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Localized juvenile periodontitis in conjunction with root resorption of a retained primary second molar: case report

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Abstract

Localized juvenile periodontitis with an angular bony defect on a first permanent molar adjacent to a retained primary mandibular second molar has been observed in a young patient. The distal root of the primary second molar was resorbed completely without replacement by alveolar bone. A similar angular bony defect on the opposite molar was adjacent to a normally erupted second premolar.

Localized juvenile periodontitis (LJP) is an alveolar bone disorder affecting first permanent molars and incisors in adolescents and young adults.[1] The frequency of occurrence of this form of early periodontitis varies according to the study population. For the most part, it occurs in a relatively small percentage of the adolescent and teenage population. LJP has been reported to occur most often in young females.[2-4] Involvement of racial factors in the etiology of LJP has been questioned recently.[4] Because LJP is more prevalent in females and has a high prevalence in some families,[5] a genetic basis has been proposed as a factor in the development of the disease.[6] Approximately 60% of LJP patients have a reduced neutrophil mobility and migration rate.[7] This finding is important to the understanding of the pathogenesis of LJP since neutrophils play a major role in defense against periodontal infections.[8]

The majority of LJP investigators agree that plaque is associated closely with the etiology of the disease.[1,4,9] However, a controversy still exists as to which bacterial species are predominant in such defects.[10] No doubt the bacterial type(s) involved stimulates a relatively rapid alveolar bone breakdown compared with adult forms of the disease.[2] Most clinicians would agree that severe rapid breakdown of periodontal tissues, such as in a periodontal abscess, frequently begins with a previously deepened gingival sulcus. However, there are relatively few clinical studies that have reported deepened sulci in recently erupted permanent molars and incisors except in prepubertal periodontitis.[11] Several clinical reports have suggested other contributing factors such as the eruption of the second premolars where the crestal alveolar bone, mesial to the first permanent molar is resorbed and not repaired[12] or root fragments of primary teeth that are retained in the area.[13]

A number of other factors may predispose periodontal tissues to rapid breakdown in otherwise healthy individuals including excessive amounts of plaque accumulation and increased gingival inflammation. These factors have been reported extensively in the literature.[14]

Many investigators have been involved in the process of identifying particular bacterial types found in juvenile periodontitis lesions. One such bacterium frequently encountered is *Haemophilus actinomycetemcomitans (H.a.)* which is known to possess several potent virulence factors.[15] Other bacterial types also have been described as potential pathogens in this disease process[16] and more recently a report suggested that *H.a.* is absent more frequently in cultures of juvenile periodontitis lesions than previously thought.[10]

Case Report

A 16-year-old black female was referred to the postgraduate periodontics clinic at the Medical College of Virginia (MCV) by her general dentist for a periodontal evaluation. The patient's chief complaint was a loose tooth in the lower left posterior segment. Her medical history was noncontributory. The tooth in question was a retained primary second molar with

moderate mobility; no other tooth had similar mobility.

The primary molar was supererupted with the distal marginal ridge above the mesial marginal ridge of the first permanent molar (Fig 1). A periapical radiograph showed the primary tooth with a resorbed distal root adjacent to an angular bony defect on the mesial aspect of the permanent lower left first molar (Fig 2). What may have been a root tip was evident near the base of the bony defect (Fig 2). In addition, the second premolar was congenitally absent. Bone had not filled in beneath the resorbed root of the primary molar and the defect communicated with that on the mesial aspect of the first molar. Additional radiographs showed another angular bony defect on the mesial aspect of the lower right first molar that was similar to the one of the lower left first molar (Fig 2). However, in this instance a lower right second premolar had erupted into occlusion apparently without incident. Another bony defect smaller than the other 2 was noted on the distal aspect of the permanent maxillary left first molar (Fig 2).

A purulent exudate was expressed on palpation from the mesial aspect of both permanent lower molars and the pocket depths ranged from 6 to 11 mm. Thick plaque and supragingival calculus had accumulated around the retained primary molar while moderate plaque and slight supra- and subgingival calculus deposits had accumulated elsewhere.

Based on the findings described, LJP was diagnosed even though there was no involvement in the incisor area. A chemotaxis assay[17] on the peripheral blood neutrophils of the patient performed by the MCV Clinical Research Center for Periodontal Disease showed no deficiency in cell migration ability. No earlier radiographs were available to help ascertain the cause of the distal root resorption on the primary molar. However, the partial locking of the first permanent molar beneath the distal contour of the primary molar suggests that the root was resorbed during ectopic eruption of the permanent molar. On the other hand, the bone resorptive process of LJP also may have resorbed the distal root.

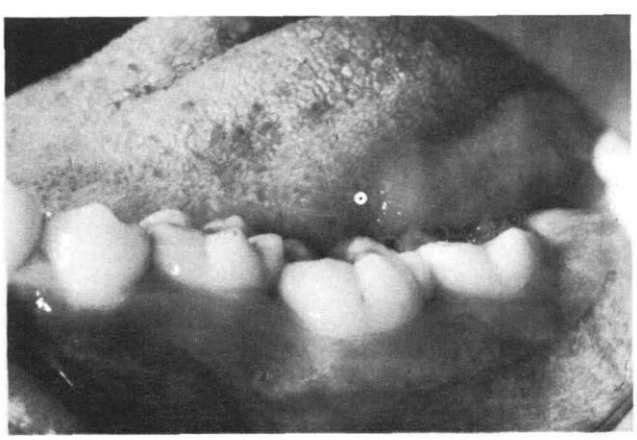

FIG 1. First permanent molar partially locked under the distal contour of the retained primary second molar.

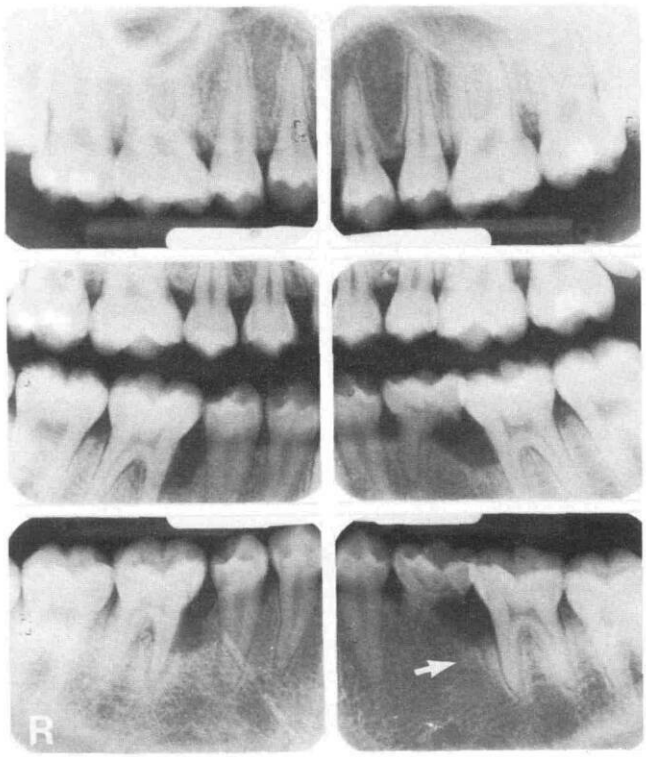

FIG 2. Angular bony defects mesial to both mandibular first molars and decreased bone density distal to the maxillary left first molar. Note the congenitally missing second premolar and the resorbed distal root of the retained primary mandibular left second molar. Also note the possible root tip at the base of the bony defect on the left (arrow) and a widened periodontal ligament space with condensing osteitis around the mesial root of the mandibular right first molar.

Treatment of the patient included extraction of the retained primary molar and scaling and root planing of all 4 quadrants under local anesthesia. Following evaluation of the initial debridement therapy, periodontal surgery was performed in the 3 quadrants with bone defects. Osseous recontouring was done only for gingival flap adaptation and was kept to a minimum. No antibiotics were prescribed during the course of treatment. Figure 3 shows the state of correction of the mandibular defects approximately 6 months posttreatment. An acid-etched bridge was made to replace the lower premolar.

Discussion

The clinical periodontal findings in the present report are consistent with a diagnosis of LJP based on specific criteria that have been described previously

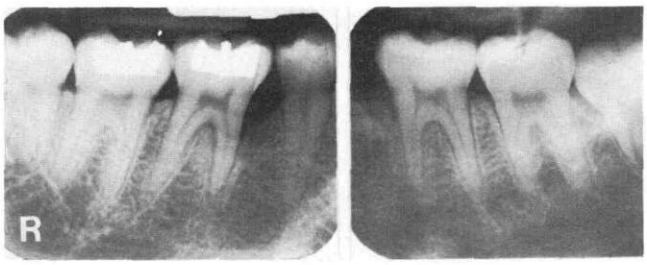

FIG 3. Mandibular right and left first molars at 7 and 5 months after periodontal surgery, respectively. Note the almost complete repair of the bony defect on the left.

by Baer[18] and Newman.[1] The uncommon feature in the present report is in the way similar appearing bony defects had developed on the mesial aspect of the lower molars even though the anatomical environment adjacent to the defects was dissimilar. In one instance, a bony defect developed without any apparent occlusal, anatomic, or iatrogenic abnormalities on or adjacent to the molar (lower right side) while, on the opposite side, a retained primary tooth having a marginal ridge discrepancy with the first molar and a resorbed distal root were adjacent to a defect. The primary molar was retained due to the congenital absence of a succedaneous tooth. McMillan[12] has attributed the development of some angular bony defects to the coronal displacement of the distal root of a primary second molar against the bone mesial to the first permanent molar. As the second premolar erupts into position the mesial bone is resorbed. In the present report, however, the congenitally missing second premolar would rule out the possibility of an aberrant eruption pattern attributing to the bone resorption on the lower left side.

The incidence of congenitally missing, mandibular second premolars has been reported to be low at ~ 4%.[19] The incidence of LJP in U.S. Air Force recruits has been estimated at 0.3%.[1] Coupling the low incidence of LJP with the relatively low incidence of congenitally missing teeth make the findings in the present case extremely rare.

The root resorption in the present case may have been the result of the ectopic eruption of the permanent lower left first molar. Since no earlier radiographs were available, the authors based this premise on the fact that the molar was locked under the distal contour of the primary molar. Usually the area occupied by the resorbed root is replaced with alveolar bone. Bone may have replaced the resorbed primary root in the present case and subsequently was resorbed during the development of the LJP lesion. On the other hand, occasionally endodontic abscesses occur in association with a partially resorbed primary molar[20] and may have accounted for bone loss on the mesial aspect of the permanent molars.

Another form of atypical root resorption in primary teeth is the "idiopathic" type which occurs occasionally during severe prepubertal periodontitis.[11,21] However, the occurrence of this type of root resorption in the present case is unlikely since the dental history did not reveal any loss of the primary dentition as a result of such early onset periodontal disease.

On the other hand, it is possible that the inflammatory process that accompanied the development of the bony defect somehow was responsible for the root resorption. Obersztyn[22] reported that inflammation frequently enhanced the resorptive process of primary tooth roots. The appearance of a possible root tip at the base of the bony defect tends to support the premise for an inflammatory etiology. In addition, retained root fragments of primary teeth have been shown to contribute to the development of periodontal bony lesions on the mesial aspect of permanent molars as a result of the development of a granulomatous lesion.[13]

Conclusion

In contrast to the rapid periodontal breakdown in LJP, periodontal disease in adults is usually a much slower process except in the case of certain, usually painful, periodontal abscesses. Many of the contributing factors to periodontal disease in adults have been identified; however, this has not been the case with juvenile forms. This report depicts the obstinate nature of LJP pathogenesis when bony defects develop regardless of the local anatomy at the breakdown site.

Initial debridement in conjunction with frequent monitoring of oral hygiene often is recommended to accomplish as much defect repair as possible before proceeding to resect or graft osseous tissue. In those patients where defects show little repair after debridement alone, antibiotic therapy may be initiated along with further debridement.

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