

Radiographic Description of the Distribution of Aggressive Periodontitis in Primary Teeth

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Objective: To describe the prevalence by site and tooth of aggressive periodontitis (AP) in primary teeth of children with AP, that will facilitate the early diagnosis of AP.

Study design: Radiographic evidence of AP by tooth and site of primary teeth of 29 children with AP was analyzed by gender, ethnicity, type of dentition (primary or mixed), alveolar bone site and type of tooth. **Results:** The range and mean \pm standard error of number of sites and teeth per patient with AP were 4 to 28, 13.1 ± 1.2 and 4 to 12, 8.3 ± 0.5 respectively. The differences in prevalence of AP by gender, ethnicity, dentition and homologous sites at both sides of the mouth were not statistically significant (Chi square). AP was mostly diagnosed at the distal area of the maxillary canine, the proximal areas of the first maxillary primary molars, and the distal area of the mandibular first primary molar. The most affected teeth with AP were the first primary molars followed by the maxillary cuspid, and the second molars. **Conclusions:** Examination of children's primary teeth should include abnormal mobility, when found it should be followed by radiographic evaluation for the presence of AP, taking in consideration the present findings.

Key words: diagnosis, aggressive periodontitis, primary dentition.

INTRODUCTION

Children and adolescents may have any of the several forms of periodontitis described in the Proceedings of the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions.¹ Accordingly, several cases of aggressive periodontitis (AP) affecting the primary dentition in 1 or more children in the same family have been reported in the literature (Figures 1-2).²⁻¹¹ The early diagnosis and treatment of AP in the primary dentition is most relevant for the child's wellbeing, growth and development, the prevention of transmission of AP among individuals, and the prevention of the development of AP at older ages.^{2, 5, 6, 8, 12, 13}

If AP is not treated soon after its inception at an early age, it will lead to fast and extensive alveolar bone loss with the

consequent spontaneous exfoliation of primary teeth without the normal root resorption process, or the need for early extraction.^{2, 3, 9} Furthermore, during the mixed dentition, there is a possibility of bacterial colonization of the periodontium of the newly erupted permanent teeth by periodontal pathogens from the periodontium of primary teeth affected with AP.¹¹

Despite that not every child with AP in the primary teeth will develop AP in his/her permanent teeth, there is still a positive statistically significant correlation in the prevalence and severity of periodontitis in the primary and permanent dentitions of the same individuals.^{4, 12, 13} On the other hand, the early treatment of AP affecting the primary dentition has been proven to be successful in preventing the development of AP during the mixed and permanent dentition.^{2, 3, 6-9}

The literature indicates that evidence of the inception of AP in children is usually found at the 1st permanent molars and permanent incisors, this being correct for the permanent teeth.⁷ While the reports of AP affecting the primary dentition describe the primary molars as being the most affected teeth,²⁻¹³ a description of the distribution of AP in the primary teeth appears to be missing in the literature; this information being most relevant since it may direct the clinician to the early diagnosis and treatment of AP affecting the primary teeth. Therefore, the purpose of the present manuscript is to describe the prevalence by site and tooth of aggressive periodontitis (AP) in primary teeth of children with AP, which should facilitate the early diagnosis and treatment of AP in children.

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MATERIALS AND METHOD

This research was approved by the University of Kentucky Office of Research Integrity (IRB 16-1054-PH). Radiographs from healthy children with a confirmed diagnosis AP that were treated by the author within his pediatric dental practice, or cases for which the author was consulted regarding their diagnosis and/or treatment were reviewed. Radiographs were selected for the present research when: 1) both sides of the mouth were present; 2) the alveolar bone crest area was evident; 3) there was no evidence of deep caries that could have elicited pulp pathology.

The child’s gender, ethnic origin (African American (AA) or Caucasian), and the presence of AP lesions located at the mesial of distal surface of all the primary teeth, and the inter-radicular area of the primary molars were examined. Since the age of the patient was not available, the database included information about the child having a primary or mixed dentition, based on the absence or presence of erupted permanent teeth. AP at the mesial and distal dental areas was recorded when the distance from the alveolar bone crest to the cement-alveolar junction was > 2 mm and there was clear evidence of the alveolar bone crest loss.¹⁴⁻¹⁷ AP at the inter-radicular area AP was recorded when an inter-radicular radiolucency appeared to be continuous with the proximal evidence of AP and with a “U shape” that has been previously described for AP in the primary dentition.⁷

Chi square statistical analysis was utilized to evaluate the significance of the differences in proportions of radiographic evidence of AP by gender, ethnicity, primary or mixed dentition, bilateral sites and teeth. All statistical analyses were performed with a statistical software program (JMP 12.1, Statistical Discovery, from SAS Institute Inc., Cary, NC, USA, 2015) with a < .05 degree probability level of significance.

RESULTS

Out of the records of 92 patients, 29 (31.5%) patients were found to have suitable BW radiographs. Radiographs of the anterior primary teeth were scarce, and evidence of primary teeth being affected with AP was found in only two cases (one affecting the 4 maxillary incisors and one the mandibular central incisors). Therefore, the research database included only the radiographic findings from bitewing radiographs; more specifically, the alveolar bone located from the distal of the primary cuspids to the distal area of the primary second molars. Information about the children’s gender and ethnic origin was available for 26 and 27 children respectively, 12 had a primary dentition and 17 a mixed dentition (Table 1).

Table 1- Demographic data.

| Category | | N | % |
|-----------|------------------|----|------|
| Gender | Male | 20 | 76.9 |
| | Female | 06 | 23.1 |
| Race | African American | 07 | 25.9 |
| | Caucasian | 20 | 74.1 |
| Dentition | Mixed | 17 | 58.6 |
| | Primary | 12 | 41.4 |

The mean number ± standard error of sites and teeth per child were 13.1±1.2 and 8.3±0.5 respectively (Table 2). The differences

Table 2- Number (#) of children by the number (#) of sites and teeth per child with evidence of aggressive periodontitis

| # of children | # of sites |
|----------------------|-------------------|
| 1 | Between 1 to 5 |
| 11 | Between 6 to 10 |
| 8 | Between 11 to 15 |
| 7 | Between 16 and 21 |
| 2 | 21-28 |
| Mean± standard error | 13.1±1.2 |
| Range | 4 to 28 |
| # of children | # of teeth |
| 5 | 4-5 |
| 9 | 6-7 |
| 5 | 8-9 |
| 10 | 10-12 |
| Mean± standard error | 8.3±0.5 |
| Range | 4-12 |

in number of sites affected with AP in males (12.3±1.3) or females (12.2±2.4, in AA (13.7±2.4) or Caucasian (12.3±1.4), during the primary (11.8±1.8) or mixed dentition (13.9±1.5), and between homologous sites at both sides of the mouth were not statistically significant (Chi square, p>0.05). Therefore, both sides were pooled together to evaluate the prevalence of AP at the various sites. AP was mostly diagnosed at the distal area of the maxillary canine, the proximal areas of the first maxillary primary molars, and the distal area of the mandibular first primary molar (Figure 3). The most affected teeth with AP were the first primary molars followed by the maxillary cuspid, and the second molars (Figure 4). All the cases with AP at the distal areas of the second primary molars were found after the eruption of the first permanent molars. When examining the prevalence of AP by tooth type in the primary or mixed dentition periods, the only statistically significant difference was found for the mandibular second primary molar (27.3% and 72.7% respectively, Chi square p = 0.006).

Figure 1. Bite-wing radiograph before the eruption of the first primary molars showing aggressive periodontitis affecting the distal surface of the maxillary primary cuspid, the mesial, inter-radicular and distal surfaces of the maxillary first primary molar, the mesial surface of the maxillary second molar, the inter-radicular and distal surfaces of the mandibular first molar and the mesial and inter-radicular surfaces of the primary second molar.



Figure 2. Bite-wing radiograph after the eruption of the first primary molars showing aggressive periodontitis affecting the distal surface of the maxillary primary cuspid, the mesial and distal surfaces of the maxillary first primary molar, the mesial surface of the maxillary second molar, the distal surface of the mandibular first molar and the mesial surface of the primary second molar.



DISCUSSION

Aggressive periodontitis is a rare form of periodontal disease that may be found in children and adolescents during the primary, mixed and permanent dentitions, it may be localized or generalized.¹⁻¹³ It is a multifactorial disease caused primarily by dental plaque microorganisms but also influenced by local and systemic factors, such as developmental changes and genetics.^{1, 14-19} The treatment of AP by a combination of extraction of severely affected teeth, localized non-surgical periodontal therapy and systemic and/or local antibiotic therapy (mainly amoxicillin and/or metronidazole) has proven to be successful in the treatment of AP after short and long terms, improving clinical and microbiological results.^{2, 3, 6, 8, 9} Moreover, it has been reported that non-surgical periodontal therapy combined with systemic antibiotics of localized aggressive periodontitis may lead to a more effective reduction in clinical attachment levels in the primary dentition than in the permanent dentition.⁷ This may be related to the fact that the

Figure 3. Prevalence of aggressive periodontitis by site in primary teeth.

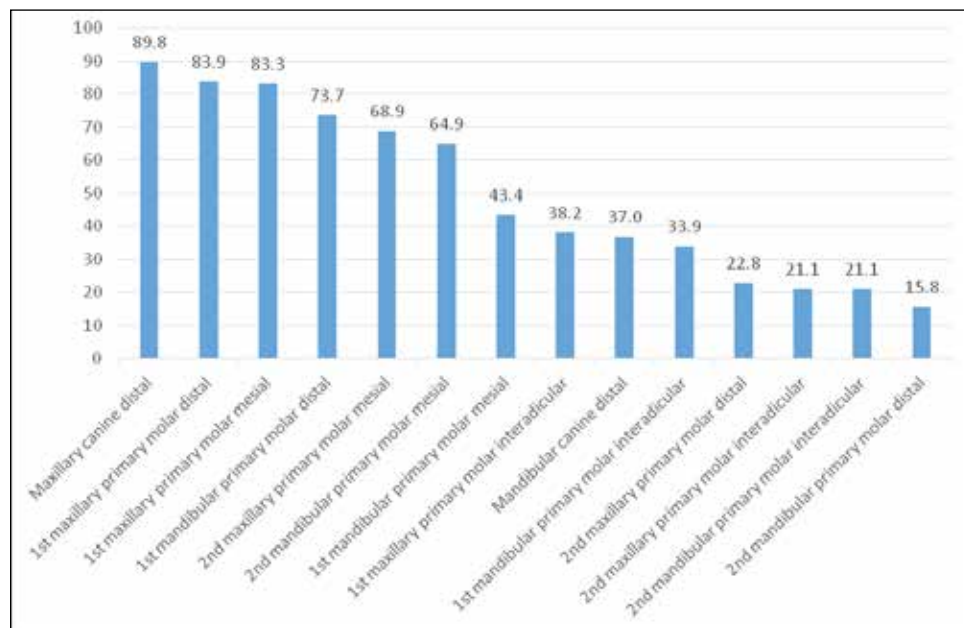
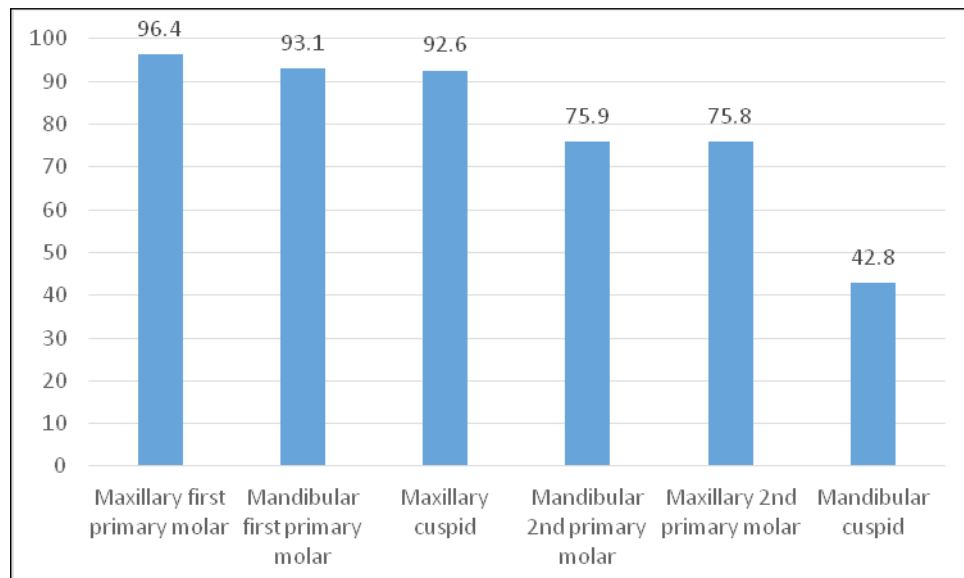


Figure 4. Prevalence of aggressive periodontitis by tooth in primary teeth.



reactivity of the periodontal tissues to bacteria and their toxins may increase with age, possibly due to changes in plaque levels, bacterial composition, increased inflammatory response, tooth eruption and exfoliation.^{14,15} In AP cases in which early extraction of primary teeth is required, space maintainers and partial dentures should be considered for the prevention of space loos, function and esthetics.^{2,3,20}

The few cases of AP affecting the primary incisors in this study and in the literature may mislead to the conclusion that the primary incisors are mostly not affected by AP; one must take in consideration that AP is mostly reported in the literature at ages after the primary incisors have already normally exfoliated, that AP may significantly affect the alveolar bone while the gingival appearance is within normal limits, and the fact that periodontal probing is not part of the standard oral examination of children.²¹ Regarding the uncertainty about which primary teeth are first affected by AP, two manuscripts describe the presence of AP in 3-year old children; in one case the mandibular central primary incisors already exfoliated as the result of severe generalized AP,³ and in the other case only the mandibular central primary incisors were described to be affected with AP;² these cases clearly indicate that AP in the primary teeth may affect the primary incisors as early as age ≤ 3 years but cannot lead to the conclusion that the primary incisors are the first ones to be affected. Interesting is the finding that the prevalence of AP in the primary teeth was found to be not statistically significant different at the primary or mixed dentition periods, suggesting that most cases of AP start at the primary dentition age, before the age of 6 years.

Diagnosis of AP in standard pediatric dental examinations is complicated since:^{2,21,22} a) AP may exist without evidence of large amounts of plaque or calculus, and with none to minimal clinical signs; b) sulcus probing depths that could indicate incipient alveolar bone loss is not part of the standard pediatric dental examination, especially in young children since it may elicit uncooperative behavior; c) anterior mandibular radiographs in the primary dentition are rarely indicated due to a relative small prevalence of caries in these teeth; d) AP affects a small proportion of children who are otherwise healthy and therefore, awareness about AP in children appear to be deficient. The question that remains is how the clinician may diagnose AP in small children? The answer is that evaluation of abnormal mobility of primary teeth should be part of the standard pediatric oral examination, followed by radiographic evaluation of the teeth with abnormal mobility, taking in consideration the findings of the present study.

CONCLUSIONS

Oral health providers should be aware that:

1. Despite that AP appears to mainly affect the primary cuspids and molars of the primary dentition, it is possible that AP may be evident in the primary anterior and posterior teeth as early as age 3-years.
2. When considering the odds of radiographic evidence of AP at the various sites of the primary teeth, AP is mostly seen ($\geq 64.9\%$ of the sites) at the distal area of the maxillary canine, the proximal surfaces of the 1st primary molars and the mesial surfaces of the 2nd primary molars.
3. The primary teeth should be evaluated for abnormal mobility and when found, radiographic examination of the teeth with abnormal mobility should be performed taking in consideration the findings of the present study.

REFERENCES

1. American Academy of Pediatric Dentistry Reference Manual. Periodontal diseases of children and adolescents. *Ped Dent* 38:388-396, 2016.
2. Bimstein E. Seven-year follow up of 10 children with periodontitis. *Pediatr Dent* 25:389-96, 2003
3. Portaro CP, Chópito YG, Cárdenas AC. Generalized aggressive periodontitis in preschoolers: report of a case in a 3-1/2 year old. *J Clin Pediatr Dent* 33:155-9, 2008.
4. Mros ST, Berglundh T. Aggressive periodontitis om children: a 14-19 – year follow-up. *J Clin Periodontol* 37:283-7, 2010
5. Casarin RC, Peloso Ribeiro ED, Sallum EA, Nociti FH Jr, Gonçalves RB, Casati MZ. The combination of amoxicillin and metronidazole improves clinical and microbiological results of one stage, full mouth, ultrasonic debridement in aggressive periodontitis treatment. *J Periodontol* 83:988-98, 2012.
6. Seremidi K, Gizani S, Madianos P. Therapeutic management of a case of generalized aggressive periodontitis in an 8-year old child: 18 months result. *Eur Arch Paediatr Dent* 13:266-71, 2012.
7. Merchant SN, Vovk A, Kalash D, Hovencamp N, Aukhil I, Harrison P, Zapert E, Bidwell J, Varnado P, Shaddox LM. Localized aggressive periodontitis treatment response in primary and permanent dentitions. *J Periodontol* 85:1722-9, 2014.
8. Muppa R, Nallanchakrava S, Chinta M, Manthena RT. Nonsyndromic localized aggressive periodontitis of primary dentition: A rare case report. *Contemp Clin Dent* 7:262-4, 2016.
9. Sharma G, Whatling R. Case report: premature exfoliation of primary teeth in a 4-year-old child, a diagnostic enigma. *Eur Arch Paediatr Dent* 12:312-7, 2011.
10. Shapira L, Shmidt A, Van Dyke Th, Barak V, Soskolne AW, Brautbar Ch, Sela MN, Bimstein E. Sequential manifestation of different forms of early onset periodontitis. A case report. *J Periodontol* 65:631-635, 1994.
11. Dibart S, Chapple IL, Skobe Z, Shusterman S, Needleman HL. Microbial findings in prepubertal periodontitis. A case report. *J Periodontol* 69:1172-5, 1998.
12. Sjödin B, Crossner CG, Unell L, Oslund P. A retrospective radiographic study of alveolar bone loss in the primary dentition in patients with localized juvenile periodontitis. *J Clin Periodontol* 16:124-7, 1989.
13. Sjödin B, Matsson L, Unell L, Egelberg J. Marginal bone loss in the primary dentition of patients with juvenile periodontitis. *J Clin Periodontol* 20:32-6, 1993.
14. Bimstein E, Matsson L. Growth and development considerations in the diagnosis of gingivitis and periodontitis in children. *Pediatr Dent* 21:186-91, 1999.
15. Bimstein E, Garcia-Godoy F. The significance of age, proximal caries, gingival inflammation, probing depths and the loss of lamina dura in the diagnosis of alveolar bone loss in the primary molars. *ASDC J Dent Child* 61:125-8, 1994.
16. Bimstein E. Radiographic diagnosis of the normal alveolar bone height in the primary dentition. *The Journal of Clinical Pediatric Dentistry*, 19:269-71, 1995.
17. Shapira L, Tarazi E, Rosen L, Bimstein E. The relationship between alveolar bone height and age in the primary dentition: A retrospective longitudinal radiographic study. *Journal of Clinical Periodontology*, 22:408-12,1995.
18. Shapira L, Schlesinger M, Bimstein E. Possible autosomal-dominant inheritance of prepubertal periodontitis in an extended kindred. *J Clin Periodontol* 24:388-93, 1997.
19. Shaddox LM, Goncalves PF, Vovk A, Allin M, Huang H, Hpu W, Aukhill I, Walleet SM. LPS-induced inflammatory response after therapy of aggressive periodontitis. *J Dent Res* 92:702-8, 2013
20. Hazan-Molina H, Zigdon H, Einy S, Aizenbud D. Periodontal and space maintenance considerations for primary teeth preenting with aggressive periodontitis: a case report. *Pediatr Dent* 34:254-8, 2012.
21. Page RC, Bowen T, Altman L, Vandestein E, Ochs H, Mackenzie P, Osterberg S, Engel LD, Williams BL. Prepubertal Periodontitis. I. Definition of a Clinical Disease Entity. *J Periodontol* 54:257-271, 1983
22. Kumar A, Masamatti SS, Virdi MS. Periodontal diseases in children and adolescents: a clinician's perspective part 2. *Dent Update* 2012 39:639-42, 645-6, 649-52, 2012.