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Dens Evaginatus of Bilateral Permanent Mandibular Premolars with Pulpal Involvement: A Case Report

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Abstract

Dens evaginatus (DE) is a rare developmental anomaly resulting in an extra cusp in teeth. This odontogenic malformation occurs during morphodifferentiation before the mineralization of hard tissue. We report an interesting case of an 11-yearold Asian boy who presented with acute pain and tooth mobility on permanent mandibular premolars due to bilateral DE. Radiographic evaluation revealed acute periapical radiolucency indicative of an abscess. Permanent mandibular right first premolar (an immature apex tooth) was treated with apexification while permanent mandibular left second premolar (a mature apex tooth) was treated with conventional root canal treatment. The differential treatments based on the degree of root formation led to favorable outcomes of preserving the teeth. Appropriate DE diagnosis is vital to timely treatments and positive prognosis while avoiding unnecessary extractions.

Introduction

Dens evaginatus (DE) is a rare developmental anomaly that takes place before the mineralization of hard tissue. Both environment and genetic factors are involved in the development of DE, but the exact cause of DE is not yet clear [1]. According to Hegde et al. [2], genetic factors such as human EDA1(Ectodysplasin A), EDAR(Ectodysplasin A receptor), and EDARADD(Ectodysplasin A receptor associated death domain) genes may play a role in changing teeth during morphodifferentiation. Abnormal proliferation of the inner enamel epithelium into the stellate reticulum of the enamel organ causes dental abnormalities [3,4]. Pulp tissue is surrounded by enamel and dentin core. DE usually presents in the occlusal surface of posterior teeth and cingulum of anterior teeth, but rarely in labial surface of anterior teeth [5]. The term "Dens evaginatus" was first named by Oehler [6] in 1967 and is also referred to as tuberculated cusp, accessory tubercle, occlusal tuberculated premolar, Leong's premolar, evaginatus odontoma, and occlusal pearl [4]. The classification of DE varies according to researchers [5,7]. Schulge in 1987 classified DE by location of the tubercle while Lau categorized DE depending on the anatomic shape. Oehlers [6] divided DE according to the pulp contents within the tubercle and Hatab et al. used position of the teeth to classify the tubercle.

DE is most commonly observed in premolars, but can also be seen in incisors, canines, and molars [8-10]. This anomaly is five times more frequently shown in the mandible than in the maxilla [11,12] with males being more commonly affected [10]. DE can occur unilaterally or bilaterally [11], but typically occurs bilaterally and symmetrically [5]. This dental cusp is predominantly observed in people of Asians and its prevalence ranges from 0.06 to 7.7 percent, depending on race [13]. According to Levitan [5], tubercles have an average width of 2.0 mm and length of 3.5mm on the occlusal surface of posterior teeth, and an average width of 3.5mm and length of 6.0mm on the lingual surface of anterior teeth. DE is prone to wear or fracture [14]. This developmental aberration can lead to severe complications, such as loss of tooth vitality, facial infection, cellulitis, or osteomyelitis of the jaw [11]. This results in pulp involvement, with treatment depending on the shape and size of the extending pulp [11]. Many studies reported pulpal and periapical involvement from 14.1 to 4.02 percent [12, 15-17]. An early diagnosis of this anomaly provides appropriate treatment and prevents malocclusion, caries, pulpal involvement, and periodnal disease [13]. The extra cusp of DE may cause occlusal interference, causing malocclusion [7]. This case report detailed a unique case of DE where each permanent mandibular premolar was fractured within the three year time span, resulting in severe acute periapical abscess. The non-surgical root canal treatment of DE resolved acute periapical lesions.

Case Report

An 11 year-old boy with noncontributory medical history came to the clinic for dental pain on his permanent mandibular right first premolar. He had noticed severe tooth pain and mobility since the day before. Clinical examination revealed fractured DE and pinpoint exposure of caries-free premolar. The tooth was very sensitive to palpation and showed severe mobility. The other erupted premolars did not show any accessory occlusal cusps. Radiographic examination demonstrated a periapical lesion around its immature apex (Figure 1). A clinical diagnosis of the tooth with associated periapical pathogenesis revealed acute pulpal inflammation. The tooth was isolated with a rubber dam, and its working length was established by Root ZX and confirmed by periapical x-ray. We gave a prescription to the patient for amoxicillin 250mg capsule T.I.D for 7 days. From the second visit, we applied calcium hydroxide (Vitapex Calcium Hydroxide Paste with Iodoform, Neo Dental, International) to reduce inflammation and induce root formation. For 2 months, we checked the tooth regularly, and obturated with gutta-percha and AH26 by lateral condensation technique (Figure 2). After 4 months, patient returned without any symptoms. The tooth was restored with Porcelain Fused Metal Crown.



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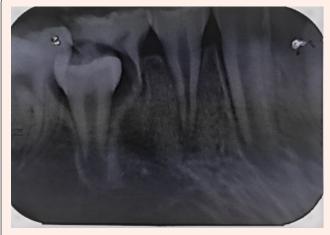


Figure 1: Periapical radiograph taken before treatment. Periapical lesion around immature apex of mandibular right first premolar due to fractured Dens evaginatus



Figure 2: Periapical radiograph taken after treatment. Applied calcium hydroxide for apexification for 2 months and obturated with gutta-percha and AH26.

After having severe symptoms on the permanent mandibular right first premolar due to DE, the patient came by again with a similar clinical feature on the permanent mandibular left second premolar two years and five months later. It was also a fracture facet of DE. Since the tooth had mature apex, root canal treatment was performed directly without having to induce root formation as with the right premolar. The patient was provided with a prescription for amoxicillin 500mg capsule T.I.D for 7 days. We took a periapical x-ray for diagnosis (Figure 3) and the tooth was filled with gutta-percha and AH26 by lateral condensation technique 5 days later (Figure 4). We restored the tooth with Porcelain Fused Metal Crown. Follow-up x-ray images (Figure 5) taken two and a half years later revealed mature apex, and the patient has since remained asymptomatic for five years (Figure 1 & 2) and two years and a half years (Figure 3 & 4), respectively.



Figure 3: Periapical radiograph taken before treatment. Periapical lesion around mature apex of mandibular left second premolar due to fracture Dens evaginatusin the same patient.



Figure 4: Periapical radiograph taken after treatment. Treated with conventional root canal treatment 5 days later.

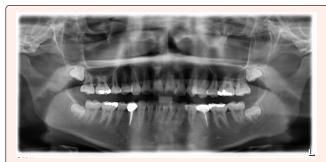


Figure 5: Panoramic radiograph for Follow-up. After five years for mandibular right first premolar and two years and a half years for mandibular left second premolar.

Discussion

An inflammatory disease of apical periodontitis is caused by continuous infection of root canal system of affected teeth. Symptoms of this infection are classified as periapical lesion, apical granuloma and cyst, and periapical osteitis [18]. Diagnosis of DE can be difficult when there are no signs and symptoms of necrotic or infected pulp. However, even if patients present signs and symptoms, the distinction between true periapical lesion and an immature apex is a difficult task. The main diagnostic methods used are pulp tests, gingival evaluation, cavity assessment, and radiographs. The pulp tests in immature teeth are known to be unreliable and periapical radiographs can only provide limited information because it is challenging to distinguish a periapical radiolucent lesion reflecting a dental follicle from general periapical pathology [11]. In our case of DE, as soon as the tubercle was erupted and occluded, the tooth was fractured and lost its vitality before the root development was complete. Because there was no apparent evidence of caries or fracture for pulpitis, the diagnosis of DE would have been challenging.

If teeth are asymptomatic or the size of tubercle is small, DE usually requires no treatment. Instead, recommended preventive measures include oral hygiene instruction, polishing, application of topical fluoride, application of fissure sealant, and flowable light-cured resin application on any exposed dentin [13]. If there is an occlusal interference, it is necessary to remove the opposing projection [13]. If pulp exposure is not severe and symptoms are mild, we can consider direct or indirect pulp capping [19], or pulpotomy with MTA using a modified Cvek technique [2]. However, as in our case, if the apex of the tooth is immature with severe pain and tooth mobility by pulpitis, treatment can be apexogenesis or apexification by using calcium hydroxide since the non-vital immature tooth becomes more susceptible to fracture due to the thin dentinal walls. The increase in fracture frequency as the stage of root development decreases may be explained by the thinner wall remaining after pulp death, which is less resistant to fracture than mature teeth [20]. If the apex is mature and closed, the tooth can be restored after conventional root canal treatment [5]. It is recommended for patients to be followed up after 3, 6, and 12 months [13]. In some cases, extraction for orthodontic treatment may be considered [7]. The knowledge of DE may be the most critical factor in accurately diagnosing and promptly treating this dental malformation. In summary, a pediatric patient with bilateral DE on mandibular premolars was successfully treated. Proper diagnosis for DE and timely treatment until complete root formation are critical. If root formation is stopped due to the occurrence of a periapical lesion, the root formation needs to be induced first by calcium hydroxide-based root canal treatment. If one DE is found, it is necessary for dentists to check the occurrence of the remaining teeth and to inquire the family history, which will assist early detection of DE.

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Page 3/3