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Case Report

Anatomical Modification for Dens Invaginatus Treatment: A Literature Review and a Case Report

F Abduljabbar[1], M Aljehani[2], E Al Sharafi[3], A Bakhsh[3], H Abed[4], A Alghamdi[5]

Contributors:

[1]Consultant Endodontist, Dental Department, King Abdulaziz Medical City, Ministry of National Guard, Jeddah City, Kingdom of Saudi Arabia; [2]Consultant Restorative Dentist, Dental Department, King Abdulaziz Medical City, Ministry of National Guard, Jeddah City, Kingdom of Saudi Arabia; [3]Teaching Assistant, Department of Conservative and Restorative Dentistry, Endodontic Division, Faculty of Dentistry, Umm Al Qura University, Mecca City, Kingdom of Saudi Arabia; [4]Teaching Assistant, Department of Basic Sciences and Clinical Dentistry, Division of Oral Medicine, Faculty of Dentistry, Umm Al Qura University, Mecca City, Kingdom of Saudi Arabia; [5]Teaching Assistant, Department of Oral Biology, Faculty of Dentistry, King Abdulaziz University, Jeddah City, Kingdom of Saudi Arabia.

Correspondence:

Dr. Abduljabbar F. Department of Dental, King Abdulaziz Medical City (NGHA), Ministry of National Guard, Jeddah City, Kingdom of Saudi Arabia. Tel.: +(0)966-544002724. Email: fgary2001@hotmail.com

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Abstract:

Dens invaginatus (DI) is a developmental anomaly which rarely occurs. It is the result of invagination of enamel organ into the dental papilla during the soft tissue stage of tooth development. As the hard tissues are formed, the invaginated enamel produces a small tooth within the pulp chamber. It begins at the crown and sometimes extends into the root. Maxillary lateral incisors are the most affected teeth. The malformation shows a morphologic variation and results in pulp necrosis. Root canal treatment may present a severe problem because of the complex anatomy of teeth. Etiology, histology, classification, prevalence, diagnosis, clinical features, radiographic features, and treatment options are reviewed. A case of DI is reported in this article. A 10-year-old girl was referred for a persistent infection and pain from the maxillary left lateral incisor. On examination clinically, the diagnosis of DI was carried out. The canal contained an invaginated central cylindrical mass of hard tissue which limited the access to the canal system and challenged the cleaning and sealing of the canal spaces. A modification of the internal anatomy of the canal system was achieved under the operating microscope. The conventional chemo-mechanical preparation was done, and the root canal was obturated with mineral trioxide aggregate. Two-year follow-up showed the regression of the lesion.

Key Words: Anatomical modification, dens invaganitus, malformation, morphologic variation

Introduction

Etiology

In general, there is no agreement on the exact cause of dens invaginatus (DI). Several theories have been suggested to clarify the etiology of this anomaly. These include: Pressure resulting from the growth of the dental arch leads to bending of the enamel organ, localized failure of growth of internal enamel epithelium.[1] In contrast to Kronfeld[2] suggested that the enfolding is the result of the aggressive and rapid growth of some internal enamel epithelial cells invading the dental papilla, a fusion of two tooth germs (the twin theory).[2] Infection was also suggested by Fischer and Sprawson.[3,4] Gustafson and Sundberg considered that trauma is a causative factor.[5] Genetic factors were included in the list of possible causes.[6-10] During tooth development, an enamel-lined channel is formed due to distortion of enamel organ and a successive protrusion of part of it. This channel ends at the cingulum or sometimes at the incisal tip.[11]

Histology

Histological studies show that the dentin below invagination could be intact without irregularities,[12,13] hypomineralized or irregularly shortened. It may also contain strains of vital tissue or even fine canals that communicate to the dental pulp.

Enamel lining the invagination was shown to be irregularly structured.[14,15] Studies reported differences in structure between the external and internal enamel. It was reported that the internal enamel showed atypical and more complex rod shapes, but its surface presented the typical honeycomb pattern without perikymata. At the base of the folding, Benyon reported hypomineralized enamel. In contrast, Morfis found that enamel at the base of the invagination has up to 8 times more phosphate and calcium in comparison to the outer enamel,[14,16] although magnesium was missing completely.

Prevalence

DI is a common dental anomaly; it is usually overlooked due to the insignificant clinical signs.[11,12,14,17,18] The range of occurrence is between 0.04% and 26%. The most commonly affected teeth are maxillary lateral incisors (47%), and it is frequently bilateral (43%). Most common types include Type I (94%), Type III (33%), and Type II (4%), respectively. It rarely occurs in the mandible and in primary dentition. Data suggest a more male gender predilection.[19,20]

In deciduous teeth, very rare cases have been reported. One of which has shown incisal invagination that is opposite to the usual finding of the permanent anterior teeth that is usually found palatally. Dental literature reported other coincident dental anomalies, malformations or even medical syndromes.

Classification

The first attempt to classify DI was proposed by Hallett who classified the invagination into four types based on clinical and radiographic criteria.[21] Other classification systems emerged later, for instance Schulze and Brand (1974) suggested 12-type classification system based on the clinical and radiographic appearance of the tooth.[22] It includes invaginations at the top of the crown or at the incisal edge with the dysmorphic root configuration (Figure 1).

However, the most common used is the one described by Oehlers possibly because of its convenience.[11] He classified DI into three categories according to the radiographic appearance determining the invagination depth and communication to periodontal ligament and periapical tissue from crown into root (Figure 2).[11,23] The types include: Type I: A minor enamel-lined invagination, which is confined to the crown of the tooth but not extending beyond the cementoenamel junction; Type II: Enamel-lined invagination extending beyond the cementoenamel junction invading the pulp chamber but ending as a blind sac.[11,23] It may or may not communicate with the pulp. Type III: The invagination extends through the root. Usually, there is no communication with the pulp. It communicates either laterally to the periodontal area through a (pseudo-foramen) (Type III A) orapically, showing a second foramen (Type III B) the invagination lining may be completely by enamel although cementum was found frequently.[11,23]

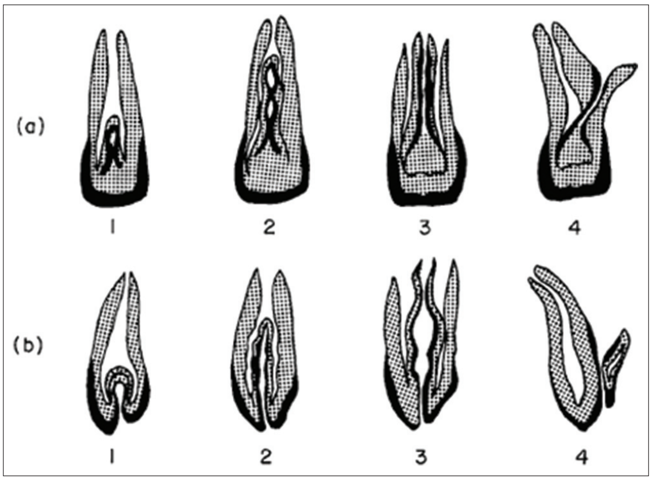

Figure 1: Classification of dens invaginatus by Schulze and Brand.[22]

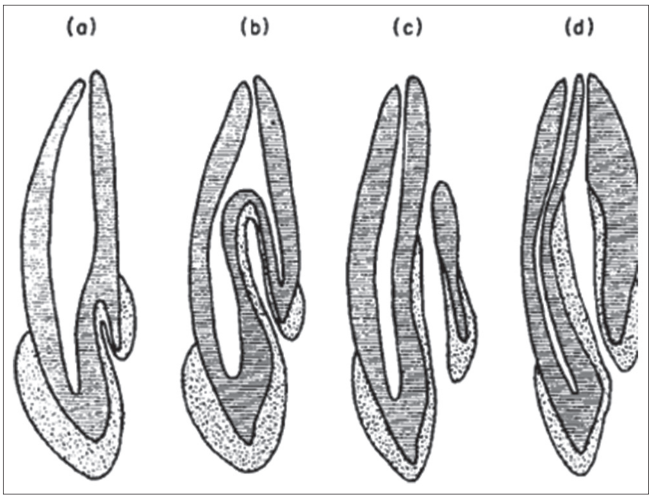

Figure 2: Classification of invaginated teeth by Oehlers.[11,23]

Diagnosis

Mostly, DI is detected by chance on a radiograph. Although, some clinical morphological changes serve as useful hints in the detection of the lesion, other teeth may not show any clinical signs of malformations. If a patient's tooth is affected, the contralateral tooth should be thoroughly investigated.[11,23] Conventional radiographic imaging does not provide enough details due to its two-dimensional nature. Spiral computed tomography is a recent three-dimensional (3D) radiographic technology introduced by Reddy *et al*., Patel *et al.,* and Sponchiado *et al*., which provides more structural details about the malformation and the root anatomy.[24,25]

Diagnosis of the invagination can be difficult as its entrance may be unremarkable or even similar to normal fissures anatomy.[26] Magnification and application of methylene blue dye can aid in the diagnosis. The use of radiopaque marker can help to define the extent and shape of the invagination.[27] Reported consequences of undiagnosed or untreated cases are: Abscess formation, displacement of teeth, internal resorption,[28,29] retention of neighboring teeth, displacement of teeth, cysts, facial cellulitis recently diagnosed,[30,31] incisal notching in association with a labial groove, and exaggerated palatal cingulum or "talon cusp."

Clinical features

The area of invagination is a site of microbial entry to the pulp since it is thinly or incompletely lined by enamel and may be connected to the pulpal tissue via channels. Pulpal necrosis usually occurs in early age; after few years of eruption or even before closure of the root end. Some helpful diagnostic hints in crown morphology include that the crown is dilated or peg-shaped, barrel-shaped, has deep foramen cecum, incisal notching or increased labiolingual and mesiodistal diameter. It might also have a conical morphology or an enlarged palatal cingulum or cusp.

Radiographic features

The radiographic image used in the diagnosis of DI is affected by several factors including the practitioner's skills and knowledge of radiographic reading, the awareness of the 3D nature of the anomaly and the quality with the angulation of the radiographic film.[32]

The radiographic image may reveal the invagination as: Varying from a narrow fissure to a tear-drop shape directed to pulpal body, or, enamel-lined fissure that is completely isolated from the pulp and is connected to the periodontal space. This appearance is known as "pseudo-canal."

The pulp shows different changes in its morphology. It is more difficult to be traced inside the root canal with changes in its outline as it approaches the invagination area.[33]

Radiographic structure of DI according to the type of invagination can be classified as Types I, II, and III. Type I and II usually begins at the coronal portion as a narrow fissure that turns into a uniglobular mass. Depending on the site where this mass ends, the classification type is different; if it ends coronally; it will be known as Type I, and if it ends radicularly, it will be known as Type II. The invagination may appear as a loop-like, pear-shaped, a slightly radiolucent body or it might be as severe as it shows the appearance of a tooth within a tooth. Type III is classified as Type III A where the root canal at this type may appear irregular. The invagination close to the root canal is deep and is considered as an ideal site for bacterial attack. Usually, infection at this site results in "peri-invagination periodontitis" which leads to the blunderbuss appearance formation. Due to the intimate communication between the root canal and the invagination, if invagination infection occurs, the pulp becomes necrotic. Type III B is more difficult to diagnose because it usually appears superimposed over the root canal. Radiographically, it presents as an immature apex or, mostly, as radiolucent periapical lesion.[26]

Treatment

Until the 1970's, extraction was the preferred method for treating DI cases[34], and it is still the treatment of choice in severe invaginations.[35-41] Root canal treatment (RCT) for the invagination part was first introduced as a method of treatment by Grossman[42] and Creaven[43] while Tagger[44] and Hovland and Block[45] were the first to present cases treated with RCT. In some cases, minimal invagination can be sealed using different preventive procedures.[10,18,32,36,37,46,47] In cases of incomplete root end closure, calcium hydroxide apexification,[28,48-51] mineral trioxide aggregate (MTA)[52,53] for root end closure or even gutta-percha can be used during treatment.[54]

However if the invagination has a distinct apical foramen, RCT is the treatment of choice.[32,42,43,55-57] In cases of complex morphology of the root canal of DI cases, the same technique is to be used.[58] For the obturation of such cases, warm gutta-percha with vertical condensation or thermoplastic filling materials can be used.[18,28,59]

Type I treatment

When the invagination is minimal and restricted to the crown, prophylactic treatment should be done. Once moisture control can be achieved, acid-etched sealant or flowable composite can be used to seal the invagination entrance. Tooth monitoring should be maintained to detect any possible pulpal disease or restoration defects. If pulpal disease was evident, RCT should be initiated.

In cases of limited inflammation with immature apex, vital pulpotomy is to be performed. In more severe cases, conventional methods should be considered. The only difference is to verify that proper cleaning of the invagination area is complete. This can be attained through extending the access cavity to include the lesion using the gates Glidden burs or ultrasonic tips with a brushing action.

Type II treatment

As the invagination is deeper, it would be more difficult to access and seal the invagination. Therefore, a coronal entrance should be gained to aid the visualization and the proper treatment. This can be achieved using a carbide bur and magnification to inspect the invagination. If caries was found, it can be removed using long-shank round bur or ultrasonic tips. The lumen should be cleaned and irrigated with chlorhexidine or 1% sodium hypochlorite before sealing the invagination. Due to the high possibility of pulpal contamination, it would be reasonable to consider all cases as if the pulp was involved and managed in the same manner as direct pulp exposure cases. After moisture control, MTA is to be compacted into the lumen. A coronal seal can be achieved using composite restoration.

Type III treatment

In this type, if the invagination is not affected neither plaque is retained, and then simple prophylactic measures, similar to those of Type I, could be applied. However, regular clinical and radiographical follow-up should be maintained. In cases of "peri-invagination periodontitis" with healthy pulps, the treatment should be directed toward preserving the healthy pulp status. In other words, the peri-invagination periodontitis should be treated separately without involving the root canal. Clinically, this can be only made possible in Type III A cases.

Proper magnification using the microscope and the placement of methylene blue dye can aid in lesion identification. A gutta-percha point or an endodontic file can be used as radiographic markers to determine the lesion orientation. Ultrasonic alloy tips are more useful in the treatment process than endodontic stainless steel or nickel-titanium instruments.

If the pulp was necrotic, the invagination and the root canal are to be combined in the treatment due to the very close proximity of each other. Ultrasonic instruments or long-shanked round burs are preferably used working from the root canal side to the invagination. Care should be taken when preparing the apical portion to avoid its destruction, and it is preferred to keep the invagination and the root canal separate. After preparation, obturation with gutta-percha points is acceptable. Due to the funnel anatomy of apical portion, MTA is the material of choice to be used. Then the remainder of the root canal can be backfilled with gutta-percha. The coronal portion is to be sealed using composite restoration.[1]

Case Report

A 10-year-old medically free girl was referred by her dentist suffering from pain and a persistent infection from the maxillary left lateral incisor. After clinical examination, an intraoral sinus tract buccal to the tooth was present (Figure 3). The tooth was non-mobile but tender to percussion. Periapical radiograph revealed a large radiolucency apical to maxillary left lateral incisor and confirmed the diagnosis of DI and was classified as Oehler's Type II due to invagination extending through the root canal with no communication with the periodontal tissue (Figure 4). The main canal contained a central cylindrical mass of hard tissue. Owing to limit the access to the canal system and the cleaning and sealing of canal spaces, a modification of the internal anatomy of the canal system was achieved under the operating microscope (Figure 5). The hard tissue core was removed using ultrasonic instrumentation and pulled out with a hand endodontic file (Figure 6). Working length was determined (Figure 7); conventional chemical and mechanical preparation with sodium hypochlorite combined with intra-canal calcium hydroxide were done, and the root canal was obturated with MTA. The coronal third was sealed with composite restoration (Figure 8). After 2 years of the treatment, the tooth was re-examined (Figure 9). The patient remained asymptomatic, and the radiographic evidence revealed apical bone healing (Figure 10).

Discussion

Many articles are published regarding treatment of DI with a low number of evidences. In general, DI occurrence ranges between 0.04% and 26% and to adequately treat this condition endodontically, knowledge of the different types and approaches to treatment are essential. These kinds of cases have often been treated with non-surgical RCT, RCT

Figure 3: Frontal view (pre-operative).

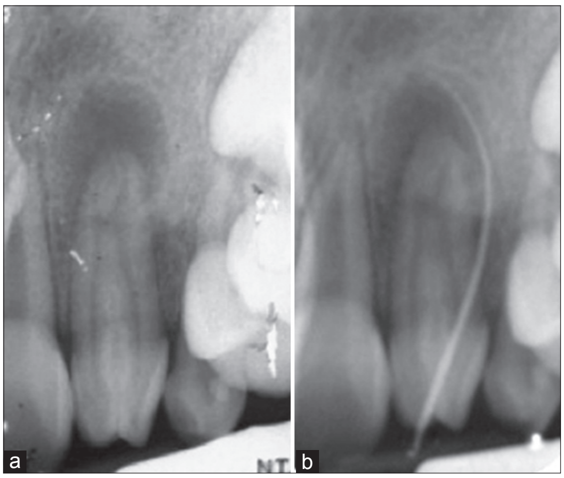

Figure 4: (a and b) Pre-operative radiographs.

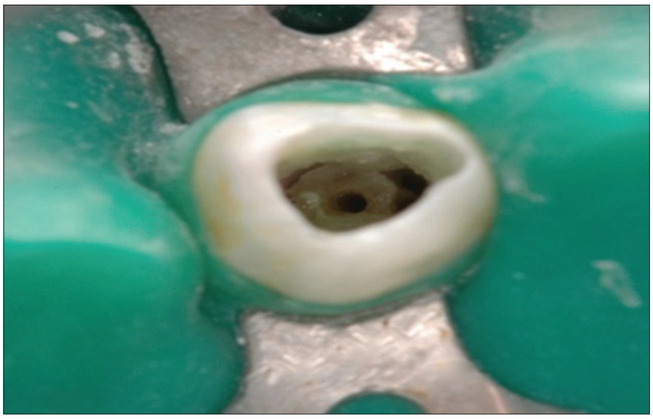

Figure 5: The access opening.

Figure 6: Hard tissue core removed from the tooth canal.

Figure 7: Working length determination.

Figure 8: Post-operative radiograph.

Figure 9: Lateral view at recall visit.

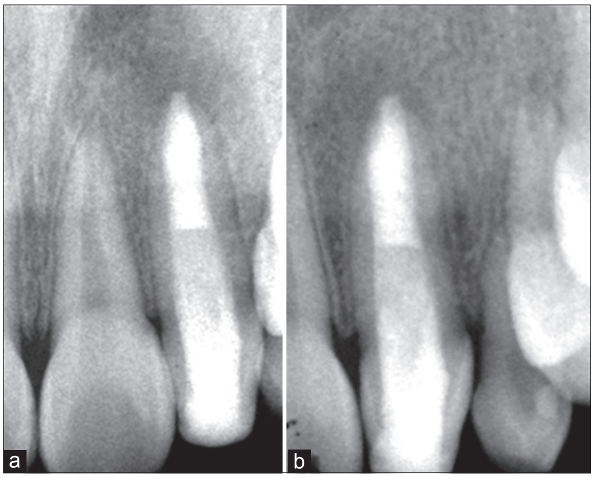

Figure 10: (a) Review at 6 months; (b) review at 24 months.

and surgery and even extraction. The successful treatment of DI is based on mainly accessibility to and disinfection of the root canal system. The present case demonstrates that RCT of DI can be successful in complicated cases, and it was achieved by the removal of the dens or hard tissue core from the canal system which provided the ability to disinfect and seal the root canal. RCT of invaginated teeth is frequently associated with problems arising from complex variations of root canal morphology or from difficult access to regular and invaginated canals. Treatment of the root canal combined with the invagination as one entity proved to show satisfactory results using ultrasonic instrumentation but destructs more tooth structure.[27,60] Ultrasonic cleaning with canal irrigation has been suggested to disinfect cases with large and irregular root volume.[61] Proper chemo-mechanical preparation of the root canal combined with MTA application resulted in regression of the periapical lesion noted at the recall visit.

Conclusion

Modificaton of the internal anatomy of the canal system may be considered by clinicians for the treatment of DI treatment, to gain better access for instrumentation, disinfection, and sealing of the root canal. Further researches have to be done to establish technical standards for treatment of complex anatomical cases.

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