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Prepubertal periodontitis: a recently defined clinical entity

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Abstract

A classification for advanced alveolar bone loss in children and adolescents is presented for the purpose of clarifying the oftentimes confusing terminology that presently exists. Page's definiton of prepubertal periodontitis is advocated, and a representative case is presented.

The classic definition of periodontosis is "a disease of the periodontium occurring in an otherwise healthy adolescent, which is characterized by a rapid loss of alveolar bone about more than 1 tooth of the permanent dentition. The amount of destruction manifested is not commensurate with the amount of local irritants present."[1] The rate of destruction is 3–4 times the rate of progression of adult periodontitis. One form affects the first permanent molars and incisors, and a more generalized form may affect most of the permanent dentition. Most of the dental community now refers to periodontosis as juvenile periodontitis (JP).

Page[2] recently has established specific diagnostic criteria to distinguish true periodontosis (juvenile periodontitis), as defined by Baer[1], from periodontosis of the primary dentition in young children, which he terms prepubertal periodontitis (PP). According to Page, PP occurs in localized and generalized forms.

The onset of PP is during or immediately after eruption of the primary teeth. The prevalence is unknown, but probably rare, and there is a possibility of a genetic basis for some types of the disease. PP may be followed by severe periodontitis of the permanent teeth or by a normal permanent dentition.

In the localized form of PP, some but usually not all of the primary teeth are affected. The onset of the disease occurs around—or even before—the age of 4 years. The gingival tissue manifests only minor inflammation, if any, and microbial plaque is minimal. Alveolar bone destruction proceeds more rapidly than in adult periodontitis or chronic periodontitis in teenagers. Otitis media is not a frequent finding, and usually there is no history of frequent infections. Functional defects are present in either neutrophils or monocytes, but not both. The disease is amenable to treatment by curettage, antibiotic therapy, and improved toothbrushing.

The hallmarks of generalized PP include a fiery red, acute inflammation pervading the marginal and attached gingiva around all the teeth, gingival proliferation, cleft formation, and recession. Onset is at the time of or soon after tooth eruption. Alveolar bone destruction sometimes is accompanied by destruction of the tooth roots and proceeds at an alarming rate. The primary teeth may be lost by age 2–3 years. The affected children suffer from otitis media and recurrent, sometimes life-threatening, infections. This form of the disease seems to be refractory to antibiotic therapy. Both neutrophils and monocytes are profoundly abnormal in this form of the disease, and the peripheral blood white cell count is elevated markedly.

Traditional definitions of periodontosis or juvenile periodontitis exclude involvement of the primary dentition. A few cases of significant alveolar bone loss affecting the primary dentition of apparently healthy children have been reported in the dental literature under such designations as juvenile periodontitis[3], juvenile periodontosis[4], Pleasants' disease[5], periodontosis of the primary dentition[6], and advanced alveolar bone loss in the primary dentition.[7] Boraz[8] recently reported a case as PP, but used the term interchangeably with JP.

The relationship between JP and PP is not understood at the present time. Spektor[9] reported on a very large family (13 living children) whose mother had rapidly progressive periodontitis. Five of the children had JP and 2 had PP, indicating a very close

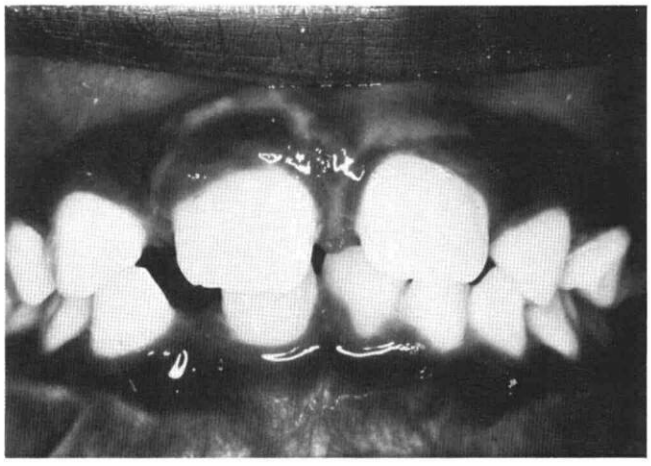

FIG 1. Representative area showing slightly edematous nature of the gingival tissues.

relationship between these diseases and possibly a common underlying mechanism. JP was not always preceded by PP, although 1 sister with PP did go on to manifest JP at age 15. In her case the alveolar bone around the primary molars had been destroyed, but it regenerated as the permanent premolars erupted. The idea that JP has its onset during the circumpubertal period is supported by the fact that 7 of the postpubertal family members had periodontitis, while only 2 of 6 prepubertal children manifested the disease.

Case Report

A 7-year-old, well-developed black male presented to the pediatric dentistry department at the Medical University of South Carolina because of parental concern for the malaligned anterior teeth. The patient was well nourished, in no apparent distress, and there was no history of recurrent infections.

Clinical examination revealed no caries, but 5–7 mm periodontal pockets involving the primary dentition. The first primary molars and maxillary canines exhibited hypermobility. The gingival tissues were slightly edematous and erythematous (Fig 1). Profuse sulcular bleeding occurred upon gentle probing. Plaque deposits were minimal, but subgingival calculus deposits could be detected.

Radiographic examination revealed significant vertical bone loss about the primary teeth (Fig 2), but not the permanent teeth. All primary molars except the mandibular right second molar revealed loss of bone into the furcation.

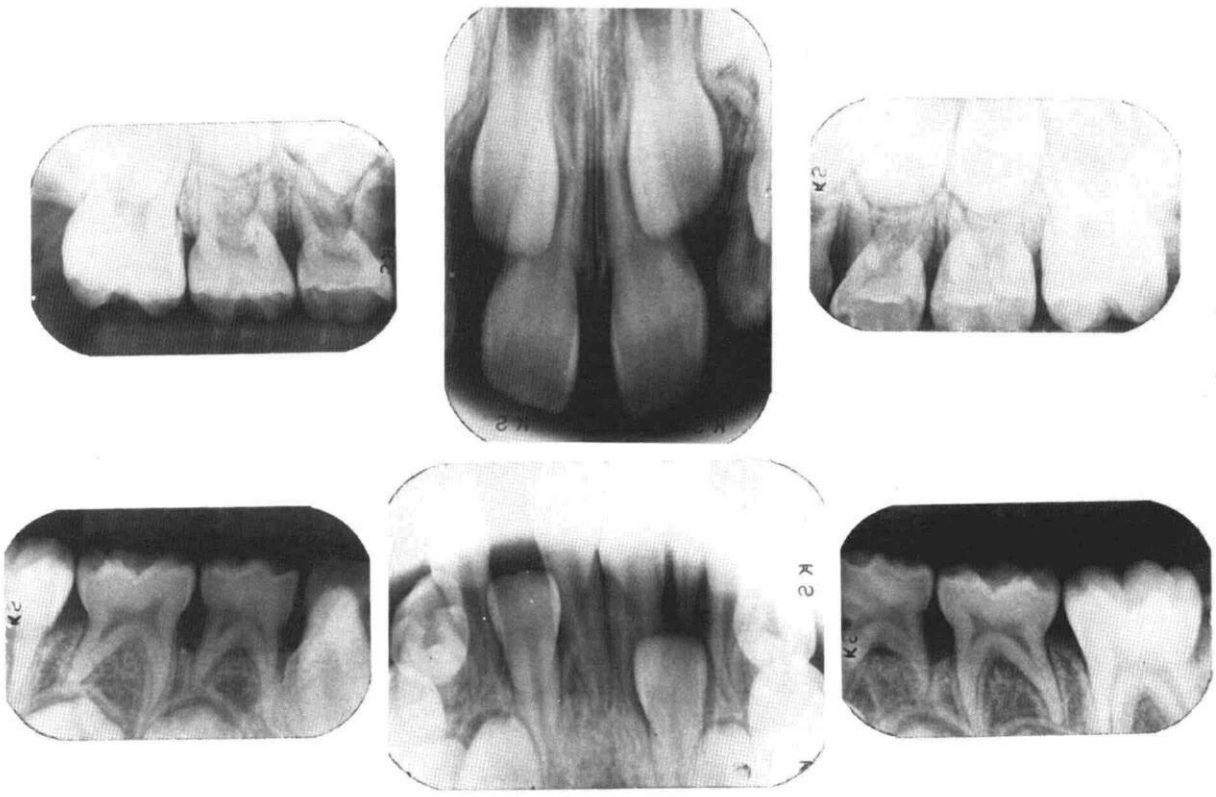

FIG 2. Radiographic survey showing bone resorption about the primary teeth, but not the permanent teeth. Clockwise from upper left: maxillary right posterior, maxillary anterior, maxillary left posterior, mandibular left posterior, mandibular anterior, mandibular right posterior.

The family history was negative except for an aunt who had scleroderma. Laboratory studies consisting of an SMA-20, CBC, and differential, were within normal limits.

A microbiological sample of a representative pocket was taken with a sterile curette and processed anaerobically according to the procedures outlined by Slots.[10] The predominant cultivable microorganisms were: *Haemophilus actinomycetemcomitans* (*Ha*), 12%; *Bacteroides gingivalis*, 23%; *B. melaninogenicus* subspecies *intermedius*, 8%; *Fusobacterium nucleatum*, 9%; *S. sanguis*, 27%; and *S. mutans*, 19%.

Despite the fact that leukocyte function tests were not carried out because of the high cost, a diagnosis of localized PP as described by Page[2] was made.

Treatment has consisted of oral hygiene instruction, scaling, and root planing, 250 mg of tetracycline elixir b.i.d., and extraction of the involved primary teeth, with placement of appropriate space maintainers. A program of 3-month maintenance scaling and OHI reinforcement will be followed, with tetracycline, an effective antibiotic against *Ha* and other gram-negative anaerobic periodonto-pathogens, utilized to combat acute exacerbations of the disease.

Discussion

The authors recognize that the case presented here would have fit more precisely into the diagnostic criteria of Page had leukocyte function tests been conducted. Since this case exhibited early-onset periodontitis involving the primary teeth only in a prepubertal child with no systemic diseases, it was felt that the diagnosis best fit localized PP.

It is suggested that for the sake of clarity and exactness, advanced alveolar bone loss involving the primary teeth be termed PP as advocated by Page[2], and advanced alveolar bone loss involving the permanent dentition in the circumpubertal period be termed JP, as advocated by Waerhaug[11] and Page.[2]

The finding of significant percentages of *Ha* and *Bacteroides* species in the subgingival plaque of this patient suggests a common link between JP and PP. Slots[10] and Vandesteen[12] have implicated *Ha* and *Bacteroides* species, respectively, as important etiologic agents in JP. *Ha* possesses numerous virulence factors such as leukotoxin,[13] a collagenase,[14] a potent endotoxin,[15] a PMN chemotaxis inhibiting factor,[16] and a soluble extract which can alter human lymphocyte function.[17] Many PP and JP patients, but not chronic adult periodontitis patients, have abnormalities in peripheral blood PMN, monocyte or serum complement function.[12] It is tempting to speculate that these alterations in host-immune response in combination with a particularly virulent microbiota may predispose these patients to rapid alveolar bone destruction.

Accelerated alveolar bone loss about the primary teeth in patients with systemic diseases such as Papillon-Lefèvre and hypophosphatasia should be classified as prepubertal periodontitis associated with that particular systemic disease.

The authors agree with Spektor[9] that there are many pitfalls and problems involved in assigning neat, specific diagnoses to individuals with early-onset periodontitis, and one must expect to encounter patients manifesting features which do not precisely fit any diagnostic criteria. This article has tried to clarify the muddled classification of early-onset periodontitis that presently exists.

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