective.
- (b) **Linear gingival erythema (LGE):** It is clinically described by a reddish, linear band of 2-3mm width on the marginal gingiva presented as petechiae-like or disperse erythematous changes on the attached gingiva which may be associated with profuse hemorrhage. It might be limited to less than two teeth but it is frequently a generalized gingival pathology.¹³⁻¹⁵

Congenital Anomalies:

- (a) **Congenital Epulis(Gingival Granular Cell Lesion):** It is an uncommon gingival tumor of unknown histogenesis that grows commonly in newborns, especially on the maxillary alveolar ridge. On presentation, the area of concern (gum pad) typically is firm, pink, smooth swelling of the gingival, and usually seen as gingival hyperplasia. Considerably large-seized growths may elevate the upper lip. The teeth which have not emerged into the oral cavity are typically unaltered and can be visualized in MRI.¹⁵
- (b) **Congenital Gum Synechiae:** This rare malformation is identified by innate adherence between various parts of the oral apparatus. It leads to trouble in breathing soon after birth. Prompt treatment is advocated involving the excision of alveolar bands. If left untreated, it may result in TMJ ankylosis, restricted jaw movement and overall growth may also be interrupted (restricted feeding).¹⁶

Traumatic Lesion:

- (a) It occurs accidentally in our daily practice and the inflammation or swelling lasts only for a transient period. Factitious gingivitis (*Gingivitis artefacta*) is a self-inflicting injury of gingival that could be habitual, accidental, or psychological. Soft tissue injury by biting unknowingly, faulty brushing techniques, idiopathic trauma, tissue laceration while replacing orthodontic components, chewing abrasive food can result in a mucogingival recession or local inflammatory reaction. Management includes removal of irritation source, habit, rectification, and wound dressing. Psychological or psychiatric consultation may be beneficial in severe cases.^{17,18}

Gingival Diseases Due to Heredity:

- (a) **Hereditary Gingival fibromatosis (HGF):** This benign condition is marked by a gradually progressive gingival enlargement. The commonest variety, HGF, is usually familial and has an autosomal dominant mode of transmission. Another rare type has been referred to as elephantiasis gingiva or hereditary hyperplasia of the gums. It manifests as non-hemorrhagic, firm, leathery which advances slowly upon eruption of permanent dentition involving attached, marginal and interdental gingiva. This may be complicated by esthetic or

functional issues such as a malaligned tooth, delayed retention of primary teeth, and delayed eruption of a permanent successor. Removal of hyperplastic tissues by conventional gingivectomy may be a treatment modality.

Amalgam Tattoo: Although it is a very unusual condition, it is manifested as a grayish-blue-black appearance on the oral mucosa (typically on the lower gums) due to the accidental diffusion of dental amalgam into the soft tissues. Occasionally, it is misdiagnosed as melanoma.

Gingival Findings Associated with Systemic Disorders:

- (a) **Infectious mononucleosis:** Epstein-Barr virus (EBV) is the causative organism and it primarily affects children and young individuals. Patients chiefly complain of fever, sore throat, and enlarged lymph nodes, but malaise, fatigue, headache is also present. The abnormal findings include gingival bleeding, palatal petechiae, ulceration of the gingiva, and buccal mucosa (White, 1998).
- (b) **Chickenpox:** Varicella-zoster is the reason of chickenpox primarily affects individuals who are less than 15 years old. Although the location of lesions in the oral cavity is variable, palate, gingiva, and buccal mucosa are commonly affected.¹⁸⁻²⁰
- (c) **Herpangina:** The causative pathogen of herpangina is the Coxsackie group A virus. It characterized by plentiful small vesicles that further form small ulcers made up of a gray-floor and inflamed border. The location of these ulcers is quite predictable like the tongue, palates, oral mucosa, and the posterior pharyngeal wall. These ulcers are typically painless and recede within a few days to a week.¹⁸⁻²⁰
- (d) **Hand-foot and mouth disease:** The bulk of cases of hand, foot, and mouth disease can affect not only the infants but also the preschooler ranging from 5 months to 5 years of age. Both the Coxsackie group A as well as group B are implicated in this disease. The signs and symptoms are identical to that of herpangina but culminate in dysphagia due to sore mouth. This disease commonly heals and abates within 1-2 weeks.¹⁸⁻²⁰
- (e) **Wegner's granulomatosis:** This vasculitis originally presents with marked alterations in the gingiva. Erythematous enlargement of the gingiva associated with this disease is often described as

strawberry gingivitis.

- (f) **Kindlers syndrome:** This condition is characterized by the presence of poikiloderma, cutaneous neonatal bullae, acral atrophy, and photosensitivity. Oral lesions similar to desquamative gingivitis can also be part of the manifesting symptoms. Traditional non-surgical periodontal management can be useful for treating gingival manifestation.¹⁸⁻²⁰

Conclusion

Gingival diseases have a wide prevalence and these can affect all the age groups. However, the common myth is that gingival diseases affect only adults. Through this review, we reinforce that gingival diseases can be troublesome in children as well. Inadequate awareness about any signs and symptoms involving the gingiva in toddlers and young ones would jeopardize the periodontium in grown-up individuals. As prevention is better than cure, it is important to streamline patient education, and parent counseling to have regular pedodontic gingival assessment to maintain a healthy oral cavity in childhood.

Funding: Nil

Conflict of Interest: Nil

Ethical Approval: Approved.

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