Dens Invaginatus, from Diagnosis to Treatment: Case Report

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ABSTRACT

Dens invaginatus is a developmental malformation in which enamel folds into dentine. This varying anatomical feature sometimes complex, can present a real treatment challenge. Therefore, early diagnosis is crucial, as preventive treatment of dental cavities can prevent pulp degeneration and pulp necrosis. This article through a case report describes the different classifications of DI, and reviews the literature to make a correct diagnosis and properly manage this type of anomalies, which can constitute a true clinical challenge because of its complex anatomy.

INTRODUCTION

Dens invaginatus (DI) is a developmental anomaly invagination of the enamel organ into the dental papilla, which cause an invagination of the crown and/or the root surface before mineralization occurs. The range of DI prevalence is 0.3% to 10%. DI is most frequently observed in the maxillary lateral and central incisors but uncommon in canines, premolars, and molars. Furthermore, bilateral DI incidence is not unusual. In 43% of cases, DI can be bilateral. Therefore, if a dens is identified, clinicians should always check the contralateral tooth. DI is often diagnosed incidentally during radiographic examinations unless the patient experiences pain and/or swelling in the affected tooth. Although some teeth with dens invaginatus may appear normal, most cases exhibit atypical crown shapes, such as conical, peg-shaped, barrel-shaped, or dilated forms, or an exaggerated bifid cingulum. Therefore, it is recommended that clinicians perform radiographic examinations on these teeth.

Oehler’s classification (1957) proposes the existence of three types of invaginations and knowing the different invagination’s types will be helpful when deciding appropriate treatment for each lesion. Type I cases, describe a partial invagination that is limited to the affected tooth’s crown. Partial invagination is seen in type II instances, when it goes past the cementoenamel junction into the root. These lesions don’t communicate with the periodontal ligament (PDL), staying inside the root’s architecture. In instances of type III, total invagination reaches through the root and interacts with the PDL.

Note that sometimes dens invaginatus can be combined with other anatomical or structural abnormalities such as gemination, taurodontism, fusion or even dentinogenesis imperfecta.

CLINICAL CASE

A 18-year-old female patient visited our Department of Conservative Dentistry and endodontics, complaining of palatal abscess, inside of the upper left incisors.
The patient had no antecedent of trauma. After clinical examination and applying the cold sensitivity, only the left lateral incisor didn’t respond to coldness accompanied with severe pain to axial percussion. The Periapical X-ray of tooth #22 showed an important periapical lesion with Oehlers’ type II DI; what was confirmed by CBCT X-ray. Tooth #22 was diagnosed with pulp necrosis, chronic apical abscess and Oehlers' type II DI. The intussusception was unique without notion of bilaterality.

Root canal treatment of tooth #22 was planned, with a follow-up to determine if surgery will be necessary later. Antibiotic medication was administered to the patient during this first visit.

A week later, we saw the patient again, this time we created an access cavity after placement of the rubber dam. We permeabilized with 10 files and processing to preparation of both of the canal and the lateral invagination, while respecting permanent irrigation with a 2.5% sodium hypochlorite solution. After disinfection, the session was completed with intracanal medication with calcium hydroxide, once again of the canal and the intussusception.

The next session (15 days later), oozing from the canal was still present, we therefore decided to renew our intracanal medication with calcium hydroxide and see the patient again in 15 days.

This time, the canals were very dry which justifies the decision to block the main canal and the intussusception, thus the material of choice belonged to the calcium silicate family.
After this incident, clinical and radiological follow up were established, and no complaints were reported by the patient after, associated to periapical healing.

**Figure 3**: Opening of the access cavity which revealed a distal main canal in addition to the invaginated canal in the mesial position.

**Figure 4**: Filling with calcium silicate bioceramic cement and fusion through an accessory endodontic canal.

**Figure 5**: 1* X-ray control after 3 months / 2* X-ray control after 9 months / 3* X-ray control after 14 months / 4* X-ray control after 20 months / 5* X-ray control after 26 months.
DISCUSSION

While preparing the canal, we encountered certain difficulties, starting with the determination of the canal entrance of the invaginated canal which was eccentric in the distal position. The preparation of this canal itself was delicate given its very narrow diameter. Enough time was sacrificed for permeabilization thanks to the k10 file and as well as abundant irrigation before being able to prepare the canal to the working length with a rotary system of 4% conicity. A final difficulty revealed itself during obturation this time, which was related to the presence of a lateral canal, which caused diffusion of the bioceramic beyond the apical foramen which was hard to control.

Since then, regular follow-up has been respected where the different x-rays showing a positive evolution in favor of apical healing and clinical silence.

In the end, the case was entirely treated endodontically without recourse to surgery.

ETIOLOGY:

The exact cause of dens invaginatus remains unknown, but several theories have been proposed:

External Factors: Atkinson (1943) suggested that dental invagination results from external forces affecting the dental germ during development, such as trauma or infections. (14)

Developmental Anomalies:
- Twin Theory: Proposes that the condition arises from the fusion of two tooth germs. (14)
- Delay Theory: Suggests that the internal enamel epithelium fails to grow properly, while the cells of the external enamel epithelium continue to proliferate. (14)
- Active Proliferation Theory: Proposes that invagination is due to a rapid and aggressive proliferation of a portion of the internal enamel epithelium invading the dental papilla. (14)
- Vascular Theory: Insufficient vascularization of the dental papilla may reduce ameloblast activity, leading to the formation of gaps in the internal enamel epithelium. (14)
- Genetic Factors: The genetic origin of the invagination is plausible due to its high prevalence in individuals from the same family and its potential association with other malformations. (14)

An underlying invagination should be ruled out with an adjunctive radiographic examination if these teeth show any clinical signs of a dens.

DI is distinguished radiographically by a radiopaque invagination that extends into the root canal from the cingulum. The invagination can be pear-shaped, loop-shaped, or slightly radiolucent, giving it the appearance of a "tooth within a tooth". (6)

The use of 3D imaging by CBCT is highly recommended in such cases, it can assist the clinician in determining which class of dent is affecting the tooth and enable them to determine whether any planned endodontic treatment is feasible.

Teeth that are impacted may develop caries and peri-radicular pathology if left untreated and patients may exhibit signs of apical periodontitis or irreversible pulpitis. When there is no apparent history of trauma or caries, the possibility of DI should be taken into account, and lastly, patients who have been diagnosed with DI in one tooth should be highly suspected of DI in the contralateral tooth. Depending on the depth of invagination, various treatments have been proposed for DI. (3,4,6)
Treatment of Oehler's Class I lesions:
Invasions of Class I may be very slight. The tooth’s palatal surface should be sealed as soon as a lesion is identified, either clinically or through radiography, in order to stop caries from developing. On the contrary, it is necessary to get root canal therapy if pulpal necrosis is visible. Generally, teeth with Class I lesions typically do not have severely malformed root canals, the access cavity may contain the entire invagination. (3)

Treatment of Oehler's Class II lesions:
Comparatively speaking, CDI type II invagination is more severe than CDI type I. It breaks into the pulp chamber and might get in contact with the pulp. Preventive fillings should also be the first option, just like for CDI type I, if the affected teeth do not exhibit caries. If affected teeth have caries at the entrance of the DI, but with vital pulp, treatment should be confined to the invagination, the invagination can be dressed with a material that will promote hard tissue formation such as mineral trioxide aggregate (MTA) or Biodentine. Finally, root canal therapy (RCT) is recommended if pulpal necrosis has been brought on by the invagination and subsequent caries. (3,4)

Treatment of Oehler's Class III lesions:
Class III lesions require more complicated management. The tooth should only be sealed if it is asymptomatic and shows no indications of pulpal disease. Because Class III invaginations are so close to the root canal system, pulpal involvement is likely to occur from prophylactic access and debridement of the invagination. As such, a more cautious strategy is advised. Depending on how the invagination and root canal system interact, if both the pulp and the invagination are infected, the invagination may need to be treated separately or in conjunction with the root canal system.

Because the pulp/dentine complex and invagination are frequently histologically related, it makes sense to treat both systems with endodontic therapy, which will enable the patient to receive full tooth treatment.

Next, the tooth needs to be maintained under long-term review. In the event that the patient's symptoms do not improve, microsurgery or extraction of the dental unit may be necessary. (3,4)

Early diagnosis enables prophylactic treatment, which, particularly in types I and II of Oehler's cases, consists of sealing the crown opening with restorative material to prevent decay of the invagination and subsequent involvement of the main root pulp. (2)

As demonstrated by Lucas et al. (2003), who controlled 80 teeth (13 type I and 67 type II) clinically and radiographically for more than six months after closing the opening on the surface of the dental element early, regular follow-up is necessary because the prognosis is not entirely favorable. Nine (11.3%) treatments failed, all of which involved type II invagination. (2)

In other situations, some authors indicate only endodontic treatment of the invagination, leaving the pulp of the main canal intact (2)

Due to its deep invagination, which nearly reaches the apical region of the dental element and hinders appropriate cleaning and modeling of the root canal system, dens invaginatus type II and III treatment is complicated. (2)

Certain authors recommend separating the invaginated area, which nearly reaches the apical region of the dental element and hinders appropriate cleaning and modeling of the root canal system, dens invaginatus type II and III treatment is complicated. (2)

Certain authors recommend separating the invaginated area for the invagination to facilitate optimal cleaning and filling using the root canal filler. This can occur when the invagination is bound only by the cervical portion and is surrounded by pulp tissue rather than adhering to the entire extension of the root walls. And according to many authors, a surgical microscope is very helpful in the treatment of dens invaginatus, both for proper access and invagination removal. (2) Certain other authors, including Ozbas et al., observed that removing root canal invagination can make thin root walls more fragile. Clinical interventions that can reinforce the remaining structure are recommended in these circumstances. (2)

CONCLUSION

Teeth with lesions of dens invaginatus are more likely to get pulpal diseases. Because the internal anatomy of teeth affected by these lesions is irregular and unpredictable, the resulting endodontic treatment is complicated. Even though the management of these anomalies has been improved by CBCT, contemporary endodontics, and surgical endodontics, early detection and prophylactic treatment of these lesions is essential to avoid the development of pulpal pathology and requiring complex and specialized endodontic treatment.

BIBLIOGRAPHY


