CLINICAL
reports

Periodontal disease in healthy children: two clinical reports

Ronald B. Cogen, DDS, PhD   Wesam Al-Joburi, BDS, MS
Page W. Caufield, DDS   Harry P. Stanley, DMD   Kerwin Donaldson, DDS

Abstract

Cases of five black pediatric patients are presented with clinical evidence of advanced alveolar bone resorption, involving both the primary and permanent dentitions. The amount of plaque accumulation and severity of gingival inflammation was not consistent with the observed advanced periodontal destruction. Accordingly, a diagnosis of juvenile periodontitis (JP) was made. It is assumed widely that JP does not affect the primary dentition in the absence of contributing systemic disease. In two of the five patients, complete medical and laboratory diagnostic procedures ruled out the presence of any known contributory diseases. In the other three patients a significant role of systemic disease was eliminated on the basis of their medical histories. The cases presented clearly demonstrate that JP can involve the primary as well as the permanent dentitions.

Classically, juvenile periodontitis (JP) has been defined as "a disease of the periodontium occurring in an otherwise healthy adolescent and characterized by a rapid loss of the alveolar bone around more than one tooth of the permanent dentition."[1] Convincing evidence exists implicating specific bacteria, namely Capnocytophaga[2,3] and *Actinobacillus actinomycetemcomitans (Aa),*[4,5] in the etiology of JP.

In addition to their intrinsic pathogenicity, these organisms adversely affect polymorphonuclear leukocyte (PMN) function,[6,7] hence altering host defenses. Although the exact role of microbial interaction with PMN in the etiology of JP is uncertain, the association of depressed leukocyte function with JP is well established.[8-11] It also is possible that cell-mediated imunity is depressed, based on decreased T-lymphocyte blast transformation in the presence of gram-negative organisms obtained from lesions of JP patients.[12]

An association appears to exist between rapidly destructive forms of periodontal disease and certain hereditary diseases.[13,14] Frequently, a familial pattern is evident in patients with JP.[15-19] The heritable nature of JP can be assumed further from the reported association of ABO blood groups and JP,[20,21] and also from the negative association of HLA-A2 phenotypes in patients afflicted with JP.[22,23]

Virtually all literature reports suggest that JP is limited to the permanent dentition in otherwise healthy patients. Destructive periodontal disease in children, especially of the primary teeth, appears to be rare. Two recent clinical reports, however, show JP with advanced alveolar bone loss occurring in the primary dentition of systemically healthy children.[24,25]

Clinical Reports

Patient 1

N.H., an alert, well-nourished, seven-year-old black female, presented to the Pediatric Dental Clinic at the Children's Hospital of Birmingham, Alabama for comprehensive care. Her past medical history was not remarkable.

The clinical oral examination (Figure 1) revealed a mixed dentition consisting of all primary teeth and the permanent first molars. Plaque and calculus were present and there was moderate gingival inflammation as well as several carious lesions. In addition, there was loss of attachment around all primary teeth and the mesial surfaces of the permanent mandibular first molars; mesial pocket depths ranged from 6 to 7 mm. All primary molars were hypermobile and the furcations were readily probeable as a result of periodontal involvement. This condition was evidenced further by pocket depths which ranged from 5 to 6 mm. The radiographic examination consisted of a panoramic film (Figure 2), and selected periapical films (Figure 3). These radiographs revealed generalized advanced bone loss around all primary teeth
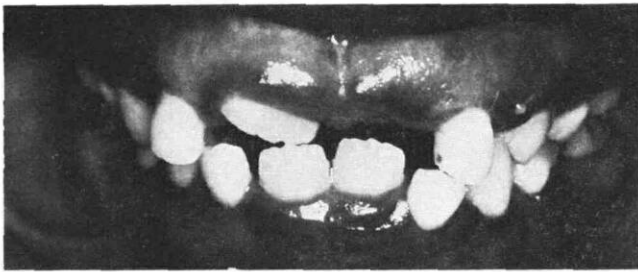
Figure 1. Clinical photograph of patient N.H. showing mixed dentition and mild gingival inflammation. Note also the presence of small amounts of plaque. Clearly the degree of plaque accumulation and inflammation is not consistent with the advanced periodontal destruction which was observed subsequently.
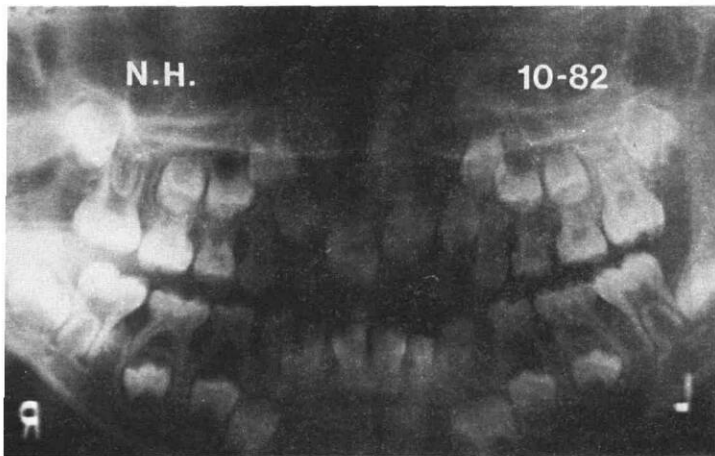

Figure 2. Panorex showing alveolar bone resorption of the maxillary and mandibular arches of the primary and permanent dentitions bilaterally in patient N.H.
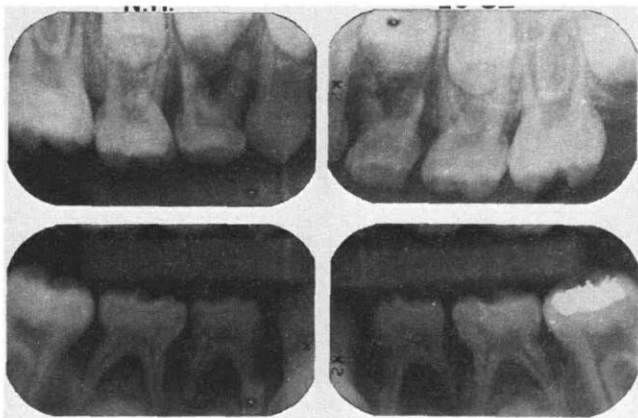

Figure 3. Periapical radiographs of posterior teeth of N.H., illustrating extent of bone resorption affecting the primary molars and mesial aspect of the permanent first molars.

and vertical alveolar bone loss on mesial surfaces of the permanent first molars. Clinical photographs were obtained at the same visit and supragingival scaling and polishing as well as oral hygiene instructions followed.

Because the pronounced bone loss involved both the primary and permanent dentitions, the patient was referred for a complete medical evaluation to confirm or rule out any underlying systemic disease. Conditions included in a differential diagnosis which could have accounted for the alveolar bone loss in the primary dentition and the permanent molars were: hypophosphatasia, Papillon-Lefèvre syndrome, histiocytosis X, neutropenia, cyclic neutropenia, leukemia, diabetes mellitus, scleroderma, fibrous dysplasia, and acrodynia.

Results of the medical evaluation and the laboratory values were all within the normal range. The blood count revealed 5.0×10^6 red blood cells/mm^3, and 6.3×10^3 leukocytes/mm^3. The hemoglobin, hematocrit values, and mean corpuscular volumes were all within normal limits, as were the differential leukocyte count, serum levels of calcium, phosphate, and alkaline phosphatase. The urinalysis values were also within normal limits and the skull and skeletal radiographic surveys revealed no abnormalities.

N.H. was evaluated further and treated for the next three months. Treatment consisted of extractions, subgingival scaling, additional oral hygiene instructions, and restoration of caries lesions. Further evaluation included culturing of several pathologic sites for anaerobic bacteria as described by Slots, et al.[5] Cultures revealed the presence of Aa from all sites sampled. Also, a wedge biopsy of the periodontium obtained along with extractions was submitted for histologic examination. The resulting diagnosis was chronic periodontal inflammation.

As a result of the clinical periodontal examination, medical evaluation and laboratory values, radiographic picture, bacterial profile, and histologic sections, N.H. was diagnosed as having JP, involving both the primary dentition and permanent first molars in the absence of any recognizable systemic diseases.

Patient 2

E.R., an alert, well-nourished, 10-year-old black male, presented to Children's Hospital of Birmingham, Alabama for comprehensive dental care. He had been treated sporadically at the Dental Clinic since 1979 and his medical history was unremarkable.

The clinical oral examination (Figure 4) revealed a mixed dentition with permanent first molars, four mandibular incisors, maxillary central incisors, and maxillary left lateral incisor. Plaque was present and there was slight gingival inflammation as well as several carious lesions. There was loss of periodontal attachment around most of the primary teeth, the mesial surfaces of the four permanent first molars, and the three maxillary permanent incisors. Most of the pocket depths were 5.0 mm ranging from 4 to 6 mm. Radiographs confirmed the clinical evaluation, revealing generalized bone loss around primary teeth, as well as bone loss localized to the mesial surfaces of the permanent first molars (Figure 5).

Development of the periodontal lesions can be observed and followed on the radiographs illustrated in
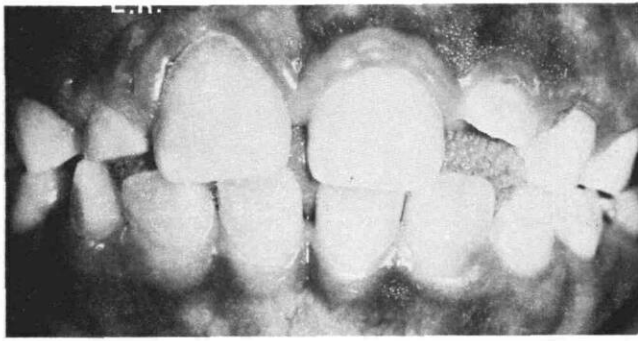
Figure 4. Clinical photograph of patient E. R. showing mixed dentition, with mild gingivitis and small amount of accumulated plaque. Clinical appearance was inconsistent with the determination of advanced alveolar bone loss.
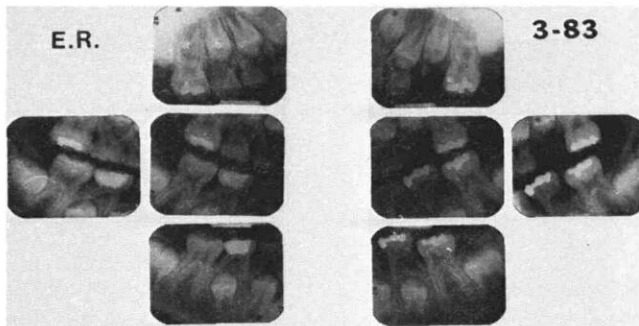

Figure 5. Bitewing and periapical radiographs of E.R. taken at the time of presentation to the clinic. They illustrate the advanced nature of the periodontal destruction.
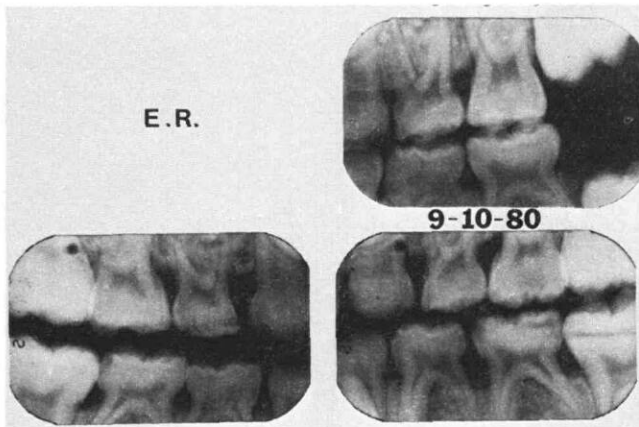

Figure 6. Bitewing radiographs of E. R. taken in May, 1979, and September, 1980, which clearly show alveolar bone loss and progressive worsening of the lesions with time.

Figure 6. The radiograph taken four years earlier (1979) suggests developing lesions involving alveolar bone on the mesial side of the maxillary left first molar. Radiographs taken three years previously (1980) clearly show lesions involving all four permanent molars and several of the primary molars. Longitudinal comparisons indicate rapid progression of the lesions in the interval of one year. The radiographs taken in 1983 (Figure 5), illustrate the rapid progress of alveolar bone destruction as a result of advanced periodontal disease.

The patient was referred for a complete medical evaluation to confirm or rule out any underlying systemic disease which may have contributed to the destructive periodontal disease observed in both the primary and permanent teeth. The results of the medical evaluation and the laboratory values were within the normal ranges. The blood count revealed 5.6×10^6 red blood cells/mm^3 and 4.0×10^3 leukocytes/mm^3. The hemoglobin, hematocrit values, and mean corpuscular volumes were all within normal limits as were the differential leukocyte count, serum levels of calcium phosphate, and alkaline phosphatase. Urinalysis values were within normal limits, and skull and skeletal radiographs revealed no abnormalities. Further evaluation included bacterial cultures from several diseased sites. These cultures revealed Aa in the periodontal pockets.

As a result of the clinical periodontal examination, medical evaluation and laboratory values, radiographic features, and microbiota recovered from diseased sites, a diagnosis of JP was made. Similar to N.H., E.R. exhibited a variation of JP which involved both the primary and permanent dentitions without contribution of systemic diseases. Treatment consisted of extractions, subgingival scaling, additional oral hygiene instructions, and restoration of carious lesions.

Patients 3, 4, and 5

Three additional children have been brought to our attention who apparently suffered from a form of destructive periodontal disease affecting both primary and permanent dentitions. U.H., a 12-year-old black female, and T.H., her 14-year-old brother, were deemed systemically healthy from their unremarkable medical histories. J.J., a 12-year-old black male, also had an unremarkable medical history. He also had a medical evaluation by a physician which uncovered no systemic diseases. It should be noted, however, that none of these patients were subjected to the same medical examination performed on patients in cases 1 and 2. With this reservation however one is tempted to assume the absence of complicating systemic disease in the etiology.

The radiographic evidence of periodontal destruction seen in Figures 7, 8, and 9, span several years in all three youngsters, and is evident in both the primary and permanent dentitions. Also, J.J. was examined clinically by a periodontist and was found to have pocket depths ranging from 5 to 7 mm in lower central incisors, 4 to 6 mm in maxillary canines and maxillary and mandibular premolars, and 5 to 9 mm in the molars. This clinical picture suggests the generalized form of JP.

Discussion

Inflammatory periodontal disease is common in children; however, the inflammation usually is limited to the gingiva and usually does not result in loss of attachment or resorption of alveolar bone. In cases of destructive periodontal disease in young people, onset is usually circumpubertal and is termed juvenile periodontitis.
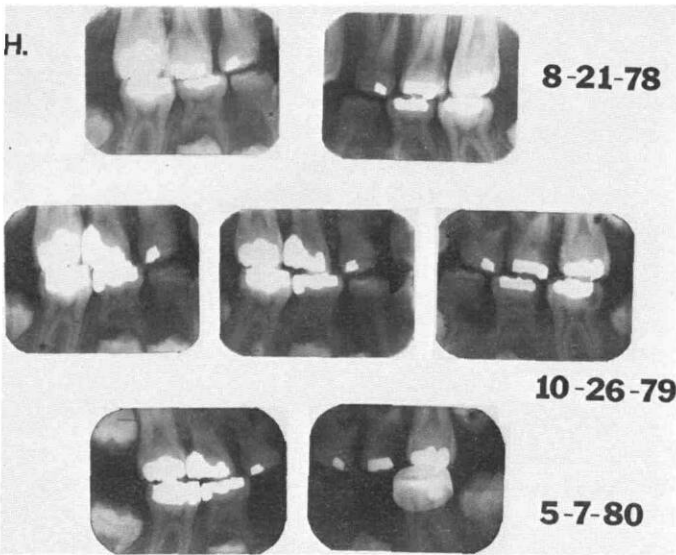
Figure 7. Bitewing radiographs of patient U.H. observed over a period of three years. These show sequential alveolar bone resorption affecting both the primary and permanent dentitions.
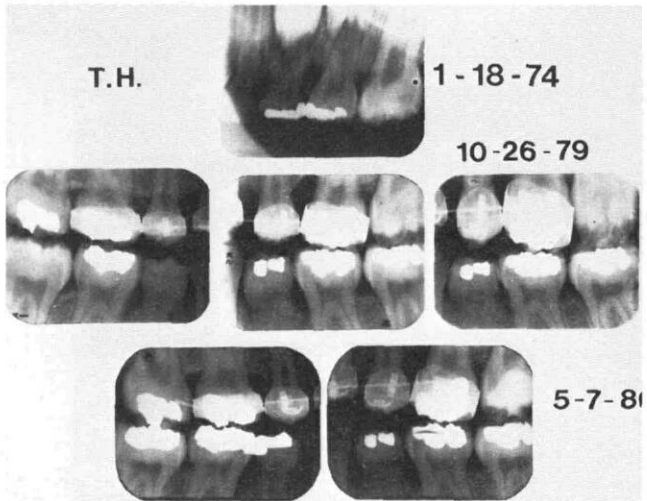

Figure 9. Periapical and bitewing radiographs of J.J. which span a six-year period. Once again the progressive nature of alveolar bone loss which involves both primary and permanent dentitions is obvious. The alveolar bone destruction can be observed as early as 1977 in this sequence.

In the past, the disease was known as periodontosis; again, it seems likely that the definition of JP quoted in the introduction of this paper needs to be updated on at least two counts.

Figure 8. Periapical and bitewing radiographs of T.H., a brother of U.H. These allow observation of progressive alveolar bone loss over a period of seven years and illustrate involvement of both dentitions.

First, now it is accepted commonly that most JP patients suffer polymorphonulear leukocyte dysfunction,[8-11] and so are not, strictly speaking, "healthy." Other etiologic agents which may play a role in JP include the presence of specific bacteria, immunologic deficiencies, and genetic predisposition.

Second, this study and others,[24,25] amply illustrate that JP does indeed occur in the prepubertal child and affects the primary as well as permanent dentitions. In this report at least two of the patients, N.H. and E.R., and probably all five of the reported subjects were free of systemic diseases previously considered necessary for the occurrence of JP in primary teeth. This contention is supported by previous studies,[24,25] which found no evidence of complicating systemic conditions that might be contributory to the destructive periodontal disease. It seems inconsistent moreover that the etiologic factors suggested to be present would manifest only in the permanent dentition. At present it is difficult to explain pathogenic mechanisms involving specific bacteria, immunodeficiencies, leukocyte dysfunction, and genetic predispositions which manifest in the permanent and not the primary dentitions.

A more likely explanation is that a diagnosis of JP is not being made when it involves the primary dentition — for several reasons. First, by definition the disease is not thought to occur in prepubertal children; second, loosening and loss of primary teeth are considered normal occurrences; third, it appears that the prevalence of JP may be largely in the black community, and it may be that these patients have had less access in the past to dental services, especially pediatric dentistry.

It is tempting to speculate that JP and its possible prepubertal onset is really more common than previously was supposed. Increased understanding of this disease will lead to earlier diagnosis, facilitating treatment and thereby improving the prognosis.

Diagnosis and treatment early in the course of the disease is extremely important for a successful out-
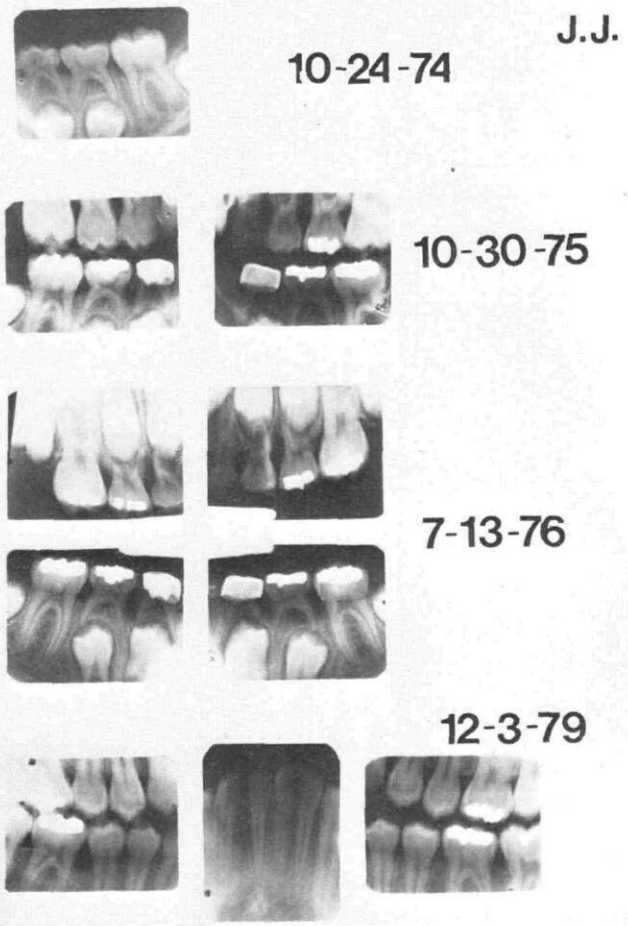
come.[26] Although not enough data has been collected to be able to predict long-term success with confidence,[27] several approaches do appear promising. These JP patients frequently display good resolution to a regimen of thorough scaling, curettage, and meticulous daily plaque control — especially if combined with antibiotics such as tetracycline at a standard dose for 14 days.[28,29] As an alternative to antibiotics, surgical curettage combined with scaling and meticulous home care may be attempted. In the young child thorough scaling, curettage, and meticulous daily plaque control may be sufficient. However, it should be emphasized that regardless of the modality chosen, frequent recalls at two- to three-month intervals, should be established. Progress of the disease can thus be monitored carefully and alternative therapies established if needed.

Conclusion

These clinical reports suggest that the traditional definition of periodontitis or juvenile periodontitis may be incorrect. The dental evaluations indicated advanced destructive periodontal disease of both primary and permanent dentitions in the absence of any of the systemic diseases thought to contribute to such destructive periodontal disease. The amount of plaque present as well as the ages of the patients were clearly inconsistent with the observed alveolar bone loss. Moreover, the extent of bone loss involving both crestal regions and furcations was pathologic and could not be deemed to result from a process of normal exfoliation.

Thus, rapidly destructive periodontitis does indeed affect pediatric patients. Recognition of this will lead to earlier diagnosis and more successful treatment of this potentially disfiguring disease.

Dr. Cogen is an associate professor, and Dr. Al-Joburi is an instructor, Department of Periodontics, University of Alabama School of Dentistry, Birmingham, Ala. 35294. Dr. Caufield is an assistant professor, and Dr. Stanley is a resident, Department of Pediatric Dentistry, University of Alabama School of Dentistry. Dr. Donaldson is a practicing pedodontist in Alexandria, La. Requests for reprints should be sent to Dr. Cogen.

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