

Acute Generalized Gingivitis with Spontaneous Bleeding and Oral Ulceration in a 10-Year-Old-A Case Report

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ABSTRACT

Background: Gingivitis in children is commonly associated with dental biofilm and is generally reversible. However, severe gingival inflammation accompanied by spontaneous bleeding, fever, oral ulceration, and poor nutritional status warrants evaluation for underlying viral, necrotizing, hematologic, or nutritional disorders. **Case Presentation:** A 10-year-old girl presented with gingival swelling of one-week duration, preceded by fever and associated with spontaneous bleeding during tooth brushing and sleep. Pinpoint ulcerations were observed on the labial mucosa and lips. Intraoral examination revealed generalized reddish-pink, swollen, and hyperemic gingiva with loss of stippling, rolled gingival margins, bulbous papillae, and bleeding on probing. The patient's height and weight were 161 cm and 29 kg, respectively, corresponding to a calculated body mass index of approximately 11.2 kg/m². In view of the clinical presentation and nutritional status, physician assessment was advised. Panoramic examination did not reveal generalized alveolar bone loss. Based on the clinical findings, a diagnosis of acute generalized gingivitis was established. Complete hand scaling, oral-hygiene instruction, chlorhexidine mouth rinse, vitamin C supplementation, multivitamin supplementation, and physician referral were advised. Follow-up photographs obtained at 10 and 25 days demonstrated marked clinical resolution of the gingival inflammation. **Conclusion:** Severe generalized gingival inflammation in a child, particularly when accompanied by spontaneous bleeding, fever, oral ulceration, or poor nutritional status, requires prompt local biofilm control and consideration of systemic and nutritional modifiers. Clinical improvement following scaling and supplementation should not, in isolation, be interpreted as confirmation of a specific vitamin deficiency.

Keywords

Gingivitis, Child, Gingival Hemorrhage, Oral Ulcer, Vitamin C Deficiency.

Introduction

Gingivitis is the inflammation confined to the gingiva, clinically expressed as redness, edema, altered contour and bleeding without periodontal attachment loss. Dental biofilm is the major etiologic factor in children, while eruption, puberty, local plaque-retentive factors, nutrition and systemic conditions can modify the inflammatory response [1-5].

Spontaneous gingival bleeding accompanied by fever and oral ulceration often requires a broader differential diagnosis that includes primary herpetic gingivostomatitis, necrotizing gingivitis,

hematologic disease and vitamin C deficiency [6-10]. This report presents an acute generalized gingivitis case in a 10-year-old child with a markedly low calculated BMI and substantial clinical improvement following scaling, oral-hygiene measures and adjunctive supplementation.

Case Presentation

Patient information

A 10-year-old female child reported with the chief complaint of swollen gums for the past one week. Her parents initially noticed swelling in the mandibular anterior region, with ulceration around the gingiva, mouth and lips. A history of fever four to five days earlier was reported. Gingival bleeding occurred during brushing and spontaneously during sleep. Child was on paracetamol for fever the previous week which was administered as and when

needed. Parents also reported that the child was having difficulty while eating and also reduced appetite for the past one week. The child was not taking any long-term medication and had no reported systemic illness, hospitalization, surgery or drug allergy.

This was her first dental visit. Prenatal, natal, postnatal and family histories were noncontributory. She brushed once daily with fluoridated toothpaste for two to three minutes using a circular technique and used no additional oral-hygiene aids. The recorded height was 161 cm and weight was 29 kg, giving a calculated BMI of 11.2 kg/m².

Clinical findings

Extraoral examination showed an apparently symmetrical face, a slightly convex profile, normal temporomandibular joint movements and no clinically enlarged lymph nodes. Intraoral soft tissue examination revealed generalized reddish-pink gingiva with absence of stippling, soft and edematous, swollen hyperemic consistency, rolled margins and bulbous interdental papillae. Bleeding on probing was elicited. Pinpoint ulcerations were present on the labial mucosa and upper and lower lips.

The child was in the mixed-dentition stage. Dental caries was recorded in tooth 46 and pit-and-fissure caries in 16 and 26. Grade II mobility was recorded in 84 and grade III mobility in 73 and 74, interpreted as preshedding mobility.

Diagnostic assessment

The provisional diagnosis was acute generalized gingivitis, dental caries in 16, 26 and 46, and preshedding mobility in 73, 74 and 84. A panoramic radiograph was obtained in order to verify periodontal involvement. The radiograph showed mixed dentition and developing successors without evidence of bone loss.

The differential diagnosis included primary herpetic gingivostomatitis because of preceding fever and oral ulceration; necrotizing gingivitis because of bleeding and ulceration; leukemia or another hematologic disorder because of spontaneous bleeding; and nutritional deficiency because of the low recorded weight and possible dietary inadequacy. The absence of punched-out necrotic papillae, pseudomembrane, fetor and severe gingival pain argued against necrotizing gingivitis. No drug history was present to support drug-induced gingival enlargement. Neither leukemia



Figure 1: Pretreatment clinical photographs showing generalized gingival erythema, edema, altered contour and anterior involvement.



Figure 2: Pre-operative panoramic radiograph. No obvious generalized alveolar bone destruction is visible

nor a vitamin deficiency can be excluded without hematologic investigations.

Therapeutic intervention

Day-1 - Complete supra and subgingival hand scaling was done. The child and parent received oral-hygiene maintenance instructions and dietary counselling. Vitamin C chewable preparation (Limcee - 500 mg contains Ascorbic acid 100 mg, Sodium ascorbate 400 mg) twice daily for 15 days, multivitamin tablet/capsule (A-Z Gold contains gelatin, omega-3-fatty acids, vitamins, minerals, alpha lipoic acid, antioxidants) once daily for 30 days and chlorhexidine mouth rinse (5ml in 1:2 dilution) once daily was prescribed to the child. Referral to a physician for evaluation of possible vitamin deficiency and the low BMI was advised. Composite resin was planned for 16, 26 and 46 along with wait and watch for natural exfoliation of the mobile primary teeth.



Figure 3: Intraoral photographs – immediate post-op following complete hand scaling.

Follow-up and outcome

At the 10-day review, full mouth supra and sub gingival ultrasonic scaling was done, the photographs showed a marked reduction in gingival erythema, edema and reduced bleeding while brushing , also the child’s appetite has increased and she is facing no disturbances while eating. At 25 days, the gingival tissues appeared substantially healthier, with improved marginal contour and further resolution of inflammation. The follow-up pictures did

not contain plaque or gingival bleeding.

Clinical Timeline

Time	Finding	Management/outcome
Initial visit history	One-week gingival swelling, spontaneous bleeding, recent fever and oral ulceration	Clinical and radiographic examination
Initial clinical findings	Generalized acute gingival inflammation; dental caries in 16, 26, 46; pre-shedding mobility in 73, 74, 84	Diagnosis of acute generalized gingivitis; physician referral advised
Initial visit-treatment	Inflamed gingiva with local deposits	Complete hand scaling, OHI, chlorhexidine mouth wash , vitamin C and multivitamin prescribed
10 days follow-up	Marked clinical reduction in gingival inflammation	Ultrasonic scaling and continued preventive care
25 days follow- up	Further improvement in gingival color and contour	Maintenance and systemic/ nutritional follow-up advised

Review of Literature

Pediatric gingivitis comprises a heterogeneous group of conditions ranging from plaque-induced inflammation to eruption gingivitis, puberty-associated gingivitis, nutritional deficiencies, viral infections, necrotizing periodontal diseases, and systemic disorders such as leukemia, highlighting the importance of a comprehensive differential diagnosis in children [1]. The current classification proposed by Chapple et al. defines gingivitis as bleeding on probing affecting ≥10% of sites in the absence of clinical attachment loss or radiographic bone loss and emphasizes the influence of local and systemic modifying factors on disease expression [2].

Among systemic modifiers, nutritional deficiencies have gained increasing attention. Tada and Miura demonstrated a significant association between reduced vitamin C levels and poorer periodontal parameters, although most available evidence

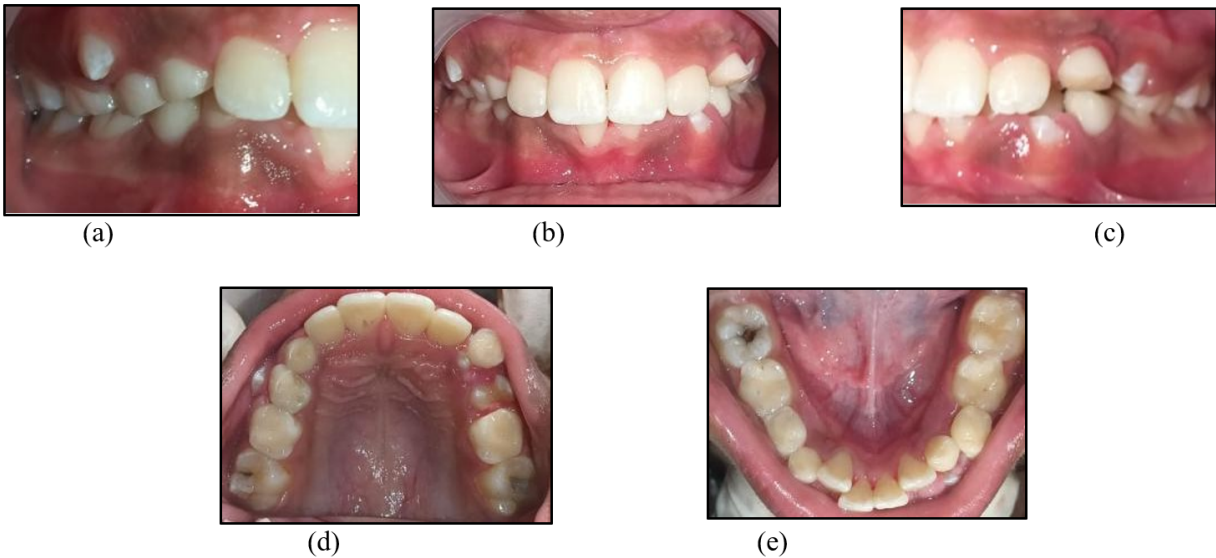


Figure 4: Follow-up photographs at 10 days (a-e).

was observational and derived from adult populations [3]. Subsequently, Ustianowski et al. highlighted the biological roles of vitamins C and D in collagen synthesis, antioxidant defense, immune regulation, and periodontal wound healing, providing to be an important factor for their role in gingival health [4]. Meher et al. reported resolution of gingival enlargement following vitamin D supplementation in a laboratory-confirmed deficient child, suggesting that nutritional correction may serve as an adjunct to conventional periodontal therapy [5]. However, El-Sayed et al. concluded that despite promising findings, the available evidence remains heterogeneous and insufficient to recommend vitamin supplementation as a substitute for effective plaque control [6].

Several recent pediatric studies have further emphasized the relationship between nutritional status and gingival health. Lešić et al. associated pediatric vitamin C deficiency with gingival petechiae, spontaneous bleeding, ulceration, and impaired wound healing, reinforcing the need to consider nutritional deficiencies in children presenting with unexplained gingival bleeding [7]. In a large epidemiological study involving 3,558 children, Olczak-Kowalczyk et al. identified poor oral hygiene, dental caries, and diet-related factors as significant determinants of gingivitis [8]. Their subsequent population-based study also demonstrated that children receiving vitamin D supplementation had lower odds of gingival bleeding, although the cross-sectional design precluded causal inference [9]. More recently, the European Federation of Periodontology (EFP)–European Academy of Paediatric Dentistry (EAPD) consensus report emphasized age-specific periodontal assessment and recommended evaluation of systemic modifying factors whenever gingival inflammation appears disproportionate to local plaque accumulation [10].

The literature also highlights the importance of excluding serious systemic diseases before attributing gingival inflammation solely to plaque or nutritional deficiencies. A systematic review and meta-analysis by Angst et al. demonstrated a high prevalence of gingivitis among children with leukemia, emphasizing that severe gingival inflammation may represent an early manifestation of hematological disease [11–13]. Similarly, Chaluvraj et al. reported generalized bleeding gingiva as the presenting feature of laboratory-confirmed vitamin C deficiency in a child, supporting medical referral when nutritional deficiency is suspected [14]. Coppola et al. further identified primary herpetic gingivostomatitis as an important differential diagnosis in children presenting with fever, painful oral ulcerations, and diffuse gingival inflammation [15].

Recent systematic reviews and case reports continue to reinforce this multidisciplinary approach. Silveira et al. documented gingivitis, mucositis, and lymphadenopathy as common oral manifestations of pediatric leukemia, whereas Sethi et al. demonstrated the critical role of dental practitioners in recognizing atypical gingival enlargement and referring children for medical evaluation, ultimately leading to the diagnosis of vitamin C deficiency [16,17]. Likewise, Kirubakaran et al. emphasized that spontaneous gingival bleeding and ulceration disproportionate

to plaque accumulation should prompt complete blood count evaluation to exclude neutropenia and other hematological disorders [18]. Pavlidou et al. reported hemorrhagic gingivitis as a characteristic manifestation of pediatric scurvy while noting that dental plaque may exacerbate the clinical presentation [19]. Finally, the systematic review by Tsilingaridis et al., involving 269 studies, estimated that dental biofilm-induced gingivitis affects approximately half of all children and adolescents worldwide and confirmed that effective plaque control, supervised toothbrushing, oral hygiene reinforcement and patient education remain the cornerstone of management despite the influence of systemic and nutritional modifying factors [20].

Discussion

Dental plaque is the primary etiologic factor responsible for gingival inflammation in children and adolescents. Plaque is a structured microbial biofilm that develops on tooth surfaces within hours after cleaning and, if left undisturbed, matures into a complex community capable of eliciting a host inflammatory response [11]. In pediatric patients, several local and systemic factors—including erupting teeth, mixed dentition, inadequate oral hygiene practices, malocclusion, orthodontic appliances, nutritional deficiencies and socioeconomic influences—may enhance plaque retention and increase susceptibility to gingival inflammation [1,20]. The recent EFP–EAPD consensus also emphasizes that although plaque-induced gingivitis is the most common periodontal condition in children, careful evaluation of modifying factors is essential because the pediatric periodontium differs biologically from that of adults [10].

With prolonged plaque accumulation, mineralization by salivary calcium and phosphate ions results in calculus formation. Although calculus itself is not the primary etiologic agent, its rough surface promotes further plaque retention, impedes effective oral hygiene and perpetuates gingival inflammation [11]. Consequently, plaque and calculus act synergistically to sustain chronic inflammatory changes, making complete mechanical debridement the cornerstone of treatment. In the present case, generalized plaque deposits together with calculus accumulation were observed clinically, explaining the extensive gingival erythema, edema, spontaneous bleeding and bleeding on probing. The marked improvement following complete hand scaling followed by ultrasonic scaling which further supports the predominant role of local biofilm in disease initiation and progression.

Gingivitis represents a reversible inflammatory response confined to the gingival tissues without loss of periodontal attachment or alveolar bone [2]. Clinically, affected children exhibit erythema, edema, loss of stippling, enlarged interdental papillae and bleeding on gentle probing [2]. The panoramic radiograph in the present case demonstrated no generalized alveolar bone destruction, confirming that the inflammatory process remained limited to the gingiva. This finding, together with rapid clinical resolution following plaque removal, supports the diagnosis of acute generalized plaque-induced gingivitis rather than destructive periodontal disease.

Nevertheless, the severity of inflammation in the present child appeared disproportionate to the amount of local deposits alone. The presence of spontaneous bleeding, preceding fever, oral ulcerations and a markedly low body mass index suggested that systemic or nutritional modifiers could have amplified the inflammatory response. Current pediatric periodontal recommendations advocate that children presenting with severe gingival inflammation associated with constitutional symptoms should undergo comprehensive medical evaluation in addition to local periodontal therapy [10]. Accordingly, physician referral was advised in the present case for assessment of nutritional status and possible underlying systemic conditions.

Several published reports describing children with severe generalized gingival inflammation have adopted management strategies similar to those used in the present case. Chaluvarej et al. reported a child presenting primarily with diffuse gingival enlargement and spontaneous bleeding that was subsequently diagnosed as vitamin C deficiency after medical investigations [14]. Their management included physician referral, dietary correction and vitamin C supplementation in addition to meticulous plaque control. Similarly, Sethi et al. described a toddler with hypertrophic bleeding gingiva in whom dental recognition of the condition prompted medical referral and laboratory confirmation of scurvy before nutritional therapy was initiated [17]. Therefore, although vitamin C supplementation was prescribed empirically because of the child's poor nutritional status and low BMI, improvement following therapy cannot be interpreted as proof of vitamin deficiency. Rather, the favorable outcome most likely reflects the combined effects of professional debridement, improved oral hygiene, reduction of microbial biofilm and supportive nutritional care.

Recent epidemiological evidence further supports this conservative approach. Olczak-Kowalczyk et al. demonstrated that gingivitis in children is strongly associated with poor oral hygiene, dental caries and dietary habits, emphasizing plaque removal and oral hygiene reinforcement as the primary therapeutic interventions [8]. Likewise, the recent systematic review by Tsilingaridis et al. identified dental biofilm as the principal determinant of gingivitis in children and adolescents and concluded that supervised tooth brushing, professional prophylaxis, parental education and regular maintenance remain the most effective preventive and therapeutic measures [20]. These recommendations closely parallel the management protocol followed in the present case, which consisted of complete hand scaling, oral hygiene instruction, dietary counselling, chlorhexidine mouth rinse and ultrasonic scaling close follow-up over the course of 3 weeks and more.

An important aspect of this case was establishing an appropriate differential diagnosis because spontaneous gingival bleeding associated with fever and oral ulceration is not pathognomonic of plaque-induced gingivitis. Primary herpetic gingivostomatitis was considered because the child reported fever preceding the onset of oral lesions. Typically, primary herpes simplex virus infection presents with diffuse painful gingivitis, multiple vesicles that

rupture to form ulcers, cervical lymphadenopathy and difficulty in eating or drinking [15]. Although fever and ulceration were present in our patient, vesicular lesions, cervical lymphadenopathy and severe oral pain were absent and the lesions resolved rapidly following periodontal therapy without antiviral treatment, making primary herpetic gingivostomatitis less likely.

Necrotizing gingivitis was another important differential diagnosis because it commonly presents with spontaneous bleeding and ulceration. However, the characteristic punched-out interdental papillae, pseudomembranous necrosis, severe pain and fetid oral odor were absent clinically [1]. Consequently, necrotizing gingivitis was excluded based on the clinical presentation.

Hematological disorders, particularly leukemia, also warrant consideration in children presenting with severe gingival bleeding. Gingival enlargement, spontaneous hemorrhage, petechiae and oral ulceration may be the earliest manifestations of acute leukemia before systemic symptoms become evident [13,16]. Likewise, neutropenia may present with gingival inflammation, painful oral ulceration and recurrent infections [18]. Although the present patient exhibited spontaneous bleeding, no generalized lymphadenopathy, pallor, persistent fever, bruising or radiographic evidence suggestive of malignant infiltration was identified. Nevertheless, complete blood count and hematological evaluation would have strengthened diagnostic certainty and should be considered whenever gingival bleeding appears disproportionate to local plaque accumulation.

Nutritional deficiency, particularly vitamin C deficiency, also formed an important differential diagnosis because the child exhibited a markedly low body mass index together with spontaneous gingival bleeding and oral ulceration.

Vitamin C plays a fundamental role in collagen synthesis, capillary integrity and wound healing, and deficiency may produce swollen friable gingiva, petechiae, gingival hemorrhage and delayed tissue repair [3,4,7]. Recent pediatric case reports have demonstrated that laboratory-confirmed scurvy often presents initially as severe gingival inflammation before systemic manifestations become apparent [14,17,19]. However, vitamin deficiency remained only a clinical possibility rather than a definitive diagnosis. The child's response to vitamin supplementation therefore should be interpreted cautiously.

Overall, the successful outcome in the present case highlights that comprehensive management of acute generalised gingivitis in mixed dentition should extend beyond mechanical plaque removal alone. Careful history taking, detailed clinical examination, exclusion of potentially serious systemic disorders, reinforcement of oral hygiene practices, nutritional counselling, physician referral when indicated and close clinical follow-up together contribute to successful resolution. At the same time, this case underscores the importance of avoiding attribution of spontaneous gingival bleeding solely to plaque accumulation until hematologic, infectious and nutritional conditions have been reasonably excluded.

Conclusion

Acute generalized gingivitis in a child can present with dramatic swelling and spontaneous bleeding yet respond rapidly to meticulous debridement and improved oral hygiene. Fever, oral ulceration and low nutritional status require a structured differential diagnosis and medical referral. Vitamin supplementation may be used when clinically indicated, but a specific deficiency should not be claimed without appropriate laboratory or physician confirmation.

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