Root Surface Characteristics of Children Teeth with Periodontal Diseases

Bimstein E*/ Wignall W**/ Cohen D***/Katz J****

The present study examined the root surfaces of teeth from children with or without periodontal diseases and with or without systemic diseases. Light microscopy revealed that when compared to control teeth: teeth with chronic periodontitis had similar radicular histology; teeth from children with leukocyte adhesion deficiency, Down syndrome and aggressive periodontitis had narrower cementum areas; teeth from children with hypophosphatasia showed cementum aplasia. Cementum anomalies may facilitate the establishment and progress of periodontitis in children.

Keywords: *Microscopy, root surface, cementum, periodontitis, children, systemic condition.*
J Clin Pediar Dent 32(2): 101–104, 2007

INTRODUCTION

Aggressive periodontitis (AP), and periodontal diseases related to systemic diseases are often found in children.[1-13] Furthermore, it has been demonstrated that there is a relationship between the prevalence and the severity of aggressive periodontal diseases at different ages in the same individual.[14-16]

The onset and rapid progression of periodontitis in children may be facilitated by several factors including an abnormal periodontal attachment to defective cementum, which is highly susceptible to microbial invasion and destruction.[17, 18] In fact, cementum hypoplasia or aplasia have been related to extensive alveolar bone loss in cases with hypophosphatasia,[1-2, 8, 9] adolescent aggressive periodontitis,[19] Papillon-Lefèvre Syndrome (PLS),[5] and leukocyte adhesion deficiency (LAD).[20] Moreover, it has been suggested that AP in children with no evidence of systemic diseases or syndromes may be related to undiagnosed hypophosphatasia.[4] Very few studies describe the histological characteristics of teeth of children affected with periodontitis or compare the histological characteristics of various types of periodontitis in children. The purpose of the present study was to describe the histological root surface characteristics of teeth from children with or without various types of periodontal diseases, and with or without systemic diseases that have been previously related to periodontal diseases.

MATERIALS AND METHODS

This study was approved by the Institutional Review Board of the University of Florida and included: a) 30 primary and 2 permanent teeth (from 13 children) with severe periodontitis that previously underwent histological examination for the purpose of pathological assessment; b) 21 primary teeth (from 9 children) with greater than half of their root still present that were extracted after informed consent from the children and their parents due to severe periodontal disease, caries or orthodontic reasons. The 53 teeth were classified based on the absence/presence and type of periodontal diseases[22] and absence or presence and type of systemic disease (Table 1).

Periodontal disease was recorded when the distance from the cemento enamel junction to the alveolar crest was > 2 mm and the lamina dura over the alveolar bone crest was absent.[23] In addition, the presence or absence of periodontitis local facilitating factors such as interproximal caries,

Table 1. Distribution of teeth examined by group characteristics.

Group	Number of teeth	CEJ-ABC* distance	Lamina dura over the alveolar bone	Local periodontitis facilitating factors	Systemic condition
Control	10	⩽2 mm	Present	None	Healthy
Chronic periodontitis	5	⩾2 mm	Absent	Yes	Healthy
Aggressive periodontitis	16	⩾2 mm	Absent	None	Healthy
Systemic disease periodontitis	22	⩾2 mm	Absent	None	Unhealthy

*Cementoenamel junction to alveolar bone crest

* Enrique Bimstein. Department of Pediatric Dentistry, University of Florida College of Dentistry.
** Whitney Wignall. Graduate on Pediatric Dentistry, University of Florida College of Dentistry, actually in private practice.
*** Don Cohen. Department of Oral and Maxillofacial Surgery and Diagnostic Sciences, University of Florida College of Dentistry.
**** Joseph Katz. Department of Oral and Maxillofacial Surgery and Diagnostic Sciences, University of Florida College of Dentistry.

Send all correspondence to: Dr. Enrique Bimstein, Department of Pediatric Dentistry, UFCD, P. O. Box 100426, Gainesville, Florida, 32610-0426

Telephone: 352 392 4131
Fax: 352 392 8195

E mail: ebimstein@dental.ufl.edu

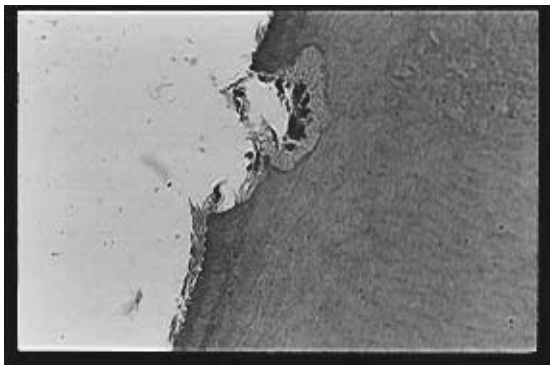

Figure 1. Histological section of the root surface of a primary tooth showing a regular cementum layer and abnormal active root resorption involving the cementum and dentin (H & E, original magnification X 100).

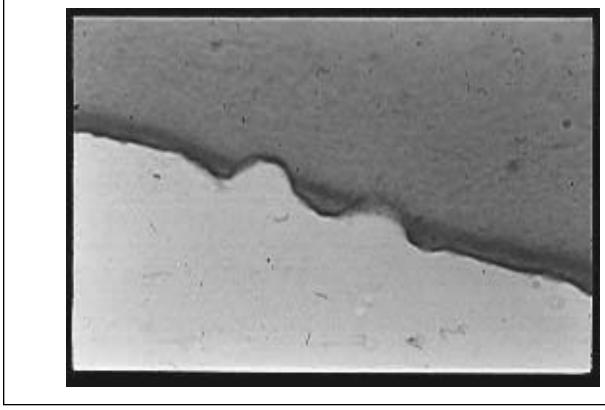

Figure 2. Histological section of a root surface of a primary tooth showing a areas of cementum narrowing and abnormal root resorption (H & E, original magnification X 100).

inadequate restorations and evidence of food impaction,[23] and the systemic condition of the child were recorded. The teeth with periodontitis associated to systemic diseases included 3 teeth from 1 child with Down syndrome, 15 teeth from 1 child with leukocyte adhesion deficiency, and 1 tooth each from 4 children with hypophosphatasia; 2 of them being permanent.

For the purpose of light microscopy, the teeth were firmly wrapped in gauze, decalcified by suspending the gauze-wrapped specimens in a constantly stirred EDTA solution, dehydrated and embedded in paraffin. Consecutive sections of 7 micron thick were cut in the buccal-lingual plane, mounted on glass slides and stained with hematoxylin and eosin. Preliminary data demonstrated that the minimal length and depth of the cementum abnormal resorption lacunae was 9 and 20 microns respectively. Therefore, we concluded that 7 microns sections were small enough to include all the root resorption areas present.

The light microscopy examination of the primary teeth included the root surfaces which were not located adjacent to the permanent successor, i.e., the mesial surface of the mesial root of a mandibular first molar that was not expected to be affected by the physiological root resorption. The following histological features were recorded, when present in one or more histological sections per tooth: a regular cementum layer (Figure 1); areas of thicker or narrower than normal cementum layer (Figure 2); cementum aplasia (Figure 3); root resorption lacunae (Figures 1 and 2). The root resorption areas were characterized based on the classification by Henry and Weinmann,[24] modified by Bimstein *et al*:[25] a) active resorption, when a resorption line and cementoclast were located in the resorption lacunae; b) active repair, when cementum, a cementoid layer and cementoblasts were present over an area that had been resorbed; c) aplastic state, where active resorption had ceased and cementum deposition had not begun; d) anatomic repair, when the root surface had been restored to its original form following previous resorption; e) functional repair, when the cementum had been restored without the original outline of the root;

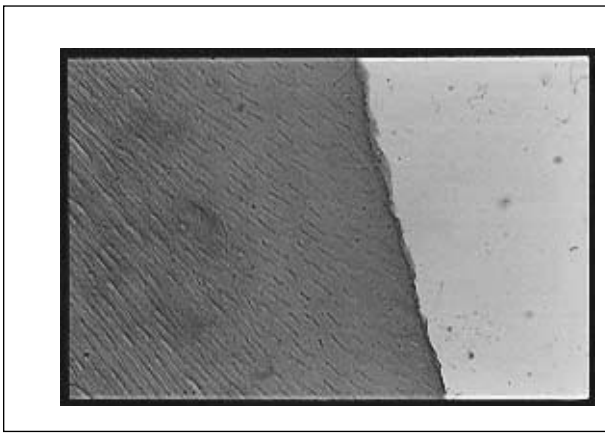

Figure 2. Histological section of a root surface of a primary tooth showing a areas of cementum narrowing and abnormal root resorption (H & E, original magnification X 100).

f) alternate resorption and repair, when > 1 resorption area was evident, the outermost resorption encroaches repaired formerly resorbed areas, and a mosaic pattern was produced by alternating resorption and repair areas.

For the statistical analysis a standard statistical computer program (JMP, version 5, 1989-2002, SAS Institute Inc. Cary NC, USA) was utilized. Analysis of variance (ANOVA) was used to examine the statistical significance of the differences in distribution of the abnormal cementum width areas, presence and types of abnormal root resorption types by the periodontal or the systemic conditions.

RESULTS

The distribution of cementum width characteristics by the periodontal and the systemic conditions is presented in Table 2. Analysis of the distribution of cementum width irregularities by the periodontal condition indicated statistically significant differences (ANOVA, p=0.03): a regular cementum layer was evident in 100% of the control and chronic periodontitis teeth, in 62.5% of the teeth with aggressive periodontitis, and in 63.6% of the teeth from children with a systemic disease. Cementum aplasia was found only in children

Table 2. Number of primary teeth with the various cementum width characteristics by the periodontal and systemic conditions.

		Cementum width characteristics			
		Normal	Areas narrower than normal	Areas wider than normal	Absent
Periodontal condition*	Control	10	0	0	0
	ChP*	5	0	0	0
	AP**	10	5	1	0
	PS***	14	4	0	4
Systemic condition**	Healthy	25	5	1	0
	LAD****	13	2	0	0
	Hypophosphatasia	0	0	0	4
	Down syndrome	1	2	0	0

*Chronic periodontitis, **aggressive periodontitis,
***periodontitis associated with a systemic disease,
****leucocyte adhesion deficiency.

Table 3. Number of primary teeth with or without root resorption lacunae in the root surface not adjacent to the permanent tooth, by the periodontal and systemic conditions.

		Abnormal root resorption	
		Present	Absent
Periodontal condition*	Control	2	8
	ChP*	4	1
	AP**	5	11
	PS***	9	13
Systemic condition*	Healthy	11	20
	LAD****	5	10
	Hypophosphatasia	2	2
	Downs	2	1

*Chronic periodontitis, **aggressive periodontitis,
***periodontitis associated with a systemic disease,
****leucocyte adhesion deficiency.

with a systemic disease related periodontitis Analysis of the distribution of cementum width characteristics by the various systemic conditions indicated statistically significant differences (Table 2, ANOVA, p=0.0001): cementum aplasia was found only in the children with hypophosphatasia, areas of narrower than normal cementum were found in 16.3% of the teeth from children with no systemic diseases, 66.7% of the teeth from the child with Down syndrome, and 13.3% of the teeth from the child with LAD.

The distribution of teeth with abnormal resorption lacunae by the periodontal and the systemic conditions is presented in Table 3. Statistical analysis of the differences in distribution of abnormal resorption lacunae of the periodontal or systemic condition indicated statistically not significant differences (ANOVA, p>0.05).

DISCUSSION

Cementum defects may result in an abnormal attachment, which is highly susceptible to invasion by periodontal pathogens.[17, 18] Therefore, defective cementum has been related to the extensive attachment loss that takes place in cases of hypophosphatasia, in which cementoblasts may either fail to develop or are incapable of elaborating an organic matrix which can calcify.[1, 2, 8, 9] The present finding of cementum aplasia in primary and permanent teeth from children with hypophosphatasia is consistent with previous ones that indicate that in children with hypophosphatasia the severe periodontal disease is related to a lack of cementum.[1-3, 8, 9] The fact that the cementum aplasia is found in the whole root of the teeth from children with hypophosphatasia, regardless if the whole root is already affected or not by the periodontal disease, supports the concept that cementum aplasia is an etiologic factor for the establishment of periodontitis and not its consequence. Cementum aplasia has been previously described to be present in adolescents with aggressive periodontitis,[19] in the present study however, cementum aplasia was limited to the teeth from the children with hypophosphatasia.

Previous findings suggest that root surfaces of primary teeth from children with systemic diseases, such as LAD or PLS may have cementum hypoplasia or aplasia.[5, 20] The present findings confirm that areas of cementum narrowing may be found in teeth from children with LAD and Down syndrome. However cementum narrowing was also found in children with no evidence of systemic or periodontal diseases. Therefore we may consider that cementum hypoplasia may be related to at least 3 possibilities: a) a relatively benign undisclosed systemic disease which affects the resorption-deposition of the cementum; b) a genetic pattern, as indicated by the evaluation of members of the same family;[4, 12, 26, 27] c) the cementum layer being affected by periodontal disease as may be observed in Figure 2; cementum narrowing or complete resorption most likely will facilitate the rapid progression or recurrence of the periodontal disease.

Previous manuscripts report the presence of resorption lacunae in primary teeth affected with AP[28, 29] or hypophosphatasia,[2, 3, 8] and in permanent teeth affected with aggressive periodontitis,[19, 30] periodontitis related to LAD,[26] and chronic neutropenia,[6] indicating that resorption lacunae are common findings when extensive alveolar bone loss takes place. The present finding that abnormal resorption lacunae was evident mostly in teeth from children affected with aggressive periodontitis and severe periodontitis associated with hypophosphatasia and LAD is consistent with previous findings. The fact that resorption lacunae was also found in control teeth, may be related to ectopic eruption of an adjacent permanent tooth.[31]

Radiographic evidence of atypical root resorption of deciduous teeth has been previously reported in children with hypophosphatasia and in otherwise healthy children with AP.[8, 12, 25, 28, 29, 32] However, the atypical root resorption is not a common finding, and even in cases where it takes place it does not affect every tooth or even every root in the same tooth.[12, 25] These findings suggest that atypical root resorption of primary teeth may take place in the areas affected by severe periodontal disease in which the cementum repair is unable to compensate for the resorption.

CONCLUSION

Cementum narrowing or cementum aplasia may be related to the establishment, severity and progress of periodontitis in children.

REFERENCES

1. Bruckner RJ, Rickles NH, Porter DR. Hypophosphatasia with premature shedding of teeth and aplasia of cementum. Oral Surg Oral Med Oral Pathol, 15: 1351–1369, 1962.
2. Beumer J, Trowbridge HO, Silverman S, Jr, Eisenberg E. Childhood hypophosphatasia and the premature loss of teeth. Oral Surg Oral Med Oral Pathol, 35: 631–640, 1973
3. Baab DA, Page RC, Morton T. Studies of a family manifesting premature exfoliation of deciduous teeth. J Periodontol, 56: 403–409, 1985.
4. Baab DA, Page RC, Ebersole JL, Williams BL, Scott CR. Laboratory studies of a family manifesting premature exfoliation of deciduous teeth. J Clin Periodontol, 13: 667–683, 1986.
5. Vrahopoulus TP, Barber P, Liakoni H, Newman HN. Ultrastructure of the periodontal lesion in a case of Papillon-Lefevre syndrome (PLS). J Clin Periodontol, 15: 17–26, 1988.
6. Carrassi A, Abati S, Santarelli G, Vogel G. Periodontitis in a patient with chronic neutropenia. J Periodontol, 60: 352–357, 1989.
7. Bimstein E, Lustman J, Sela MN, Ben Neria Z, Soskolne WA. (1990) Periodontitis associated with Papillon Lèfevre Syndrome. J Periodontol, 61: 373–377, 1990.
8. Plagman H.-C, Kocher T, Kuhrau N, Caliebe A. Periodontal manifestation of hypophosphatasia A family case report. J Clin Periodontol, 21: 710–716, 1994.
9. Olson A, Matsson L, Blomquist HK, Larsson Å, Sjödin B. Hypophosphatasia affecting the permanent dentition. J Oral Pathol Med, 25: 343–347, 1996.
10. De Vree H, Steenackers K, De Boever JA. Periodontal treatment of rapid progressive periodontitis in 2 siblings with Papillon-Lefèvre syndrome: 15-year follow-up. J Clin Periodontol, 27: 354–360, 2000.
11. Lopez-Perez R, Borges-Yanez SA, Jimenez-Garcia G, Maupome G. Oral hygiene, gingivitis, and periodontitis in persons with Down syndrome. Spec Care Dentist, 22, 214–220, 2002.
12. Bimstein E. Extended kindred with 10 children with periodontitis: a seven-year follow-up report. Pediatr Dent, 25: 389–396, 2003.
13. Page RC, Sims TJ, Delima AJ, Bimstein E, Needleman HL, Van Dyke TE. The relationship between periodontitis and systemic diseases and conditions in children, adolescents and young adults. In *Periodontal and Gingival Health and Diseases. Children, Adolescents and Young adults*, eds. Bimstein E, Needleman, LH, Karimbux, N, Van Dyke, TE. London UK, Martin Dunitz, 2001:107–143.
14. Sjödin B, Crossner CG, Unell L, Östlund P. A retrospective radiographic study of alveolar bone loss in the primary dentition in patients with juvenile periodontitis. J Clin Periodontol, 16: 124–127, 1989.
15. Sjödin B, Matsson L, Unell L, Egelberg J. Marginal bone loss in the primary dentition of patients with juvenile periodontitis. J Clin Periodontol, 20: 32–36, 1993.
16. 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. J Periodontol, 65: 631–635, 1994.
17. Lindskog S, Blomlöf L. Cementum hypoplasia in teeth affected by juvenile periodontitis. J Clin Periodontol, 10: 443–451, 1983.
18. Page R, Baab D. A new look at the etiology and pathogenesis of early onset periodontitis. J Periodontol, 56: 748–751, 1985.
19. Blomlöf L, Hammarstrom L, Lindkog S. Occurrence and appearance of cementum hypoplasias in localized and generalized juvenile periodontitis. Acta Odontol Scand, 44: 313–320, 1986.
20. Waldtrop TC, Hallmon WW, Mealey BL. Observations of root surfaces from patients with early-onset periodontitis and leukocyte adhesion deficiency. J Clin Periodontol, 22: 168–178, 1995.
21. Bimstein E, Shapira L, Landau E, Sela MN. The relation between alveolar bone loss and proximal caries in children: prevalence and microbiology. ASDC J Dent Child, 60: 99–103, 1993.
22. AAPD Reference Manual. Periodontal Diseases of Children and Adolescents. Pediatr Dent, 28: 204–211, 2006–2007.
23. Bimstein E. Radiographic diagnosis of the normal alveolar bone height in the primary dentition. J Clin Pediatr Dent, 19: 269–271, 1995.
24. Henry JL, Weinmann JP. The pattern of resorption and repair of human cementum. JADA, 42, 270–290, 1951.
25. Bimstein E, Wagner M, Nauman RK, Abrams RG, Shapira L. Root surface characteristics of primary teeth from children with prepubertal periodontitis. J Periodontol, 69: 337–347, 1998.
26. Shapira L, Schlesinger M, Bimstein E. Possible autosomal-dominant inheritance of prepubertal periodontitis in an extended kindred. J Clin Periodontol, 24: 388–393, 1997.
27. Bimstein E, Sela MN, Shapira L. Clinical and microbial considerations for the treatment of an extended kindred with 7 cases of prepubertal periodontitis: an 2 year follow up. Pediatr Dent, 19: 396–403, 1997.
28. Myers DR., O'Dell NL, Clark JW, Cross RL. Localized prepubertal periodontitis: literature review and case report. ASDC J Dent Child, 56: 107–111, 1989.
29. Lopez NJ, Gigoux C, Canales ML. Histological differences between teeth with adult periodontitis and pre-pubertal periodontitis. J Periodontol, 61: 87–94, 1990.
30. Douglas KD, Cobb CM, Berkstein S, Killoy WJ. Microscopic characterization of root surface-associated microbial plaque in localized juvenile periodontitis. J Periodontol, 61: 475–484, 1990.
31. Barberia-Leache E, Suarez-Clua MC, Saavedra-Ontiveros D. Ectopic eruption of the first permanent molar: characteristics and ocurrence in growing children. Angle Orthod, 75: 610–615, 2005.
32. Goepferd SJ. Advanced alveolar bone loss in the primary dentition. J Periodontol, 52, 753–57, 1981.

Acknowledgement: This study was supported by a University of Florida College of Dentistry Seed Research Grant.