Nonsyndromic localized aggressive periodontitis of primary dentition: A rare case report

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Abstract

Periodontitis is an inflammatory disease of the gingiva and tissues of the periodontium. It is characterized by pocket formation and destruction of supporting alveolar bone. Periodontal diseases of aggressive nature are not very common in children. They are usually associated with systemic conditions. The present case report is of a 5-year-old male child who reported with rapid attachment loss and bony defects of the gingiva and supporting structures. His family and medical history gave no contribution for the diagnosis. Blood investigations did not reveal any abnormality. The microbial examination of culture revealed the presence of periodontal pathogen *Aggregatibacter actinomycetemcomitans*. The treatment objective in the present case was to prevent the further progress of the condition, restore esthetic and function in the child which would psychologically benefit the child.

Keywords: *Aggregatibacter actinomycetemcomitans*, furcal involvement, localized aggressive periodontitis

Introduction

In healthy children, periodontitis in the primary dentition is a distinct clinical entity termed pubertal periodontitis.[1] The etiology is usually unknown. *Aggregatibacter actinomycetemcomitans* has been the organism found in the gingival pockets of children with this condition.[2] The amount of bone destruction is usually not correlated with amount of local irritants present. The rate of destruction is 3–4 times faster in children. The onset of prepubertal localized aggressive periodontitis is during or immediately after the eruption of the primary teeth. The systemic conditions affecting the periodontium include leukocyte adhesion deficiency, hypophosphatasia, cyclic neutropenia, Chediak–Higashi syndrome, leukemia, Papillon–Lefevre syndrome, and diabetes mellitus.[3] Genetic predisposition for the periodontal disease has been observed.[4,5]

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Case Report

A 5-year-old male child reported to the department after referral from a local dentist due to severe attachment loss of gingiva. The child was accompanied by parents and his medical history did not reveal any abnormality. Detailed history, extraoral examination, intraoral examination, and the radiographic evaluation were done. The child has shown symptoms of bleeding from gingiva for the past 2 months and unable to chew food due to pain. The clinical oral examination revealed full primary dentition, little plaque, moderate gingival inflammation, severe attachment loss in relation to 52, 53, 62, 63, 54, 55, 64, and 65 and presence of supernumerary tooth (mesiodens, 51 and 61 region). Bleeding on probing was seen and periodontal pockets measured 5 mm around the first primary molars with furcal involvement [Figure 1]. The panoramic radiograph revealed severe generalized vertical and horizontal bone loss [Figure 2]. Underlying systemic condition was evaluated by complete medical evaluation. The complete blood count, creatinine, alkaline phosphatase, coagulation factors, and T4 lymphocyte counts were analyzed and found to be within normal limits. Absolute monocyte and neutrophil counts and erythrocyte sedimentation rates were slightly elevated. Microbiological evaluation was performed by collecting unstimulated saliva, and the microorganisms were identified for aerobic and anaerobic flora. Tissue

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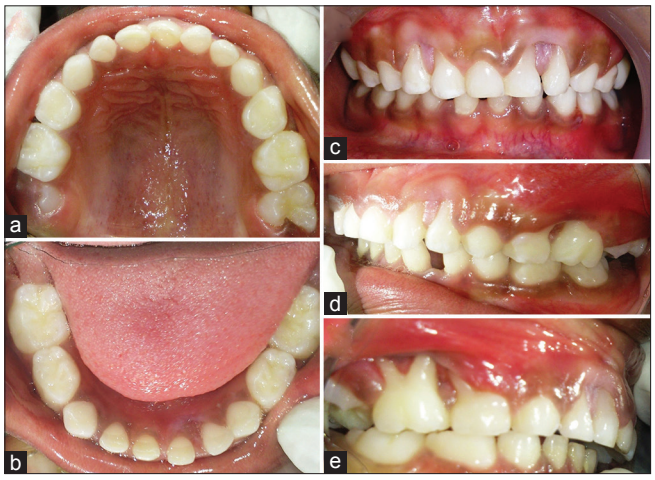

Figure 1: Preoperative – (a) Maxillary arch showing the presence of mesiodens. (b) Mandibular arch. (c) Gingival recession in relation to 52, 53, 61, 62. (d) Left side occlusion showing recession in I molar area. (e) Right side occlusion showing furcal involvement and severe gingival recession

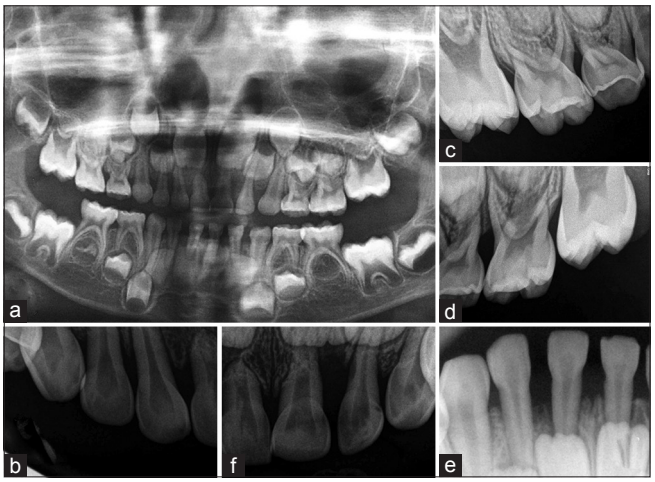

Figure 2: Preoperative – (a) Orthopantamograph. (b-f) Intraoral periapical radiographs revealing bone loss

biopsy was done in the area of severe attachment loss, i.e., 54, 55, 64, and 65. The samples were cultured in brain–heart infusion agar, trypticase soy agar, sheep blood agar, dextrose starch agar, KOH mount and incubated at 37°C in anaerobic chamber with an atmosphere of 80% N, 10% H, and 10% CO_2 for 72 h. This revealed the presence of *A. actinomycetemcomitans* and *Porphyromonas gingivalis* from tissue biopsy confirming prepubertal localized aggressive periodontitis.

Treatment plan

The child was cooperative, and thorough oral prophylaxis and root planing were done. Systemic antibiotics of amoxicillin (50 mg/kg/day) (body weight in three divided doses) along with metrogyl 30 mg/kg/day for 15 days were given. Tetracyclines are the proven drugs in periodontal therapy but have been ruled out in the present case as the child is only 5 years old. Stringent measures for maintaining oral hygiene with tooth brushing and 0.12% chlorhexidine three times a day were advised under parental supervision. Further topical application of metronidazole in chlorhexidine (Rexidin-M gel) base was advised for 2 weeks. Vitamin B complex syrup was also included. Regular checkups and motivation were done for 1½ years. The response was good and can be appreciated in the clinical pictures [Figure 3].

Discussion

The most striking feature of localized aggressive periodontitis is the severity of bone loss in affected areas mainly the molars and incisors. In the present report, primary dentition is affected. In contrast, it has been suggested more recently that localized periodontitis presenting in children is not only associated with *A. actinomycetemcomitans* but is also likely result of polyinfection by a mixture of bacteria (especially *P. gingivalis* and intermedia) similar to adult or chronic disease.[6] Other species likely to be involved include *Capnocytophaga* species, *Eikenella corrodens*, *Campylobacter rectus*, *Spirochaetes,* and *Eubacterium* species.[6,7] Other reports have stated that *P. gingivalis* and *Treponema denticola* were not detected in periodontally healthy children, but several putative periodontal pathogens can colonize early in childhood.[8] Alterations in immunologic factors such as immunoglobulins are known to be present in aggressive periodontitis. Immunoglobulins appear to be influenced by both genetic and environmental factors and have important protective disease limiting effects in aggressive periodontitis patients.[9-13] Human immunoglobulin G (IgG) antibody molecules are categorized into four subclasses designated as IgG1-4. Most of the antibody reactive with *A. actinomycetemcomitans* are specific for high molecular weight lipopolysaccharide and are of the IgG2 subclass. This antibody response appears to be protective as early-onset periodontitis patients having high concentrations of antibody reactive with *A. actinomycetemcomitans* lipopolysaccharide have significantly less attachment loss (a measure of disease severity) than patients who lack this antibody.[9,10] Some authors have observed that extracted teeth in patients with aggressive periodontitis exhibit thin cementum areas and have suggested that this alteration may be a major determinant of disease progression due to the increased risk of pathogen invasion.[14,15] In the present case report, thorough oral prophylaxis and root planing along with systemic antibiotic therapy with proper maintenance gave very good response which required no extraction, and Figure 3 shows the result of good oral care taken by the child as well as parents.

Conclusion

Successful outcome of localized aggressive periodontitis depends on early diagnosis, antibiotic therapy, followed

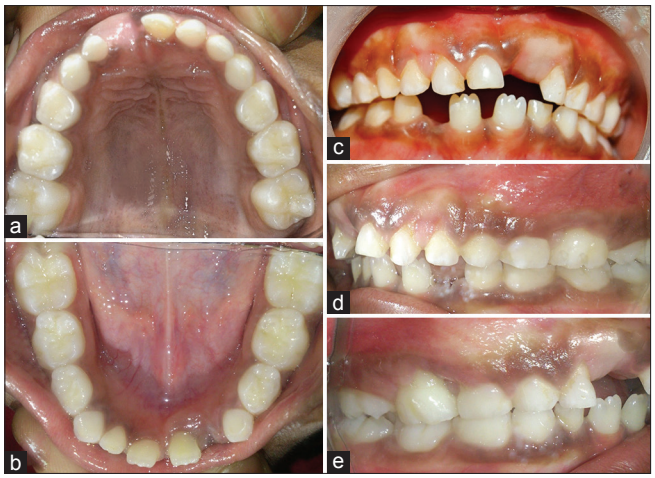

Figure 3: Postoperative – (a) Exfoliated 51, maxillary arch. (b) Mandibular arch. (c) Permanent mandibular incisors showing improvement in gingival condition. (d and e) Improvement in the attachment and gingival health

by maintenance of meticulous oral hygiene providing an environment free of infection. The degree of localized lesions and their reversal to predict success depends on the systemic disease which can be a major contributing factor. Hence, early diagnosis and combined antibiotic therapy with regular periodontal examination for 1½ years helped in managing the localized aggressive periodontitis.

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Conflicts of interest
There are no conflicts of interest.

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