Aggressive Hyperplastic Dental Follicle: Report of a Bilateral Case.

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Aggressive Hyperplastic Dental Follicle: Report of a Bilateral Case.

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Abstract

This paper reports the case of an 11-year old boy exhibiting a unique form of aggressive bilateral hyperplastic dental follicle of his unerupted maxillary canines. He was asymptomatic and unaware of this occurrence. Biopsy of the overlying tissue associated with the impacted canines revealed no significant pathological process other than focal inflammation and some hyperplasia within the dental follicle.

Introduction

Pericoronal radiolucencies are common radiographic findings observed in dental practice, especially in the orthodontic clinic. They usually represent a normal or enlarged dental follicle that requires no intervention; alternatively, they may represent a pathological entity that requires appropriate management and histopathological interpretation. A pericoronal space greater than 2.5 mm on an intraoral radiograph and greater than 3 mm on a panoramic radiograph should be regarded as suspicious (1).

Two structures form the pericoronal follicle: the reduced enamel organ and the ectomesenchyme. Both can be the origin of several types of diseases during or after odontogenesis. Hamartomas, cysts and others changes like hyperplasia have been reported (2,3).

Case Report(s)

An 11-year old white boy was referred to the private Orthodontic Clinic, Volta Redonda, Brazil, with clinical absence of the maxillary canines and no history of those teeth ever being present. The patient's medical history was noncontributory. Panoramic and periapical radiographs were obtained, which revealed expansive, well-circumscribed radiolucent lesions associated with unerupted maxillary canines. The width of the pericoronal space was 18.3 mm on the panoramic radiograph and 13.3 mm on the periapical radiograph. The radiographs also revealed severe root resorption of the central and lateral incisors (Figure 1 A, B, C and D).

The patient was admitted on Department of Oral Surgery, Dental School, University Center of Volta Redonda, Brazil for surgical management under local anesthesia and the lesion was removed performed through an intraoral approach. The central incisors surrounded by the lesion were maintained, but the lateral incisors were lost. The specimen consisted of a hard and well-demarcated capsule about 12 mm in diameter each one.

Hematoxylin and eosin-stained sections revealed a hyperplastic dental follicle similar to the tissue around the developing tooth, with proliferation of odontogenic epithelium with superficial cuboidal cells and stratification of the underlying layers, resembling typical or reduced ameloblasts, besides a larger dense connective tissue with a mononuclear inflammatory component (lymphocytes and plasma cells) (Figure 1 E and F). No tumor characteristics such as odontogenic fibroma, odontogenic myxoma or ameloblastoma were evident in the lesions.

At two months after surgery, the patient interrupted the treatment because of change of residence to another state and was thus lost to follow-up.

Discussion

There are many etiologic factors associated with this phenomenon, but the exact cause is often difficult to diagnose. These lesions may enlarge considerably if allowed to develop unchecked, and have the potential for pathological transformation (1).

Differential diagnosis should include principally with the dentigerous cyst. Recent reports have supported this conclusion, emphasizing the fact that the microscopic features of hyperplastic dental follicles and dentigerous cysts are similar, with difficult of differentiation (4,5). The dentigerous cyst is a lesion frequently associated with unerupted teeth. In the past, however, many cysts considered to be dentigerous turned out to be inflammatory paradental cysts (6) or normal follicular variations like hyperplasia erroneously diagnosed as cysts (3). Reported bilateral or multiple DC are extremely rare usually associated with developmental syndromes such as mucopolysaccharidosis, basal cell nevus syndrome and cleidocranial dysplasia (7).

Tooth eruption is a complex and tightly regulated
process that involves cells of the tooth organ and the surrounding alveolus. Mononuclear cells (osteoclast precursors) must be recruited into the dental follicle prior to the onset of eruption (8,9). In mechanical stress condition like the eruption pressure, it release substances like a arachidonic acid, prostraglandins and cytokins (Interleucin 1 and Tumor Necrose Factor). The presence of the high levels of this mediadors in the dental follicle have been describe on the literature, and play a important role in bone remodeling, bone resorption, and new bone deposition (10,11). In our case, is it possible that the eruption physical power of the permanents canines with tissue hyperplasia caused by chronic inflammation, and osteoclast recruitment associate with the substances above will be responsible for the aggressive external root resorptions of the adjacent teeth. New researches will be achieved to elucidate the etiology of this lesion.

References

Illustrations

Illustration 1

Figure 1: Panoramic radiograph (A); Periapical radiograph of the right side (B); Central periapical radiograph (C); Periapical radiograph of the left side (D); Larger dense connective tissue with a mononuclear inflammatory component lined by nonkeratinised epithelium- Microscopy low magnification (E and F); Proliferation of nonkeratinised epithelium with superficial cuboidal cells- Microscopy high magnification (G and H).
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