

Periodontosis of the primary dentition: A case report

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Introduction

The literature suggests that the onset of periodontosis occurs in the adolescent age group, initially affecting only the permanent first molars and incisors. The individuals so affected show no apparent systemic or local factors associated with these dental findings.

Since Gottlieb first described a condition of rapid alveolar bone destruction in 1920, much research has been done in an attempt to identify its etiology.¹

Newman and Socransky have postulated a bacterial etiology for this condition. Using sophisticated culturing techniques, isolates of periodontosis lesions were obtained. A significant number of the organisms obtained were gram-negative anaerobic rods, belonging to the genus caphocytophagia.² When these organisms were introduced to gnotobiotic rats, a characteristic alveolar bone loss was observed.³ In view of these results, it is tempting to assign a significant role for these organisms in the etiology of periodontosis.

Studies by Cianciola *et al.* have found that patients with periodontosis have polymorphonuclear leukocytes with diminished chemotactic and phagocytic responses. They suggest that the organisms implicated by Newman *et al.* may release factors which enter the systemic circulation and adversely affect the polymorphonuclear leukocytes.⁴ A recent study by Shurin *et al.* adds support to this hypothesis. They found the culture medium of caphocytophagia to contain a dialyzable substance which caused normal neutrophils to behave like neutrophils obtained from periodontosis patients.⁵

In addition, some feel that a diminished cellular immune response may also contribute to this condition. Findings of Lehner *et al.* have shown that T-lympho-

cytes from affected individuals do not undergo blast formation when subjected to host gram-negative organisms, indicating impairment of the cellular immune system to these organisms.^{6,7}

Interesting is the fact that these purported etiologies should be felt to affect only the permanent dentition.

Report of a Case

F. G., a well-developed, well-nourished 10-year-old black male presented to our clinic for comprehensive care. His past medical history was unremarkable.

Oral Findings: An intraoral examination revealed gingiva normal in color and configuration. Loss of attachment was evident around all the primary molars with pocket depths of four to six millimeters. These teeth exhibited hypermobility with readily probed furcation involvement. Pocket depths of the first permanent molars and incisors were all one to two millimeters. No suppuration or fistulus tracts were present.

Radiographic Findings: A radiographic exam was remarkable for extensive crestal bone loss (horizontal) around all the primary molars, and severe internal resorption of the distal root of the maxillary left second primary molar. External resorption was evident along the distal surface of the mandibular right first primary molar. Interproximal caries of the primary molars were present in all but the maxillary left quadrant (Figures 1 and 2).

Since the degree of bone loss did not correlate with the local dental findings, the patient was referred for a medical work-up to rule out any underlying systemic disease. The results of the medical exam and all laboratory values were well within normal limits.

A CBC revealed 4.8 RBC (millions/mm³) and 7100 WBC/mm³. The differential leukocyte count was 39% neutrophils, 2.5% eosinophils, 0.6% basophils, 52% lymphocytes, and 8% monocytes. The erythrocyte sedi-

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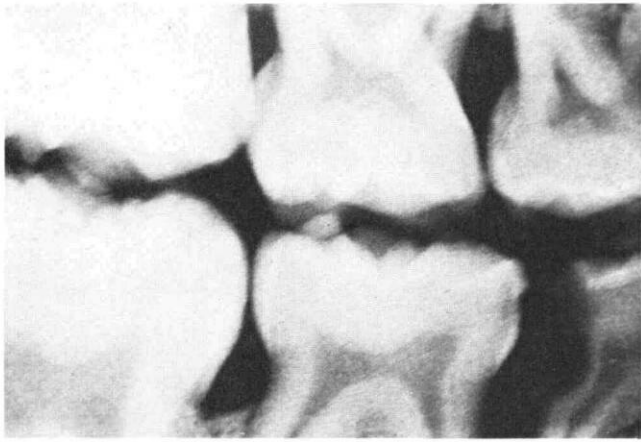


Figure 1

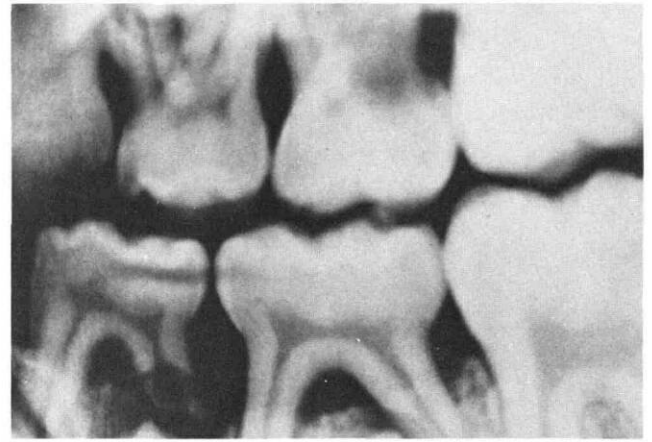


Figure 2

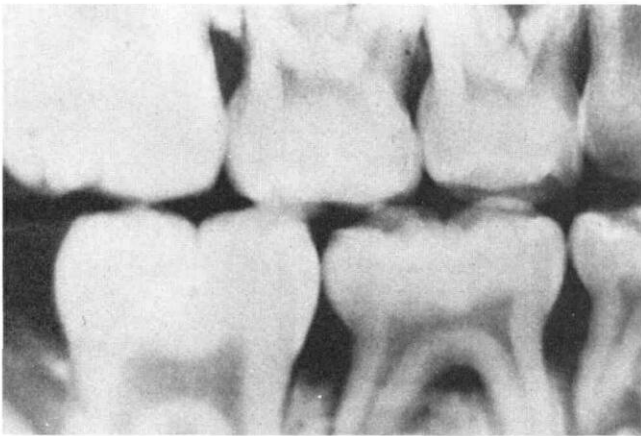


Figure 3

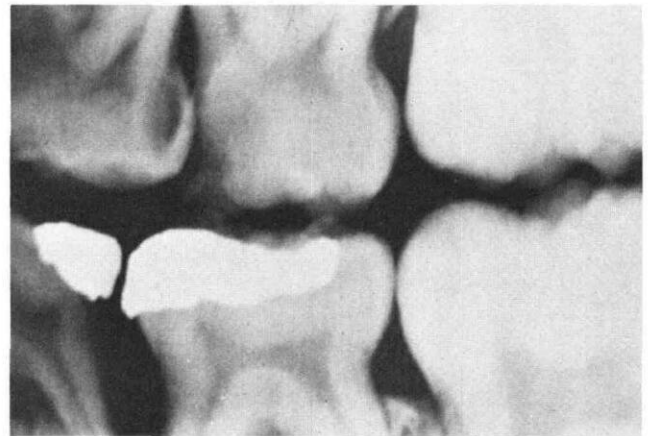


Figure 4

mentation rate, serum phosphate, alkaline phosphatase, calcium, and phosphoethanolamine were all observed to be within normal limits. Urine phosphoethanolamine was also unremarkable.

A wedge biopsy of the periodontium of the mandibular left first primary molar was read as chronic periodontal inflammation, consistent with a diagnosis of periodontitis. An intraoral and radiographic exam of the patient's siblings was remarkable for similar bone loss in the primary dentition of a nine-year-old brother (Figures 3 and 4).

Treatment of both affected children consisted of extraction of all involved primary molars with the placement of appropriate holding appliances with the hope that the erupting premolars would initiate new alveolar bone growth.

Discussion

Periodontal disease in the primary dentition is well documented throughout the dental literature.^{8,9} However, there are few reported cases of idiopathic bone loss in the primary dentition.

In 1969, a case report by Butler presented two siblings inflicted with severe bone loss of the primary dentition. The areas involved were the mandibular and maxillary molars and the mandibular incisors. He suggested a progression of disease beginning with furcation involvement of the primary molars and spreading to the mesial of the first permanent molars. As the primary molars exfoliated, perhaps masking their early periodontal involvement, the erupting bicuspids would bring new alveolus with them, leaving what would appear to be a localized vertical bone defect of the first permanent molars.¹⁰

Fourel, in 1973, presented three more cases in which well children exhibited extensive vertical bone loss of the primary dentition without any apparent local or systemic etiology. Histologic examination of involved areas revealed chronic inflammation.¹¹

A report by Moffitt described a case of premature exfoliation of a primary maxillary central incisor with accompanying alveolar bone loss of the primary dentition. A medical work-up of the child revealed her to be in good health except for her dental condition. Treatment consisted of the extraction of the periodon-

tally involved teeth. An improvement in periodontal health was observed in the mixed dentition.¹²

In 1975, Pleasant presented another case of precocious alveolar bone loss in the primary dentition. Treatment consisted of full mouth extraction of the primary dentition with subsequent fabrication of pediatric dentures. Long-term follow-up revealed eruption of the permanent dentition with normal periodontal support.¹³

Treatment protocols suggested have ranged from simple extraction and space maintenance to periodontal surgery and broad-spectrum antibiotics.^{14,15} As the etiology remains unknown, and the cases so few, it is difficult to arrive at the ideal treatment and management of these cases.

Conclusion

Classically, periodontosis 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."¹⁶ Possible etiological factors suggested include specific bacterial organisms, immunologic deficiencies, polymorphonuclear leukocyte dysfunction, and genetic predisposition. It is puzzling that most authors feel that these factors should only manifest themselves in the permanent dentition.

This case report seems to contradict the classic definition of periodontosis. The clinical and radiographic findings clearly show substantial alveolar bone loss of the primary dentition without any apparent systemic or local etiology. A thorough medical work-up ruled out known diseases with oral manifestations similar to our findings; i.e., Papillon-Lefevre syndrome, hypophosphatasia, cyclic neutropenia, and acrodynia. Some might argue the bone loss observed may represent the process of exfoliation. However, the extent of furcal and crestal bone involved clearly represents a pathologic condition.

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