

Dens invaginatus: diagnosis and management strategies

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In brief

Provides an overview of the aetiology and classification of dens invaginatus lesions.

Discusses the clinical features of this anomaly which can lead to early diagnosis.

Provides an in depth discussion on the treatment options and management strategies for dens invaginatus.

Reviews the clinical challenges of treating such lesions.

Dens invaginatus is a developmental malformation, in which there is an infolding of enamel into dentine. These infolds represent stagnation sites for bacteria and can predispose to dental caries. The carious infection can spread via enamel and dentine to contaminate the pulp and cause soft tissue necrosis. The altered and sometimes complex anatomy of affected teeth can make endodontic management challenging. Early diagnosis is therefore essential as prophylactic treatment of the dens can prevent degeneration and pulpal necrosis. The aim of this article is to review the aetiology, classification, diagnosis and management of teeth affected with dens invaginatus. Emphasis will be placed on describing the clinical features of this anomaly. Treatment options, management strategies and the challenges faced in managing this condition will be discussed.

Introduction

Dens invaginatus (DI) is a developmental dental anomaly where there is an invagination of the enamel organ into the dental papilla, before calcification is complete.¹ The invagination begins in the crown and may extend into the root (Fig. 1).

As a result, an infolding of the enamel into dentine occurs which creates a pocket of organic material underneath the enamel surface. These lesions are clinically relevant as bacteria from the oral cavity can contaminate and propagate within these malformations, leading to the development of early caries and consequently pulp death.¹

Although these lesions usually form under the palatal pit or cusp tip, they can be extensive and grossly distort the anatomy of the root canal system (Fig. 1).²

The altered tooth morphology and concurrent pulpal necrosis (in an immature tooth) can disturb root formation. Therefore teeth with DI may present with wide open or 'blunderbuss' open apices, further complicating endodontic

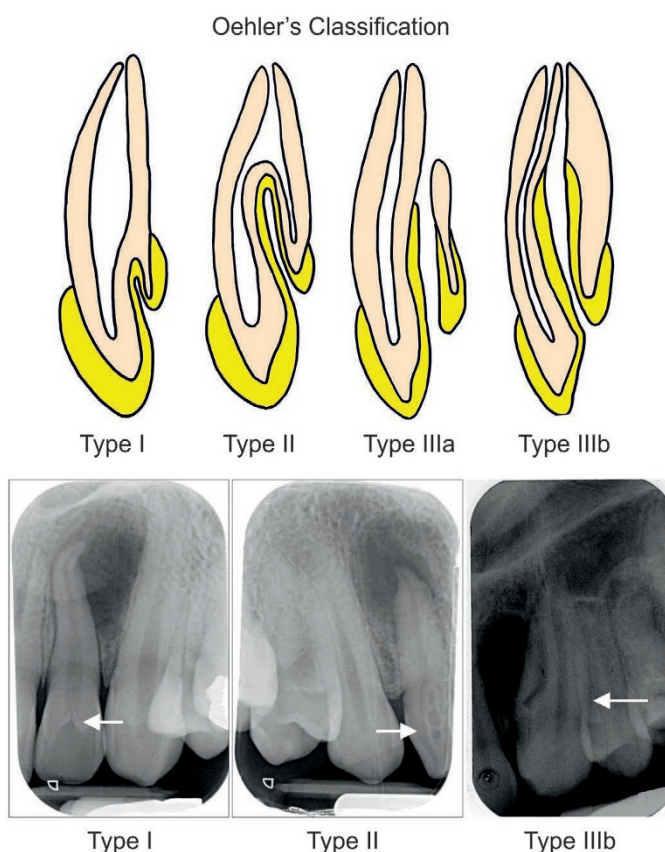


Fig. 1 a) Oehlers Classification of dens invaginatus lesions; Type I: the invagination is confined to within the crown of the tooth and does not extend beyond the level of the amelo-cemental junction. Type II: the invagination extends into the pulp chamber but remains within the root canal with no communication with the periodontal ligament. Type IIIa: the invagination extends through the root and communicates laterally with the periodontal ligament space through a pseudo-foramen. Type IIIb: the invagination extends through the root and communicates with the periodontal ligament at the apical foramen; b) Intraoral radiograph illustrating a Type I Oehlers dens invaginatus on a lateral incisor. Notice the large periapical radiolucency; c) Intraoral radiograph of a lateral incisor with a Type II lesion; d) Intraoral radiograph of a canine with an Oehlers Type IIIa dens lesion

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management. Therefore early diagnosis and prophylactic treatment of these lesions is important if pulp vitality is to be maintained.

Dens invaginatus lesions were first described in 1794 after being discovered in the tooth of a whale by Ploquet.² A number of different terms have been used to describe the condition including dens in dente, dentoid in dente, invaginated odontome, dilated gestant odontome, dilated composite odontome and tooth inclusion.¹

Aetiology

A number of theories regarding the aetiology of DI have been proposed. These include infection,³ trauma,⁴ growth pressure on the dental arches during odontogenesis causing infolding of the enamel⁵ and rapid proliferation of the internal enamel epithelium into the underlying dental papilla.⁶ However, the definitive cause of the lesions remains unclear.¹

Authors have looked at genetic factors that could be responsible for dens invaginatus. During tooth development, growth molecules have been shown to regulate the folding and development of the enamel organ.⁷ If these genetically determined

growth factors are absent or altered, the morphology of developing teeth can be disturbed.⁸⁻¹² This theory is supported by the fact that DI can have a familial tendency, with the parents and siblings of DI patients also being affected by similar lesions,⁸ and that these lesions are associated with other genetically determined anomalies.¹³⁻¹⁵

Classification

There have been numerous attempts to classify DI.¹⁶⁻²⁰ The most widely used classification system was produced by Oehler¹⁷ and this system separates these lesions into three simple categories. An understanding of these categories will help when discussing the appropriate treatment options for each lesion (Fig. 1).

- Class I: This is a partial invagination that is confined to the crown of the tooth. These lesions involve dentine and enamel but do not extend past the cement-enamel junction (CEJ) or involve the pulp.
- Class II: This partial invagination extends beyond the crown of the tooth and into the root, beyond the CEJ. These lesions may or may not involve pulp, but remain within

the anatomy of the root. There is no communication with the periodontal ligament.

- Class IIIa: This complete invagination extends through the root. It communicates with the periodontal ligament through a second foramen on the lateral aspect of the tooth. There is usually no involvement of the pulp itself, but it causes a significant anatomical malformation.
- Class IIIb: This complete invagination extends through the root, and communicates with the periodontal ligament at the apical foramen. Again, often there is no direct involvement of the pulpal anatomy but the lesion causes significant disruption to the dental anatomy.

Prevalence

Dens invaginatus lesions have been reported to affect 0.3% to 10% of the population.² These figures may initially seem small, however, they are significant as they suggest that dens lesions occur more commonly than other developmental dental anomalies, including peg-shaped lateral incisors (0.8%), germination (0.3%) and taurodontism (0.3%).¹

The teeth most commonly affected by DI are lateral incisors²¹ followed by maxillary central incisors, and more rarely premolar and canine teeth. Although rare, case reports have detailed DI lesions in mandibular teeth,^{22,23} deciduous teeth,²⁴⁻²⁶ supernumerary teeth^{27,28} and multiple lesions found in a single tooth.^{22,27,29} In 43% of cases, DI can be bilateral.⁸ Therefore, if a dens is identified, clinicians should always check the contralateral tooth.

Clinical examination

DI are often asymptomatic and the crowns of affected teeth may show very little external deformity. Extensive malformations are more likely to result in abnormal crown anatomy. Patients may complain of an abnormally shaped tooth which may be wider mesially/distally; wider labially/lingually,^{30,31} associated with an exaggerated talon cusp³² or even conical in shape.^{32,33}

If undiagnosed, affected teeth are prone to developing caries and peri-radiolar pathology. Clinicians should be mindful that lateral incisors are the most commonly affected teeth. If these teeth exhibit any clinical features of a dens (such as a pronounced talon cusp or incisal notching), adjunctive radiographic examination should be performed to exclude the possibility of an underlying invagination (Fig. 2).³³

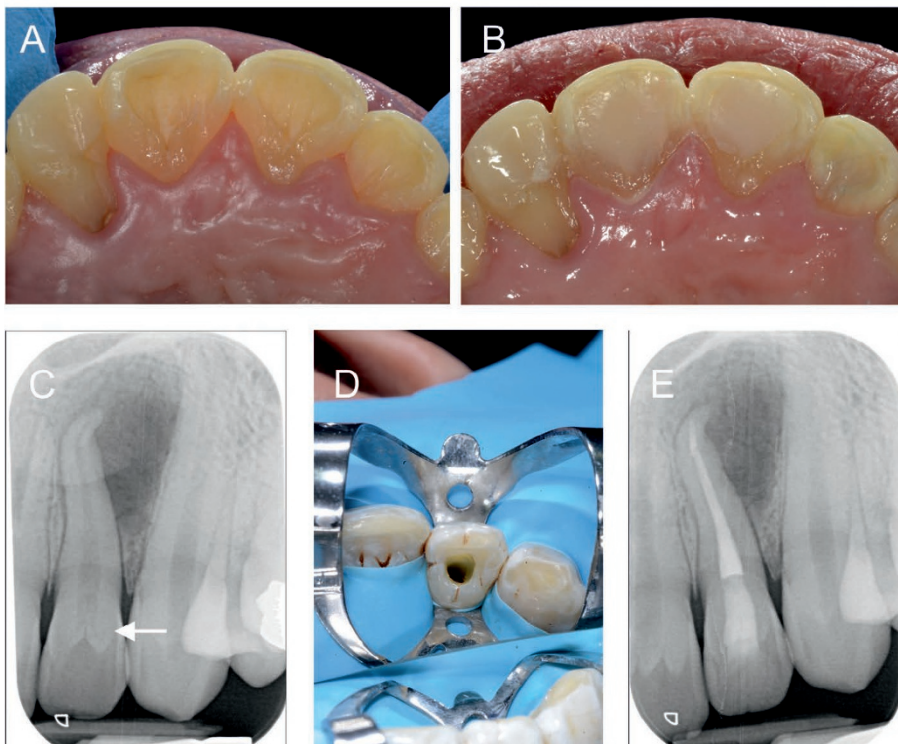


Fig. 2 a) Central incisors with bilateral Type I dens invaginatus lesions. Note the pronounced palatal gingivum; b) Post-treatment: The central incisors were sealed with acid-etched flowable composite; c) A pre-treatment lateral incisor with a Type I dens invaginatus lesion and a large periapical radiolucency; d) The Type I invaginatus lesion was incorporated into the access cavity; e) Post-treatment radiograph of the Type I dens invaginatus lesion. The lesion was fully incorporated into the access cavity and the tooth obturated with thermoplastic GP

Finally, patients may present with symptoms of irreversible pulpitis or apical periodontitis. The possibility of DI should be considered in cases where there is no obvious history of trauma or caries, especially if the lateral incisors are affected bilaterally.

Radiographic features

Invagination lesions usually appear as radiolucent pockets underneath the cingulum and incisal edges of affected teeth. These pockets can be surrounded by radio-opaque enamel and may either be confined to the crown or involve the pulp.

More extensive lesions may appear as fissures, with or without radio-opaque borders. These fissures may involve the pulp and the root canal anatomy may appear complex.³⁷ Communications between the invaginations and the periodontal ligament may be evident either on the lateral aspects of the tooth or its apex. If such communications are present and a patient develops pulpal pathology, a 'butterfly' like periapical radiolucency may be seen, corresponding to two sources of inflammation.³³

These features may be picked up as incidental findings on routine radiographs and warrant further investigation. Periapical radiographs are usually the image of choice for identifying DI lesions, however two images would be required at different horizontal angulations to ensure the lesion is not masked on a single view.³³

The use of cone beam computed tomography (CBCT) has been described in assessing dens invaginatus lesions prior to treatment.³⁸ CBCT can provide a more detailed 3D view of the complex anatomical variations seen in dens invaginatus malformations. It can help the clinician diagnose the class of dens affecting the tooth and allow clinicians to assess the feasibility of any planned endodontic treatment, especially proximity of adjacent anatomical structures if apical surgery is being planned.

Management strategies

The anatomy of DI lesions can be extremely complex.³⁹ Consequently, adequate chemo-mechanical debridement and obturation of these malformations can be challenging. Treatment is further complicated by the tendency for affected teeth to develop pulpal necrosis, often before root formation has taken place. This can result in a 'blunderbuss' apex which can complicate obturation of an affected tooth due to the lack of an apical constriction to compact gutta percha

against (Figs 3a-c). These factors had previously meant that many teeth affected with DI lesions were routinely extracted if patients presented with peri-radicular pathology. However, improvements in modern endodontics have now meant that more of these teeth can be treated successfully with a combination of non-surgical and sometimes apical surgical techniques. Despite these improvements, treatment of these lesions is still challenging and should not be underestimated. Consequently most cases tend to be treated in specialist practice or the secondary care sector. The management of invaginated immature teeth with necrotic pulps can be extremely complex.⁴⁰ In these situations a multi-professional approach may need to be considered. Teeth with complex and untreated dens may need to be extracted and replaced with a prosthesis. Alternatively the resulting

space can be orthodontically closed (to avoid a prosthesis) or unaesthetically wide spaces should be orthodontically optimised to allow prosthodontic rehabilitation.

Within this section, potential operative management options for DI lesions will be discussed.

Treatment of Oehler's Class I Lesions

Class I invaginations can be minimal. As soon as a lesion is detected either clinically or radiographically, the palatal surface of the tooth should be sealed with acid-etched flowable composite to prevent the development of caries (Figs 2a and b).²

If pulpal necrosis is evident, de nova root canal treatment should be performed. The root canals of teeth with Class I lesions are usually not grossly deformed. However, the entire

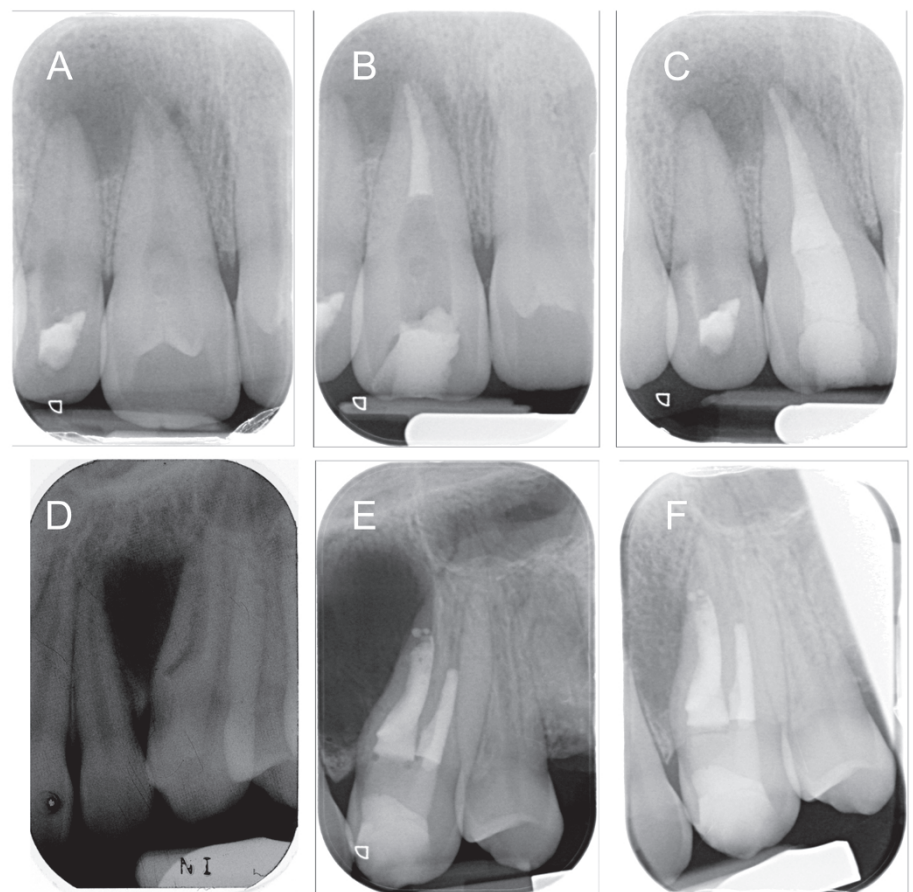


Fig. 3 a) Intraoral radiograph of a maxillary central incisor with a Type II dens invaginatus lesion and incomplete root formation; b and c) The root canal system was managed with MTA apically and obturated with thermoplastic GP coronally. Notice how the dens has been incorporated into the root canal system; d) A pre-treatment intraoral radiograph of a Type III dens invaginatus lesion. The internal root canal anatomy is grossly distorted and the invagination communicates with the periodontal ligament laterally; e) Post treatment: the lesion was filled with MTA and the root canal system was obturated with thermoplastic GP; f) One year post treatment and the lesion is starting to show signs radiographically of bony healing

invagination should be incorporated into the access cavity (Figs 2c-e).

Instrumentation of the invagination can be achieved with Gates Glidden burs or ultrasonic tips (Figs 4a and b).⁴¹ An operating microscope can be useful in ensuring that the invagination is completely eradicated.³³

Treatment of Oehler's Class II Lesions

In Class II invaginations, a more extensive defect is present. Caries can develop deep within these lesions, leaving the enamel surface appearing intact and underlying caries clinically undetectable. It is therefore not advisable to seal these lesions using the methods described for Class I lesions. Opening the entrance of the invagination will allow any caries to be visualised and removed with long-shanked burs (Fig. 4c). The invagination should be thoroughly debrided using ultrasonic instruments and hypochlorite. If the tooth does not exhibit any signs of pulpal pathology the invagination can be restored without accessing the root canal system. As Class II lesions may be in close proximity with the pulp, the invagination can be dressed with a material that will promote hard tissue formation such as mineral trioxide aggregate (MTA).³³ The remaining defect can then be restored and sealed with composite resin.

However, MTA has a prolonged setting time and can be difficult to manipulate. It is also expensive and has the potential to discolour the tooth, which is not ideal in the anterior dental segment. An alternative would be to use setting calcium hydroxide cement, for example, Dycal, when operatively managing an Oehler's Class II invagination.

If the invagination and resultant caries has caused pulpal necrosis, root canal therapy is indicated. As with Class I defects, the invagination should be incorporated into the defect using suitable diamond burs, long shanked stainless steel round burs and ultrasonic alloy tips.

Treatment of Oehler's Class III Lesions

The management of Class III lesions is more complex. If the tooth is asymptomatic and does not exhibit signs of pulpal disease, the tooth should be sealed only. Due to the close proximity of the invagination to the root canal system, prophylactically accessing and debriding Class III invaginations would likely result in pulpal involvement. Therefore, a more conservative approach is recommended.

Peri-invagination periodontitis is a condition in which the tissue within an invagination

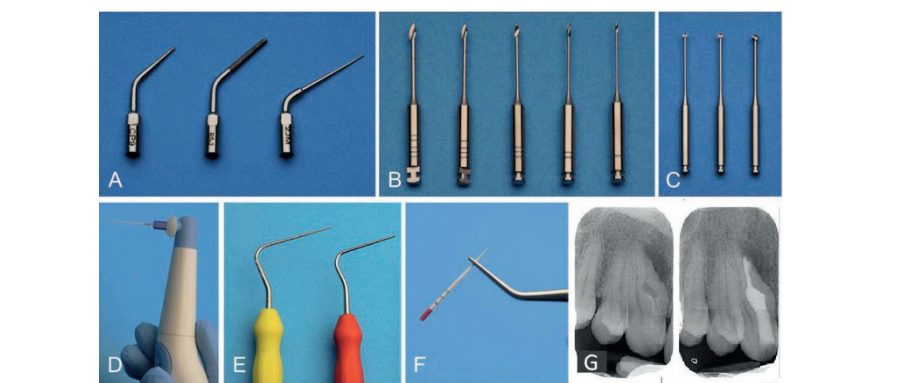


Fig. 4 a) Ultrasonic alloy tips; b) Gates glidden burs; c) Long-shanked rose head burs which can be used to debride the invagination and incorporate Type I and II invaginations into the access cavity. d) An ultrasonic tip can be used to activate irrigants and ensure penetration of the irrigant into the invagination; e) Whilst placing a MTA plug a paper point can be held upside and used to condense and adapt the MTA to the apical walls; f) Whilst placing a MTA plug a paper point can be held upside and used to condense and adapt the MTA to the apical walls; g) A Class IIIb invagination pre-and post-treatment after obturation of the invagination only

becomes infected. The pulp itself may still remain vital and healthy, with the tooth responding to pulp testing. Treatment of these lesions involves attempting to endodontically treat the infected invagination. This is complex and referral to an endodontic specialist is indicated. Depending on the relationship of the invagination and root canal system, the invagination can either be treated in isolation (Fig. 4g) or in combination with the root canal system if both the pulp and invagination are infected.

Given that histologically the pulp/dentine complex and invagination are often interconnected,³³ endodontic therapy of both systems is not unwise and will allow the patient to have the tooth treated in its entirety.⁴² To manage such lesions, good quality peri-apical radiographs (often at different angles) and an operating microscope are essential. The invagination will be lined with enamel and cementum. Endodontic files are not indicated for use to debride these lesions as they will be ineffective. Ultrasonic alloy tips can be used to debride the lesions, and irrigants should be ultrasonically activated (Fig. 4d) to maximise their efficacy and ensure that they reach all parts of the anomaly.⁴³ The invagination will communicate with the periodontal ligament space and will need obturating with MTA to promote healing of the peri-radicular tissues (Figs 3d-f). The pulpal portion of the tooth should be treated conventionally with endodontic files, thorough irrigation of sodium hypochlorite (which should be ultrasonically activated) before being obturated with thermoplastic gutta percha (to ensure that the complex anatomy has been completely sealed) (Figs 3d-f).

The tooth should then be kept under long term review. If symptoms do not improve, microsurgery can be considered or alternatively the dental unit should be extracted.

Conclusion

Teeth affected with dens invaginatus lesions are predisposed to developing pulpal disease. The resulting endodontic treatment is complex due to the unpredictable and aberrant internal anatomy of teeth affected with these lesions. Although CBCT, modern endodontics and surgical endodontics have improved the management of such anomalies, early identification and prophylactic treatment of these lesions is imperative in preventing pulpal pathology from developing and avoiding complex and specialised endodontic treatment.

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