



CASE REPORT

Diagnosis of Molar-Incisor Pattern Periodontitis in a Paediatric Patient

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Abstract

This is a case report of a 4 year old female child with deciduous dentition exhibiting rare clinical signs of gingival inflammation, clinical attachment loss and bone resorption around the primary lower first molars and upper central incisors bilaterally resulting in bone loss. Previously this condition was referred to as Localised Aggressive Periodontitis or Juvenile Periodontitis. The recent 2017 classification identifies this as Stage III Grade C periodontitis of Molar Incisor pattern. There was no syndrome or systemic condition associated.

Keywords: Aggressive periodontitis; Bone resorption; Deciduous tooth; Gingivitis; Inflammation

Introduction

Aggressive forms of periodontal disease are prevalent in most parts of the world with an incidence of 1 – 3% in African-Americans and 0.1 – 0.2% in Caucasians, 0.5 – 1% in Hispanics and South Americans and 0.4 – 1% in Asians.¹ The incidence of such a pathology in primary dentition is however rare. An epidemiological study carried out in the United States of America arrayed the incidence of 0.2 to 0.5 % in children and young adults of 14 to 17 years with severe Clinical attachment loss (CAL) on multiple teeth.² The prevalence of aggressive periodontitis in deciduous dentition is as low as 0.06%.³ When encountered, rapid loss of attachment in the primary dentition is often associated with immune system related conditions / syndromes such

as Papillon Lefevre Syndrome, leucocyte adhesion deficiency, cyclic neutropenia, Chediak Higashi Syndrome, Lazy leukocyte syndrome etc.⁴ Cases of rapid attachment loss in an otherwise healthy child are rare and only sporadically reported in literature.

The 1999 Classification of Periodontal diseases replaced the term ‘Localised Juvenile Periodontitis’ and ‘Pre-pubertal Periodontitis’ with ‘Localised Aggressive Periodontitis’. The 2017 Classification of Periodontal Diseases substituted the term ‘Localised Aggressive Periodontitis’ with Periodontitis of ‘Molar - Incisor Pattern’, which is further described by the stage or severity and grade, that is rapidity of progression.⁵ Periodic changes in nomenclature and classification may have further reduced the chances of accurately measuring the occurrence of

rapid loss of attachment around primary dentition in otherwise healthy children.

It is important that every such case is diagnosed, documented and published for future references.

Case presentation

A female child of 4-years and 3-months reported to the Paediatric Dentistry Department at the Dental and Maxillofacial Centre, Bahrain Defense Force Hospital in May 2016. The child's father reported bleeding from gums on brushing since 4 months and a swelling in the gums of teeth in the upper anterior region for the past one-month. However, no pain was experienced. There was no significant history of systemic disease, drug allergy or trauma to the upper anterior region. On extra-oral examination, no abnormalities were detected. Intra-oral examination revealed caries free deciduous dentition with plaque deposits. Tendency to bleed with slight pus discharge at the first primary molars in all the four quadrants was also noted. The tooth 61 had grade II mobility with a diffused gingival swelling. The swelling was soft and tender on palpation, but not fluctuant. The overlying mucosa was inflamed but not ulcerated. The tooth exhibited no discoloration or pain clinically. The upper standard occlusal radiograph showed angular bone loss around 51, 52, 61, 62 extending to the middle third of the root. At this juncture the differential diagnosis of the presenting case included pre-pubertal periodontitis or localized aggressive periodontitis, nutritional deficiencies, juvenile onset diabetes mellitus, peripheral blood leucocyte abnormalities and Papillon-Lefevre syndrome.

The child was recalled after one week for further evaluation and review of the results of blood examination, that is, the complete blood count (CBC) and biochemistry studies. The Alkaline phosphatase level was 260 IU/L (healthy children: 111 to 277 IU/L).⁶ The neutrophil count was 39.60% (normal range: 40 – 70%).

The child weighed 17.7 kg and was 99 cm height with Body Mass Index (BMI) of 18.09 kg/m². These findings were found to be within normal limits, and did not suggest any nutritional deficiency. Fasting blood sugar and differential leucocyte count were within normal range, ruling out juvenile

onset diabetes mellitus and leucocyte deficiency, respectively. Patients with leucocyte adhesion deficiency, weak leucocyte chemotaxis or faulty intra-lysosomal killing show signs of recurrent bacterial infections and absence of suppuration.⁷ Hence the absence of these symptoms in the patient coupled with no previous history of recurrent bacterial infections, ruled out the possibility of leucocyte function abnormalities. The absence of palmar and plantar hyperkeratosis ruled out Papillon-Lefevre syndrome. Finally, the presence of plaque deposits and gingival inflammation with angular bone loss in an otherwise healthy patient suggested the provisional diagnosis of localised aggressive periodontitis.⁷

To corroborate the initial clinical findings of the paediatric dental clinic, the patient was examined by a periodontist who subsequently participated in treatment planning and management thereafter. The patient was very anxious initially, however this was managed by the Paedodontist with good communication, tell-show-do technique and positive reinforcement which helped her cope in future visits. The dental chair was placed in a semi-supine position to facilitate visibility and access while keeping the apprehensions of the patient to a minimum.

There was generalized profuse bleeding on probing (UNC 15 probe) along the 7mm pocket in the tooth 84 with suppuration. There were pockets of 5mm in tooth 54 and 74 on the buccal aspect. The final diagnosis of localised aggressive periodontitis was made considering full mouth periodontal charting, radiographic examinations, and blood examinations. A treatment plan was formulated to arrest the progress of periodontal attachment loss. Treatment protocol included reinforcement of oral hygiene instructions, local debridement by scaling and root planing along with adjuvant antimicrobial therapy⁸ (Table 1).

Root surface debridement was performed in all the first primary molars and upper central incisors under topical local anesthesia. Oral hygiene instructions were given. Specifically, Fone's technique was demonstrated and use of a soft toothbrush was advised. Antibiotic therapy prescribed was:

Table 1: Protocol for dental treatment in young children with Generalized Aggressive Periodontitis.⁷

1. Extraction of hopeless teeth with severe bone loss exposing 2/3rd of root and grade II mobility
2. Monthly supragingival and subgingival curettage and debridement of remaining teeth for 1 year.
3. Systemic antibacterial medication like amoxicillin (50 mg/kg/day, divided into 3 doses) + metronidazole (30 mg/kg/day) for 10 days
4. Tooth brushing and mouthrinse using 0.12% chlorhexidine rinse 3-times a day for 3 months (depending on the patient's age)
5. Restoring the function and aesthetics with prosthesis when the patient's periodontal condition is stabilized.
6. Regular follow up for 1 year and or according to the disease severity

Augmentin 30mg/kg/day given twice daily and Metronidazole 30 mg/ kg/ day thrice daily for five days (as calculated using the body weight method).^{9,10} Patient was recalled after a month.

During the subsequent follow-up (after one month), there was visible reduction in inflammation and bleeding from gums. This was followed by superficial supragingival scaling. Oral hygiene instructions were reinforced. A repeat examination, similar treatment protocol and patient education steps were followed during this follow-up (after one month).

Chlorhexidine mouthwash was prescribed for three weeks in a dose of 5ml of 0.12% for 1 minute three times daily.¹⁰ The patient failed to maintain the follow-up appointments for about one and a half years. Then she re-visited the dental clinic, accompanied by her father, who complained of sensitivity in the child's lower right posterior teeth. On examination, tooth number 74 and 84 exhibited advanced gingival recession buccally with abundant plaque visible on the root surface. Similar changes were visible to a lesser extent on 54 and 64. These findings were correlated with intra-oral

periapical radiographs (Figures 1, 2) which showed profound arc-shaped interdental and inter-radicular bone loss.



Figure 1: Clinical appearance and Radiograph of tooth 54, depicting frank gingival recession (1A) and bone loss along with resorbed roots (1B)

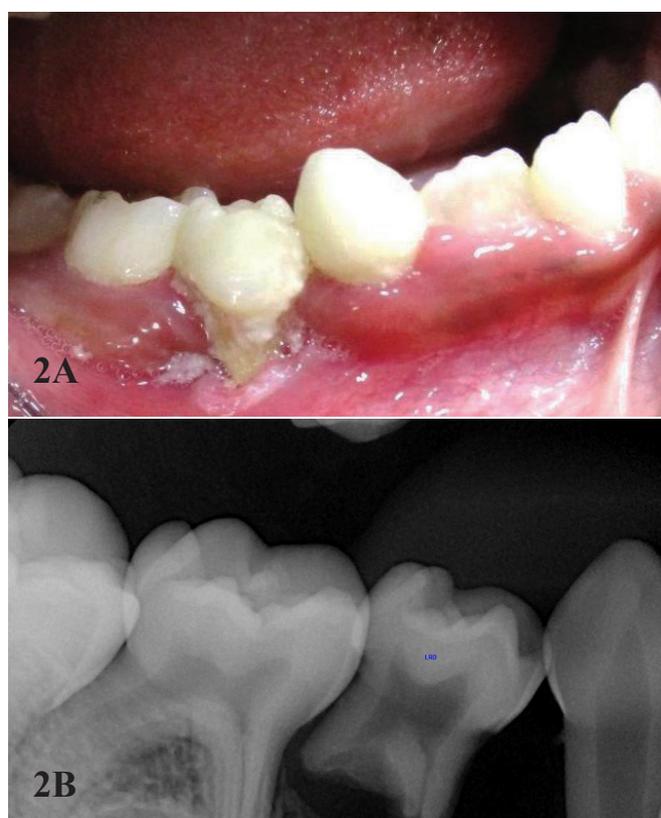


Figure 2: Noticeable gingival recession (2A) in Tooth 84 and radiograph (2B) showing floating tooth due to periodontal pocket and bone loss

Dental panoramic tomography (Figure 3) revealed the physiologic presence of all the permanent teeth buds and severe periodontal bone loss with a first primary Molar – Incisor pattern of distribution.



Figure 3: DPT shows normal presence of all permanent teeth buds with primary first molars affected

Saliva sample was collected in sterile plastic containers. Slide smear treated with Gram's staining method showed presence of gram-negative rods. This is an important finding because most periodontal pathogenic bacteria are gram-negative and their presence in easily detectable numbers in the saliva of a child of less than 5 years age represents a significant risk of periodontal disease progression.¹¹ Oral prophylaxis and fluoride application was done and oral hygiene instructions were reinforced. Antibiotics were prescribed for a week, along with Chlorhexidine mouthwash for three weeks. Since the child's oral hygiene was not found to be satisfactory at the second recall visit, the consequences of poor oral hygiene on short- and long-term prognosis were explained to the patient's father.

The patient was recalled after 3 months for a follow-up and was asked to re-visit the clinic in case of pain. Counseling for space maintainers in the event of an extraction of the primary first molars was done. However, the patient returned to the clinic after a year, complaining of intermittent pain in lower right first primary molar. Clinically, severe gingival recession was evident in lower right and left primary first molars. There was grade III mobility in the lower right primary molar and marked gingival inflammation was noted around the affected teeth with calculus deposits. Since the child was mildly anxious at this time and the mobile tooth 84 was asymptomatic, it was decided to leave the tooth in situ until it exfoliated naturally.

Discussion

Periodontitis is defined as an inflammatory disease of supporting tissues of teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession, or both. The most common causative organisms associated with periodontal diseases are *Porphyromonas gingivalis*, *Prevotella intermedia*, *Bacteroides forsythus*, *Campylobacter rectus*, *Aggregatibacter actinomycetemcomitans*, and the treponemes (Carranza's Clinical Periodontology, 2006). The occurrence of periodontal diseases in adults is a common clinical finding as compared to children. The periodontium of the child differs from the adult with respect to the gingiva being more vascular, absence of stippling; the periodontal ligament fibres being wider and less dense. The alveolar bone of the child has more marrow spaces, lesser trabeculation and calcification with greater blood supply and lymphatic drainage. (Pinkham JR et al. Pediatric Dentistry, 2005.)

As a multifactorial disease, the development of periodontitis is dependent on the presence of a susceptible host, deficient plaque control measures and pathogenic bacteria.¹² However, the periodontal pathogens associated with rapid periodontal destruction are also found in patients with healthy periodontal status. Therefore, isolation of these bacterial species is not pathognomonic of periodontal disease of any type, or indeed of periodontal disease at all. Therefore, our investigation did not include microbiological culture or Polymerase Chain Reaction (PCR) to identify the bacteria in the patient's dental plaque. The patient did not have any syndrome, significant medical history, or abnormal enzymatic results after biochemistry studies. However, the saliva sample with high gram-negative rods suggested 'localised aggressive periodontitis' or Stage III Grade C periodontitis of Molar - Incisor pattern, according to the 2017 Classification of periodontitis.⁵

There have been successful outcomes in cases of periodontitis in primary dentition with mechanical debridement with systemic antibiotics. A cohort study of 97 African American participants between

the ages of 5–21 by Merchant SN et al., reported positive treatment outcomes in paediatric patients. This was explained, as there was greater reduction in clinical attachment loss and better healing tendency in paediatric population.¹³

It is very uncommon to find gingival recession and bone loss in young children. The bone loss in furcation areas of primary second molars are known to extend to the mesial aspect of the permanent first molars and can persist as angular defects even after exfoliation of the affected primary molars.¹⁴ Due to the rarity of aggressive periodontitis in deciduous dentition, such cases may be misdiagnosed or completely undiagnosed, leading to premature loss of deciduous dentition and associated sequelae. An under-treated case may increase the possibility of the disease, later affecting the permanent dentition.

The clinical lesions in this case worsened because of poor compliance with oral hygiene instructions and irregular follow up at the dental clinic. The plaque samples were positive for gram-negative bacteria suggestive of a bacterial cause for bone loss and gingival inflammation.

The progress of localised aggressive periodontitis from primary dentition to the permanent dentition is an uncommon finding.¹⁵ However, the lack of cases being reported could be one of the reasons, as this cannot be confirmed. This rare finding in children results in smaller sample size, which could be the reason for absence of confirmation, that it affects the permanent dentition. Patient education about oral hygiene and regular dental screening are of imperative importance in preventing the progression to aggressive periodontitis in permanent dentition.

Conclusion

The patient's parents had been counselled regarding the need for periodic evaluation of siblings for similar signs and symptoms. Subsequent events justified this advice.

At the time of writing this article, in 2019, the patient's younger brother (4 years 8 months old) reported to the dental department with similar signs and symptoms. This suggests a genetic component in the aetiology of the patient's condition. This aspect needs to be verified with further investigations.

Rapid loss of attachment around primary molars is a rare occurrence and one that needs to be understood in greater detail. Until then dental practitioners must answer the questions posed by the patient's parents on the long-term effects of the disease if any, based on the findings of case reports like this published around the world.

Conflict of interests

There are no conflicts of interest to declare.

Acknowledgment

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