External resorption associated with tooth eruption

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A case is presented with severe resorption on the mesial root of the mandibular first permanent molar in a patient with Juvenile Periodontitis. The follicle of the bicuspid is seen in contact with mesial root of the molar.
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INTRODUCTION

Tooth resorption is a common sequela following injuries to or irritation of the periodontal ligament and/or tooth pulp. The course of tooth resorption involves an elaborate interaction among inflammatory cells, resorbing cells, and hard tissue structures. The key cells involved in resorption are of the classic type, which include osteoblasts and odontoclasts. Types of tooth resorption include internal resorption and external resorption. There are two types of internal resorption: root canal (internal) replacement resorption and internal inflammatory resorption. External resorption can be classified into four categories by its clinical and histological manifestations: external surface resorption, external inflammatory root resorption, replacement resorption, and ankylosis. External inflammatory root resorption can be further categorized into cervical resorption with or without a vital pulp (invasive cervical root resorption) and external apical root resorption. Other variations of resorption include combined internal and external resorption and transient apical breakdown.[1]

Root resorption is a relatively common complication of dental trauma. All pathological root resorption of dental origin is inflammatory in nature. For root resorption to occur, the protective superficial layer must be (internally or externally) damaged or changed and an inflammatory stimulator must be present.[2]

CASE

The series of radiographs, Figures 1 to 4, illustrates external root resorption occurring on the mesial root of a permanent mandibular molar. Based on these radiographs, it is reasonable to assume that the etiological factor responsible for the external resorption was the pressure exerted by the adjacent malposed bicuspid as it erupted into the oral cavity. This explanation seems reasonable since teeth and their supporting structures, in general, are able to withstand vertical forces better than horizontal ones.[3] Unfortunately, the patient never returned after the initial visit.

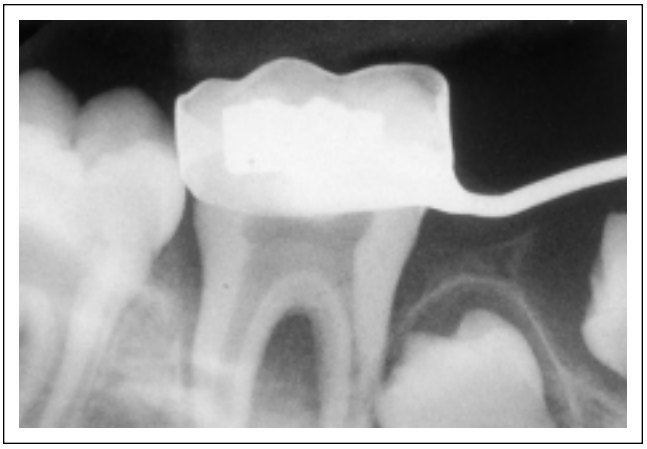

Figure 1. Follicle of mandibular 2nd bicuspid is in contact with mesial root of the molar.

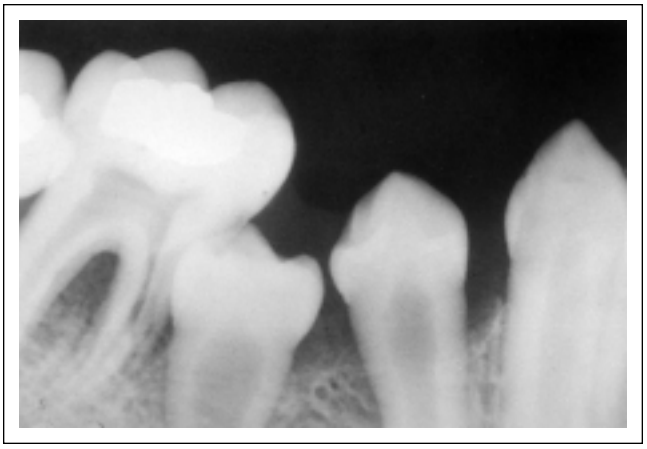

Figure 2. Bicuspid in process of erupting into occlusion.

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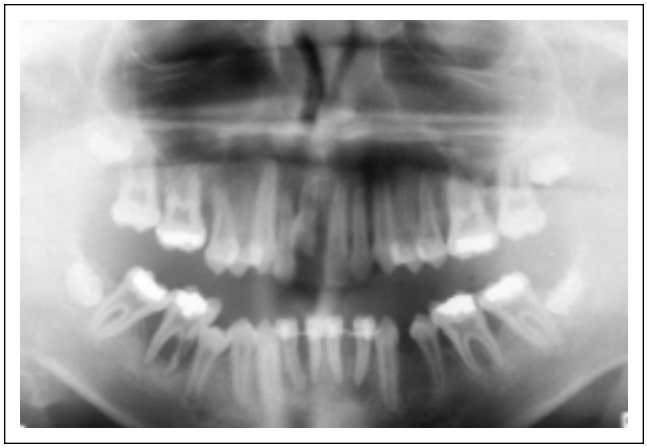

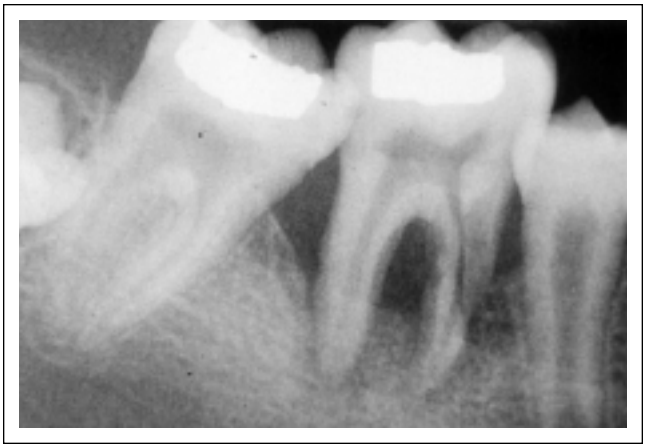

Figures 3a and 3b. Resorption on mesial aspect of mesial root of the mandibular first molar has occurred. The patient has "Juvenile Periodontitis."

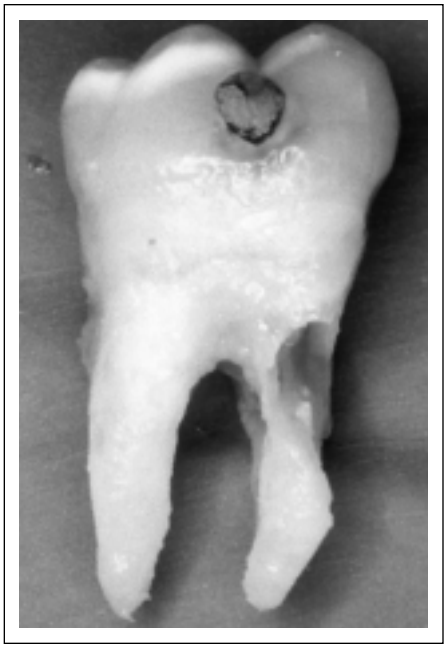

Figure 4. Note severe resorption on the mesial root of the mandibular first molar.

Although idiopathic external root resorption does occur, traumatic injuries are considered to be the primary cause of external resorption. Whether, in this patient, trauma was responsible for the unusual position of the erupting bicuspid is unknown.

It is also interesting to speculate as to whether, on a cellular level, osteoclasts were involved. There is considerable evidence that they may not have been, as osteoclasts are not essential for resorption of calcified tissues.[4]

DISCUSSION

There are several different means of studying root resorption that would be related to this case. Root resorption occurs during normal loss of the succedaneous teeth, and is sometimes found in the resorption of second molars by unerupted third molars.

Although pressure resorption of second molars is associated with impacted and/or erupting third molars, the relationship between pressure resorption and age is unclear. Yamaoka *et al*.[5] studied this relationship and found that apical root resorption may be seen long after the formation of completely impacted third molars in both genders. Thus, it is unclear whether pressure is the explanation for root resorption when studying the resorption of second molars by unerupted third molars.

The other clinically relevant model would be normal root resorption in the primary dentition. Clinically, the most apparent difference between the primary and permanent dentitions is the physiological loss of the primary tooth by root resorption. Root resorption is associated with loss of integrity of the periodontal ligament (PDL), followed by recruitment of resorptive cells that remove root structure. These findings indicate that primary dentition PDL fibroblast cells may modulate the cascade of root resorption both by regulated production of proteinases and inhibitors and by synthesis of unknown soluble factor(s) that may regulate osteoclast development.[6] It is interesting to note that this case has a pathological disturbance in the periodontal membrane caused by Juvenile Periodontitis.

Resorption of deciduous teeth is not continuous, but alternates with periods of repair or rest. Dentine surfaces in periods of rest or repair resume resorption by odontoclasts during physiological root resorption of the deciduous teeth Domon *et al*.[7] studied resorption and found that the areas with small scattered tartrate-resistant acid phosphatase activity-positive cells could be at the stage of resuming resorption, and show that the presence of preodontoclasts and odontoclasts with cytoplasmic processes extending to the covered dentine surface is a characteristic feature of the dentine surface at this stage.

When routine resorption of primary dentition is described, the role of genetics must be included. Weise *et al.*[8] studied the monocyte chemotactic protein-1 (MCP-1) gene and found that it is expressed in the dental follicle, a loose connective tissue sac that must be present for eruption to occur. The role of MCP-1 may be to recruit mononuclear cells (monocytes) to the dental follicle, where these cells, in turn, fuse to form osteoclasts to resorb alveolar bone for the formation of an eruption pathway.

Much research is being done today in the field of genetics and tooth eruption. The precise roles of genes are being defined and the roles of the proteins from these genes are being elucidated. Hopefully we will have better understanding of these clinical findings in the future.

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