Severe dens invaginatus in the maxillary central incisor

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Summary

Dens invaginatus is a developmental anomaly that results in an enamel-lined cavity intruding into the crown or root before the mineralization phase. The cause of dens invaginatus is still unknown and controversial. Some hypotheses suggested by authors are: local external forces, apical proliferation of ameloblast or local retardation, arresting of development and focal developmental stimuli. The anomaly occurs most frequently in the lateral incisors, although occasionally the maxillary central incisors, maxillary cuspsids, mandibular incisors and mandibular premolars may be affected. Treatment ranges from conservative procedures to non-surgical root canal treatment, surgical root canal treatment or extraction followed by intentional replantation. A 14-years-old female patient was referred for the treatment of infected maxillary left central incisor. After clinical and radiographic examination, type 3 dens invaginatus was diagnosed. The result of combined surgical and endodontic treatment was clinically satisfactory. After a 3-year follow-up period, there was no sign of inflammation and pathosis.

Key words: dens invaginatus, maxillary central incisor.

Introduction

Dens invaginatus is a developmental malformation resulting from invagination of the tooth crown or root before calcification has occurred [1, 2, 3, 4, 5, 6]. This dental dysplasia is characterized by the presence of an extra cusp that takes the form of a tubercule arising from the occlusal or lingual surface [4, 7]. The aetiology of this phenomenon is still unknown and controversial. Several causes have been proposed and these include: increased localized external forces, focal growth retardation, and focal growth stimulation in certain areas of the tooth bud [2, 4, 5, 6, 8, 9]. Synonyms for this malformation are: dens in dente, dilated composite odontome, invaginated odontome, dilated gestant odontome, tooth inclusion, dentoid in dente, dens invaginatus [3, 5, 10]. Today, the term dens invaginatus is most frequently used because the protuberance includes enamel as well as dentin and pulp tissue [7]. The condition may occur in either deciduous or permanent tooth. This anomaly occurs frequently in the lateral incisors (47%). Other teeth involved, in descending order of frequency, are the maxillary central incisors, maxillary cuspsids, mandibular incisors and mandibular premolars. Bilateral occurrence of the anomaly is not uncommon and occurs in 43% of all cases [6, 8, 10, 11, 12].

The classification of Oehler is as follows:
Type 1: Invagination involves only coronal part of the tooth.
Type 2: Invagination extends apically beyond the cemento-enamel junction, but never reaches the periapical tissues.
Type 3: Invagination extends beyond the level of cemento-enamel junction and reaches either periapical tissues or periodontal membrane via apical foramen [1, 5, 8, 9, 13].

The purpose of the present article is to report a maxillary left central incisor associated with severe dens invaginatus that was treated...
Case report

A 14-years-old girl was referred for evaluation of the fistula associated with the maxillary left central incisor. The clinical examination revealed that there were two fistulas vestibule to both the right maxillary central incisor and canine tooth. Most of the teeth, including the central incisor, had morphological anomalies such as crown dilacerations and hypoplasia (Figure 1). There was no apparent communication between the coronal dental pulp of the central incisor and the oral cavity. Radiographic examination revealed the root anomaly of the central incisor. A radiolucent lesion was detected in the mesial aspect and periapical area of the tooth (Figure 2). The periapical radiograph demonstrated that the central incisor had two root canals: a main root canal and an additional, rudimentary tract, extending through the apex at the mesial aspect of the central incisor (Figure 3). The tooth did not respond to thermal and electrical stimuli. The clinical diagnosis was established as dens invaginatus, necrotic pulp and acute apical abscess. The patient reported a vitamin D poisoning at the age of 5 months in her medical history.

Following instrumentation and irrigation, the root canal system was dressed with calcium hydroxide and re-dressed every week. The main canal was obturated with the lateral condensation technique and AH-plus sealer. The access of the vestibular rudimentary root canal could not be found during this procedure. After the root canal treatment, the fistula did not disappear. The rudimentary root canal was surgically treated. The invagination was redrogradely filled with MTA (Figures 4, 5). The patient was controlled

Figure 1. Intraoral examination revealed that most of the teeth, including the central incisor, had morphological anomalies such as crown dilacerations and hypoplasia

Figure 3. The periapical radiograph of the central incisor revealed the two root canals: one main root canal and a second additional rudimentary tract extending through the apex at the mesial side of the central incisor

Figure 2. Panoramic examination revealed root anomaly of the central incisor and radiolucent lesion in the mesial aspect and apical area of the central incisor

Figure 4. Surgical treatment was performed on the rudimentary root canal or invagination. The invagination was sealed with MTA (arrow) redrograde filling material
periodically with both clinical and radiological examination for 3 years (Figure 6). There was no sign of inflammation and pathosis and healing was uneventful at the end of this period.

Discussion

Dens invaginatus shows a broad spectrum of morphological variations and several classifications had been proposed. The most commonly used is Oehler's classification [2, 4, 10]. In the majority of cases, the anomaly is seen in its simplest, type 1 form, in which an enamel-lined cavity confined to the crown of the tooth. Severe invagination is encountered less frequently than the simple type. According to the classification of Oehler, the present case was type 3. In this type of dens invaginatus, invagination extends beyond the level of the cemento-enamel junction and reaches either the periapical tissues or periodontal membrane via a foramen. Therefore, the aetiology of the periapical pathosis in the present case was due to the infected root canal. This form was diagnosed with clinical and periapical radiographic examination.

In most cases, dens invaginatus can be detected by chance on the radiograph. Radiologically, this anomaly demonstrates a radiopaque invagination, equal in density to enamel, extending from the cingulum into the root canal. Clinically, an unusual crown morphology (dilated, peg shaped, barrel shaped) or a deep foramen caecum may be important hints, but affected teeth also may not show any clinical signs of the malformation [10, 11, 13, 14].

Management of dens invaginatus ranges from conservative procedures to non-surgical root canal treatment, surgical root canal treatment, or extraction followed by intentional replantation [1, 2, 3, 4, 7, 8, 10] depending on the size and the shape of affected tooth. Teeth with deep palatal or incisal invaginations or foramina caeca should be treated with fissure sealants. If there are no radiographic signs of pulp pathosis and no communication between the invagination and the root canal, root canal treatment or, in minor cases, even a composite or amalgam filling of the invagination will be adequate.

When pulp necrosis occurs before root-end closure, apexification procedure with calcium hydroxide may be necessary. Surgical treatment should be considered in cases of endodontic failure and in teeth that cannot be treated non-surgically because of anatomical problems or failure to gain access to all parts of the root canal system. Extraction is indicated only in teeth with severe anatomical irregularities that cannot be treated non-surgically or by apical surgery, and in supernumerary teeth [5, 8, 10].

The main problem of teeth associated with dens invaginatus is the risk of caries, pulp necrosis, pulp infection and acute apical periodontitis, because the invagination frequently allows the entry of irritants, either directly into the pulp tissues or by only a thin layer of dentin [2, 8, 11]. This necessitates endodontic procedures, such as apexification and subsequent obturation in teeth with immature apices or removal of the tooth. The prophylactic restoration of these teeth would be the preferred treatment. Generally, endodon-
tic treatment of these teeth is difficult, due to internal morphologic complexity, unusual location of pulp tissue and wide-open apex [8].

In the present case, the central incisor had acute apical periodontitis because of the necrotic pulp. After initial root canal treatment, acute inflammation symptoms were eliminated. However, calcium hydroxide did not provide satisfactory results because intracanal dressing ability of calcium hydroxide was inadequate due to the unusual form of the root and invagination extending through apical tissues. Consequently, there was no induction of hard tissue and inhibition of antibacterial effects. Therefore it was decided to perform surgical currettage of the bony lesion and seal the wide-open apex with a retrograde root filling material (MTA).

Calcium hydroxide has no sealing capability and is soluble [1, 3, 8, 15]. Therefore the use of MTA was used to seal the patient because it has been shown to be biocompatible, has good sealing properties, is soluble in water, and actively promotes hard tissue formation. When pulp-capping procedures were conducted using MTA in teeth of monkeys, all pulps showed dentin bridge formation after 5 months. The histopathologic studies revealed that the calcified bridge had probably formed much earlier, because MTA has been shown to promote hard tissue formation actively [15]. The positive result of the present case demonstrates that MTA can be an alternative to calcium hydroxide in the management of the surgical endodontic treatment of dens invaginatus. In the present case, the combined surgical-endodontic intervention was clinically satisfactory. After a 36-month follow-up period, there was no sign of inflammation or pathosis.

References


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