

Generalized Aggressive Periodontitis in Preschoolers: Report of a case in a 3-1/2 Year Old

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Destructive forms of periodontal disease in children are uncommon. Severe periodontal destruction can be a manifestation of a systemic disease; however, in some patients, the underlying cause of increased susceptibility and early onset is still unknown.

Objective: To describe an effective therapeutic approach to Generalized Aggressive Periodontitis (GAgP) in children, based on a 3-1/2 year-old male patient referred to the Hospital due to early loss of incisors, gingivitis, and tooth mobility in his primary dentition. Intraoral examination revealed severe gingival inflammation, dental abscesses, pathological tooth mobility, bleeding upon probing and attachment loss around several primary teeth. Dental radiographs revealed horizontal and vertical bone loss. Treatment consisted on the extraction of severely affected primary teeth, systemic antibiotics, deep scaling of remaining teeth and strict oral hygiene measures. Once the patient's periodontal condition was stabilized, function and esthetics were restored with "pedi-partial." After a follow-up period of nearly 4 years, the patient's periodontal status remains healthy, facilitating the eruption of permanent teeth.

Conclusion: Prompt diagnosis and good treatment regimen may provide an effective therapeutic management of Generalized Aggressive Periodontitis.

Keywords Generalized aggressive periodontitis, early-onset periodontitis, prepubertal periodontitis, primary dentition, premature dental loss, *Aggregatibacter actinomycetemcomitans*

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INTRODUCTION

The importance of a prompt diagnosis and treatment of periodontitis in children is emphasized by the association between the presence of periodontitis in primary dentition and periodontitis at older ages in the same individual.¹⁻⁴ The 1999 *International Workshop for a Classification of Periodontal Diseases and Conditions* classified periodontal disease in children as follows: Dental plaque-induced gingival diseases; aggressive periodontitis (previously known as "prepubertal" or "early onset periodontitis");

chronic periodontitis; periodontitis as a manifestation of a systemic disease; and necrotizing periodontal diseases.

Aggressive and chronic periodontitis is subdivided into localized or generalized, depending on the size of the area affected.^{5,6} Most of the literature reports of severe periodontal destruction in children are associated with systemic diseases such as hypophosphatasia, cyclic neutropenia, agranulocytosis, histiocytosis X, leukocyte adhesion deficiency, Papillon-Lefèvre syndrome and leukemia.⁵

Although destructive forms of periodontal disease in infants are relatively uncommon, children and adolescents may manifest any form of periodontitis. However, it has been shown that aggressive periodontitis may be more common in children and adolescents, while chronic periodontitis is more frequent in adults.^{5,6}

Prevalence estimates range widely in different geographical regions, and demographic and ethnic groups. The estimates of prevalence rates of early onset aggressive periodontitis in the general populations in different continents are: 0.4 – 0.8% in North America, 0.1 – 0.5% in Western Europe, 0.3 – 1% in South America, 0.5 – 5% in Africa and 0.4 – 1% in Asia.

In terms of race-ethnic groups, the prevalence varies from 0.1 – 0.2% in Caucasians, 1 – 3% in Africans and African-Americans, 0.5 – 1% in Hispanics and South Americans and 0.4 – 1% in Asians.⁷

Patients with Generalized Aggressive Periodontitis

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(GAgP) present a history of rapid gingival attachment loss, bone loss, severe periodontal inflammation, and heavy plaque and calculus accumulation.^{5,8,9} GAgP patients exhibit generalized interproximal attachment loss including at least three teeth in addition to first molars and incisors.⁵ Although in young subjects the onset of these diseases is often circumpubertal, GAgP may appear at any age and often affects the entire dentition.^{5,8,9}

The etiology of aggressive periodontitis may be broadly divided into two categories: bacterial plaque with highly pathogenic bacteria, and impaired host defense mechanism.⁴ As far as pathological microflora is concerned, the most important bacteria appear to be highly virulent strains of *Aggregatibacter actinomycetemcomitans* previously known as *Actinobacillus actinomycetemcomitans* in combination with *Porphyromonas gingivalis*, *Prevotella intermedia* and *Treponema denticola*, however other bacteria may be present.^{10,11} Although there seems to be a genetic predisposition for periodontal diseases,^{12,13} it has also been shown that periodontopathic bacteria are transmissible among family members or between children and their caregivers.^{14,15}

Regarding the impaired host defense mechanism, neutrophils from patients with GAgP frequently exhibit suppressed chemotaxis or altered phagocytosis.^{16,17} Moreover, alterations in immunologic factors are known to be present in Aggressive Periodontitis. Immunoglobulins, with their important protective disease-limiting effects, appear to be influenced by patients' genetic background and environmental factors such as bacterial infection.^{11,18,19} Patients with Aggressive Periodontitis often present impaired immune function, particularly neutrophil dysfunction. In these cases, clinicians should always rule out systemic diseases that can affect host defense mechanisms.⁴

Successful treatment of patients with Aggressive Periodontitis depends on early detection, mechanical debridement and antibiotic therapy to provide an infection-free environment.^{13,20} However, while the use of antibiotics in conjunction with root debridement appears to be effective for the treatment of Localized Aggressive Periodontitis, GAgP does not always respond well to conventional treatment or to antibiotics commonly used to treat periodontitis.^{21,22} Several

authors report successful antibiotic therapy, combining amoxicillin and metronidazole and excluding tetracyclines, which may stain developing teeth.^{13,23-25} Most cases of young patients with GAgP require the extraction of the affected primary teeth to prevent bacterial spread to the erupting permanent dentition.^{24,26}

Case Report

A 3 years 7 months white male was referred by his pediatrician to the Pediatric Dentistry Service of the *Hospital Sant Joan de Déu*, Barcelona due to early loss of incisors and severe gingival inflammation. His medical history appeared non-contributory, as was not taking medication, referred no allergies, and had no history of episodic illness nor orofacial trauma. His parents reported that they were healthy and denied any history of periodontal disease. The gingival inflammation had started 6 days previously and their son was not eating well due to pain.

The clinical oral examination revealed a full primary dentition, heavy plaque accumulation, absence of lower incisors (71, 81), severe gingival inflammation, generalized gingival recession and abscesses at the level of the maxillary second primary molars (55 and 65) (Figure 1). There was bleeding upon probing, periodontal pockets measured at 5 mm around all first primary molars. Halitosis and second-degree mobility almost throughout the dentition (based on the modified Miller index of horizontal tooth mobility) was present.²⁷ There was no evidence of caries. The panoramic X-ray revealed severe generalized vertical and horizontal bone loss (Figure 2). The patient was referred for a complete medical evaluation to rule out any underlying systemic disease. His complete blood count was within normal limits, including basal glucose and creatinine levels, coagulation factors, alkaline phosphatase levels, absolute T4 lymphocyte count, immunoglobulins G, A, M and IgG subclasses. Absolute monocyte and neutrophil counts were slightly elevated. For further evaluation, subgingival plaque was sampled from the deepest pockets using paper points. The results revealed aerobic and anaerobic flora, especially *Streptococcus Viridans* and *Peptostreptococcus spp.* Microbiological tests on selective media *Aggregatibacter (actinobacillus actinomycetemcomitans)* or *Prevotella intermedia* were not available.

Due to the age of the patient, the severity of bone loss,



Figure 1.



Figure 2.



Figure 3.



Figure 4.



Figure 5.

and the lack of a detectable systemic disease, the diagnosis of Generalized Aggressive Periodontitis was made. The patient was treated with systemic antibiotics: amoxicillin (50 mg/kg/day, divided into 3 doses) in combination with metronidazole (30 mg/kg/day) for 10 days. Teeth with bone loss equal to or greater than two thirds of root length and mobility equal to or greater than grade II were extracted. The



Figure 6.

following teeth were extracted: 54, 52, 51, 61, 62, 64, 74, 72, 82 and 84. Canines and second primary molars were maintained. Due to uncooperative behavior, dental extractions were done under general anesthesia (Figure 3). We observed that the extracted teeth had an irregular external resorptive pattern (Figure 4). The remaining teeth underwent root planing and scaling every month during the first year. Moreover, parents were advised to brush the boy's teeth with a 0.12% chlorhexidine rinse three times a day during 3 months. Once the patient's periodontal condition was stabilized 12 months post-treatment, at parent and patient's request, and in view of his good compliance, he was rehabilitated with partial acrylic appliances ("pedi-partials") to restore function and esthetics (Figure 5). At this point the patient was referred for another complete blood count; all the results were normal, including the absolute monocyte and neutrophil counts.

More than 3-1/2 years post-treatment and with monthly recall appointments, the gingival and periodontal health of the patient remains good. Clinical and radiographic examinations reveal that permanent incisors and first molars have erupted without signs of periodontal disease (Figure 6 and 7).

Few articles have been published on Generalized Aggressive Periodontitis in children since the last Classification of Periodontal Diseases. None of them have described the condition at this early age. Equally, few epidemiologic studies of aggressive periodontal disease have been conducted in



Figure 7.

preschoolers, probably because it is a rare finding during the first decade of life.

Any association between susceptible systemic diseases and the case presented here was ruled out because of the results of the clinical and laboratory examinations. We believe the slight rise in the absolute monocyte and neutrophil counts was probably related to bacterial infection responsible for the aggressive periodontitis.

In some children, as in this case, the underlying cause of the increased susceptibility and early onset of the disease is not known. However, as scientific investigation on genetics and host resistance to infection is constantly advancing, we believe cases such as this one may be clarified in the near future.

Table 1. Dental protocol for young children with Generalized Aggressive Periodontitis

- Prescription of systemic antibiotics: amoxicillin (50 mg/kg/day, divided into 3 doses) + metronidazole (30 mg/kg/day) for 10 days
- Extraction of hopelessly involved teeth: teeth with bone loss \geq to two thirds of root length and mobility \geq to grade II
- Supragingival and subgingival curettage and debridement of remaining teeth (if any) every month during the first year
- Strict measures of oral hygiene: tooth brushing with 0.12% chlorhexidine rinse 3-times a day during 3 months (consider parental involvement, depending on the patient's age)
- Consider prosthetic rehabilitation once the patient's periodontal condition is stabilized, to restore function and aesthetics
- Recall appointments every month during the first year, then decide the period of time between visits based on findings in the follow-up and the disease severity

Previously, 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.^{25,28,29} We hypothesize that the presence of extensive eroded areas devoid of cementum in the extracted primary teeth of this patient could have facilitated the progress of periodontal disease. Alternatively, the external root resorption may have been a pulp reaction to periodontopathic bacteria.

CONCLUSION

We believe that a successful outcome can be achieved with an early diagnosis and conservative treatment, preventing its recurrence in the primary and permanent teeth. Once the dental treatment is accomplished, stabilization of the periodontal condition in children could be influenced by their own immunological maturity. The therapeutic approach includes the prescription of systemic antibiotics in combination with mechanical treatment, as well as strict oral hygiene measures and frequent recall appointments. Moreover, we believe therapeutic management must also take into account the functional and esthetic needs of these young patients and prosthetic rehabilitation should be considered in preschool-

ers. As the patient grows into a mixed dentition, “*pedi-partial*” could include an expansion screw to compensate transverse bone growth.

Finally, it is extremely important that children with GAgP be carefully monitored to provide early treatment when necessary due to an increased susceptibility for periodontal diseases at older ages.

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