

# Gingival Diseases in Childhood-A Review

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## Abstract

Children are exposed to various gingival diseases, similar to those found in adults, yet differ in some aspects. These diseases could be plaque or non-plaque induced, familial, or may be associated with a systemic condition. It is crucial to diagnose and manage gingival diseases as early as possible as they have the potential to further progress, causing a severe breakdown of periodontal support. Consequently, the final result may lead to tooth loss at an early age, which in turn will affect the nutrition and overall development of a pediatric patient. Therefore, greater emphasis is given to the prevention, early diagnosis, and treatment of gingival disease in children. As a dentist, it is necessary to be able to distinguish and differentiate all possible gingival conditions to successfully manage them. By establishing excellent oral hygiene habits in children, which will carry over to adulthood, the risk of periodontal disease is lowered. This paper will review various gingival conditions that are found in children, their main clinical features and management.

**Index terms**— gingival diseases in children, plaque induced gingivitis, non-plaque induced gingivitis, early diagnosis, pediatric gingivitis.

## 1 Introduction

Periodontal disease may have its origins in childhood. Studies confirm a high prevalence of gingival inflammation in children, which may progress to periodontitis, resulting in the loss of primary and permanent teeth. Therefore, promptly diagnosing and treating gingival diseases in childhood may reduce the risk of carrying forward the disease in adulthood. Gingival diseases affecting children may be broadly classified into Dental Plaque-induced and Non-plaqueinduced gingival diseases (table 1). 1

## 2 III.

Associated with nutritional deficiency:  
? Ascorbic Acid Deficiency Gingivitis

## 3 C. Modified by medications

4 I. Drug-induced gingival enlargement conditions (rare)  
? Pemphigus vulgaris? Kindler syndrome ? Lichen planus ? Allergic reaction ? Wegener's Granulomatosis

## 5 I. Gingival Abscess

Modified from Armitage GC: Development of a classification system for periodontal diseases and conditions, Ann Periodontol 4:1, 1999

Gingiva of children is different in many aspects. Gingiva of the primary dentition generally appears as pale pink, but less pale than that of an adult. 2 The marginal gingival is also more vascular and contains fewer connective tissue cells. 3 The thinner, more red appearingepithelium with a lesser degree of keratinization may

## 7 DENTAL PLAQUE-INDUCED GINGIVAL DISEASES

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40 be interpreted as mild inflammation. 3 The width of attached gingiva is less variable in the primary dentition,  
41 causing fewer mucogingival problems 3 ; however, the width increases with age. 4 Stippling in children usually  
42 appears at about 3 years of age without significant inter-arch difference. 5 Interdental papilla is broad bucco-  
43 lingually and narrow mesiodistally. 6 The junctional epithelium tends to be thicker of the primary dentition than  
44 the permanent. 7 Gingival sulcular depth ranges from 1-2 mm which is shallower than that found in adults. 8  
45 There are normal physiological changes associated with tooth eruption that may appear as gingival pathology  
46 and must be distinguished. The gingival prominence caused by the crown of an underlying erupting tooth is firm  
47 and pink, with mild inflammation from mastication; however an eruption cyst will present as a bluish or deep  
48 red enlargement of the gingiva over the erupting tooth 6 . The gingival margin of a newly erupted tooth appears  
49 rounded, edematous and reddened and may mimic gingivitis. This paper will present various dental plaque and  
50 non-plaque induced gingival diseases affecting children and adolescents.

### 51 6 II.

## 52 7 Dental Plaque-Induced Gingival Diseases

53 Chronic gingivitis is common in children and adolescents, where inflammation is generally limited to the marginal  
54 gingiva with undetectable loss of bone or connective tissue attachment 6 . The primary cause is dental plaque  
55 related to poor oral hygiene. 6 Clinical features include red linear inflammation, increased vascularization,  
56 swelling, and hyperplasia 9 . Bleeding and increased pocket depth are found less frequently in children than in  
57 adults, but may be observed in severe gingival hypertrophy or hyperplasia. 9 Calculus deposits are rarely seen  
58 in infants but may increase with age 6 ; however, children with cystic fibrosis have higher incidences of calculus,  
59 which may be caused by increased salivary calcium and phosphate concentrations 10 .

60 Plaque control procedures 11 in the primary dentition can be accomplished by rubber-cup coronal polishing (if  
61 no calculus is evident) or by selective supragingival scaling (if calculus is evident); however as permanent teeth  
62 erupts, addition targeted sub-gingival scaling may also be necessary. Oral hygiene measures should be instructed  
63 to parents and children in terms that both understand. The dynamic process of developing manual dexterity  
64 impacts the ability of a child to perform expected procedures. Children are encouraged to use a simple scrub  
65 technique; more refined brushing techniques can be introduced during adolescence. Flossing should be added  
66 to the home care routine as interdental contacts develop, and is usually not indicated in the primary dentition  
67 stage. Antimicrobial mouth rinses for chemical plaque control are not indicated in very young children because  
68 of the risk of ingestion.

69 Plaque induced gingival enlargement occurs due to prolonged plaque exposure which may be complicated by  
70 local factors like mouth breathing, or orthodontic appliances. 12 Clinically, it ranges from pale and fibrotic  
71 to red and friable. 12 There is localized or generalized enlargement of the interdental papilla and/or gingival  
72 margin. 12 Meticulous plaque control is required, and sometimes, gingivectomy or gingivoplasty may be indicated.  
73 12 Eruption gingivitis is a temporary type of gingivitis seen in young children during teeth eruption. 13 Tooth  
74 eruption itself does not cause gingivitis; in fact it is the inflammation associated with plaque accumulation around  
75 erupting teeth is common 7 . Eruption gingivitis is usually mild which requires no treatment other than improved  
76 oral hygiene. 13 Mouth breathing and lip incompetence may result in increased plaque and gingival inflammation  
77 which is often limited to the gingiva of the maxillary incisors due to frequent drying out of the gingiva. 11,14  
78 Treating the cause of mouth-breathing may resolve the problem for example gingivitis secondary to mouth  
79 breathing caused by allergic rhinitis can be treated by antihistamines 6 and incompetent lips can be corrected  
80 by orthodontic treatment.

81 Crowding gingivitis is due to irregular arrangement of the dentition, preventing self-cleansing of the mouth. It  
82 is worse in children who do not brush their teeth regularly. Oral hygiene instructions and orthodontic treatment  
83 can alleviate the gingivitis. 11 Gingival changes due to orthodontic appliances can occur within 1 to 2 months of  
84 appliance placement due to difficult plaque removal. 11 Changes are generally transient, rarely producing long-  
85 term damage to periodontal tissues. 11 Use of special toothbrushes (e.g. powered tooth brushes) and additional  
86 cleaning tools may be recommended for better plaque control 15 .

87 Pubertal gingivitis peaks at 9 to 14 years of age and generally subsides after puberty. 7 Hormonal changes  
88 during puberty accentuates the vascular and inflammatory response to dental plaque 9 and also alters reactions  
89 of plaque-microbes 16 that could explain this modified gingival response. Frequently, it presents as enlargement,  
90 bleeding and inflammation in interproximal areas without concomitant increase in plaque levels affecting both  
91 males and females. 17 It generally subsides after puberty however severe cases are treated by improving oral  
92 hygiene 13 , removing all local irritants 13 , restoration of carious teeth 13 and improving nutritional status (e.g.  
93 administration of 500mg of ascorbic acid orally for 4 weeks 19 ).

94 Diabetes mellitus Type 1 occurs more frequently in children and adolescents than Type 2 . Gingival  
95 inflammation and periodontitis are more prevalent and severe in affected children with poor metabolic control  
96 than in unaffected individuals. 20 Premature tooth loss and impaired immune response to oral flora occurs in  
97 severe cases. Treatment includes controlling diabetes, disease prevention 21 and early training and motivation of  
98 children to maintain efficient plaque control 21, 22 .

99 Leukemia is the most common type of cancer in children, and acute lymphoblastic leukemia is the commonest  
100 amongst them. It is accompanied by oral symptoms that include acute gingival enlargement, ulceration, bleeding

101 and infection. 23 These patients have low tissue-resistance to infection, owing to decreased circulating leukocyte  
102 count, which is further complicated by cytotoxic drugs (interfere with epithelial cell replication) that are used  
103 in the treatment of leukemia. Therefore, rigorous plaque control measures must be implicated both before  
104 commencing cytotoxic treatment and during medical treatment. 22,24 Gingivitis associated with vitamin C  
105 deficiency can lead to edematous and spongy gingiva, spontaneous bleeding, and impaired wound healing. 12 The  
106 underlying deficiency must be corrected, along with plaque control. 12 Drug-induced gingival enlargement can  
107 occur in children taking anticonvulsants (phenytoin, 25,26 valproate 26 ), calcium channel blockers (nifedipine  
108 26 , diltiazem 26 , verapamil 26 ), and immunosuppressives (cyclosporine A 27 ). Although complicated by  
109 increased plaque along the gingival margin, the features of this condition differ from that of chronic marginal  
110 gingivitis. 9 The clinical features are very similar irrespective of the drug involved. The first signs of change  
111 usually appears 3 to 4 months after drug administration. Enlargement appears mulberry-shaped, pink, firm and  
112 stippled in patients with good hygiene, however, in subjects with pre-existing gingivitis, or a poor standard of  
113 plaque control, the enlarged tissues shows classical signs of gingivitis 3 . To manage such enlargement, strict oral  
114 hygiene instructions and scaling must be implemented. 3 Severe cases inevitably need to be surgically excised  
115 and re-contoured (gingivectomy and flap surgery). 3 A follow-up program is essential to monitor plaque control  
116 and to detect any recurrence, in which case drug modification may be needed. 3

## 117 8 III.

### 118 9 Non-Plaque Induced Gingival Diseases

119 Primary herpetic gingivostomatitis is an acute infectious disease of the gingiva caused by herpes simplex viruses  
120 (HSV) Type-1 most commonly affecting children between 2-5 years of age. 28 Clinical features include febrile illness,  
121 headache, malaise, oral pain, mild dysphagia, and cervical lymphadenopathy 3,9,13,28,29 . Gingivitis is the most  
122 striking feature, with markedly swollen, erythematous, friable gums 3,13,29 The goal of treatment is to make the  
123 patient comfortable, and to prevent secondary infections or worsening systemic illness. Supportive management  
124 involves bed rest, eating a soft diet, and maintaining adequate hydration and treating pyrexia using paracetamol  
125 suspension. 3,29 Secondary infection of ulcers is prevented using chlorhexidine. 3 Systemic treatment includes  
126 antivirals (acyclovir) and analgesics (acetaminophen). Topical anesthetics may also be used; however, do not  
127 speed healing. 3,13,29 Candidiasis caused by candida albicans following a course of antibiotics or as a result  
128 of congenital or acquired immunodeficiencies. In neonates, infection can be contracted during passage through  
129 vagina. It is less common in children and is rarely associated with a healthy child. 30 It presents as raised,  
130 furred, white patches, which if removed leaves bleeding underlying surface. 13 Infants can be treated topically  
131 by a suspension of 1mL (100,000 U) of nystatin 4 times a day. Older children can be treated using clotrimazole  
132 troches or nystatin pastilles. Severe cases can be managed by systemic fluconazole (infants-suspension 6mg/kg or  
133 less per day; older children-100mg tablet for 14 days). 13 Catarrhal gingivitis (streptococcal gingivitis) is caused  
134 by hemolytic streptococcus. Clinical features include fever, headache, myalgia, and arthralgia 31 . The gingiva  
135 is painful, appears red, soft and friable, and tend to bleed spontaneously. Improved oral hygiene, mouthwashes  
136 and antibiotics are recommended for treatment. 31 .

137 Acute necrotizing ulcerative gingivitis (ANUG) is a broad anaerobic infection caused by fusiform bacteria,  
138 spirochetes, and other gram-negative anaerobic organisms. ??,29,32 Malnutrition, stress, lack of sleep are few  
139 predisposing factors. 29,32 It is common in young children in less-developed countries. ANUG is rapid in onset  
140 and very painful. "Punched out" ulceration and necrosis occur in the interdental papillae and marginal gingival,  
141 covered by yellowish-grey pseudomembranous slough. 3 Eventually, involve the alveolar crest and may progress  
142 to necrotizing ulcerative periodontitis in immuno-compromised individuals as recurrence is inevitable. Treatment  
143 include intense oral hygiene, professional plaque removal, mouthwash rinse (0.5% hydrogen peroxide -removal  
144 of necrotic tissues and 0.2% chlorhexidine-prevents plaque formation), antibiotics (penicillin or metronidazole),  
145 and NSAIDs for pain. 33 Congenital epulis a rare gingival tumor that occurs along the alveolar ridge in  
146 newborns, without additional congenital malformations or associated teeth abnormalities. Clinically presents  
147 as a smooth, welldefined erythematous mass arising from gum pad. Small lesions may regress and larger lesions  
148 must be resected, as they often interfere with airway and cause feeding difficulties. The un-erupted teeth are not  
149 affected usually. 34 Congenital gum synechia presents as unilateral or bilateral adhesions between the maxilla  
150 and mandible in the form of fibrous bands that makes feeding, swallowing and respiration difficult soon after  
151 birth. Early treatment is recommended which involves excision of alveolar bands. If not treated, it may result  
152 in TMJ ankylosis, restricted jaw growth and overall growth may also be affected (restricted feeding).

153 Traumatic lesions can be factitious, iatrogenic or accidental and can occur as a result of chemical physical  
154 or thermal injury. 37 Toothbrush abrasion due to faulty brushing technique is very common which presents as  
155 painful ulceration with surrounding erythematous halo. These may usually get superinfected by normal mixed  
156 flora of oral cavity when these ulcers may get covered with yellowish exudates. 33 Initial professional cleaning  
157 followed by cessation of toothbrushing for 7-10 days is recommended, during which child should rinse 2 times  
158 daily with 0.1% chlorhexidine. 33 The right brushing technique must also be taught to the child.

159 Factitious gingivitis (Gingivitis artefacta) is a self-inflicting physical injury of gingiva that could be habitual,  
160 accidental or psychological in origin. 3 , 38 The minor form is caused by rubbing or picking of the gingival with  
161 fingernail or abrasive foods while, the major form is more severe and widespread, involving deeper periodontal

## 10 CONCLUSION

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162 tissues. 3 Other areas of the mouth may be involved, as well as extra-oral injuries found on the scalp, face or  
163 limbs. Management includes removal of irritation source, habit correction, and wound dressings. 3,38 In major  
164 cases, psychological or psychiatric consultation may be advised. 3,38 Hereditary gingival fibromatosis is a rare  
165 overgrowth usually transmitted as dominant trait 40 . Enlarged gingival tissues are usually normal, pink, firm  
166 and leathery with little inflammation and involves attached, interdental and marginal gingiva. 39,40,41 There  
167 may be esthetic or functional problems, such as mal-positioning of teeth, prolonged retention of primary teeth and  
168 delayed eruption of permanent successors. 41 In addition, the hyperplastic region produces conditions favorable  
169 for accumulation of dental plaque causing secondary inflammatory changes. 41 Treatment include removal of  
170 hyperplastic tissues by conventional gingivectomy. 42 Strawberry gingivitis is gingival manifestation of Wegener's  
171 Granulomatosis, a necrotizing granulomatous vasculitis affecting upper and lower respiratory tract and kidney  
172 44 which may also affect pediatric age group45.

173 Oral manifestations include the gingiva exhibiting erythema and enlargement, typically described as "straw-  
174 berry gums". 43,46 Treatment include administration of immunosuppressives like prednisolone and cyclophosph-  
175 amide 43,44 for which child patient must be referred without delay for medical evaluation and management  
176 43 .

177 IV.

## 178 10 Conclusion

179 To summarize, the differences in the causation and pathogenesis of gingival diseases in children are as varied as  
180 their adult counterpart with similar clinical Gingival Diseases in Childhood-A Review presentations of gingival  
181 bleeding, pain and swelling. Nevertheless the importance of recognizing these gingival manifestations in childhood  
182 can give a clue towards an underlying pathology like nutritional deficiency, immunological disease or even  
183 a leukemic state. Therefore the thorough knowledge of gingival diseases in childhood and their treatment  
184 contributes not only towards better oral care but also augments a comprehensive general pediatric care of  
185 the individual. Kindler syndrome is an autosomal recessive disorder 47 that may present with oral lesions  
186 that are clinically consistent with desquamative gingivitis, along with Cutaneous neonatal bullae, poikiloderma,  
187 photosensitivity, and acral atrophy. 48 Traditional nonsurgical periodontal treatment can be beneficial for  
188 treating gingival manifestations. 47 Pericoronitis inflammation of gingival covering partially erupted tooth  
189 (most commonly third molars). 12 Food entrapment creates an ideal environment for bacterial growth leading  
190 the pericoronal flap to become inflamed and swollen. 12 The enlarged flap, traumatized by occlusion, is very  
191 painful. Debridement, chlorhexidine irrigation and antibiotics are used for management. 12 Gingival abscesses an  
192 acute, localized, painful lesion of marginal gingiva or interdental papilla, caused by an embedded foreign objects.  
193 12 Treatment is done by debridement, drainage and chlorhexidine irrigation. 12

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# 1

Table1-Gingival Diseases: Classification

Dental Plaque-induced Gingival Diseases

A. Gingivitis Associated with Dental Plaque Only

I.

Without local contributing factors:

- ? Chronic gingivitis
- ? Plaque-Induced gingival

II.

enlargement

With local contributing factors:

- ? Eruption gingivitis
- ? Mouth breathing
- ? Crowding gingivitis
- ? Gingival Changes Related to

Orthodontic Appliances

Non-plaque-induced Gingival Diseases

A. Gingival diseases of Viral origin

- ? Primary Herpetic

Gingivostomatitis

B. Gingival diseases of Fungal origin

- ? Acute Candidiasis (Thrush, Candidosis, Moniliasis)

? Linear gingival erythema

C. Gingival diseases of Bacterial origin

? Acute necrotizing ulcerative gingivitis (ANUG)

? Streptococcal infection (Catarrhal gingivitis)

D. Congenital gingival Anomalies

? Congenital gum synechiae

? Congenital epulis

Figure 1: Table 1 :



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## 10 CONCLUSION

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