

Endodontic treatment of three types of *dens invaginatus*: Report of four cases

Jefferson J. C. **MARION**¹

Maria L. **MESQUITA**²

Thais Mageste **DUQUE**³

Francisco J. **SOUZA FILHO**⁴

ABSTRACT

Dens invaginatus, also known as dens in dens, is a developmental abnormality that presents alterations in the form and volume of teeth, which may affect the crown and root. Its complex anatomy makes endodontic treatment much more difficult to be performed. Four cases of endodontic treatment in teeth with this type of anomaly are presented; one case type I, one case type II and two cases type III, according to Oehlers' classification. Endodontic treatment only was performed in three of these cases, and endodontic re-treatment and surgical complementation in one case.

Long-term preservation of cases 2 and 4 demonstrated periapical repair with apical closure. Case 1 demonstrated total removal of invagination and the formation of an apical mineralized barrier. Preservation for case 3 was not possible because the patient moved away and contact was lost. Although the treatment of teeth with dens invaginatus is complex, it may be successfully performed when supported by correct diagnostic and planning. If necessary it can be complemented with surgical intervention.

Keywords: Endodontics. Calcium hydroxide. Advanced treatment.

How to cite this article: Marion JJC, Mesquita ML, Duque TM, Souza Filho FJ. Endodontic treatment of three types of dens invaginatus: Report of four cases. Dental Press Endod. 2012 Apr-June;2(2):71-9.

» The authors report no commercial, proprietary or financial interest in the products or companies described in this article.

¹PhD student of Endodontics, UNICAMP/Piracicaba. Professor of the Department of Endodontics, ABO and UNINGÁ.

²Specialist in Periodontics, Bragança Paulista College.

³MSc student of Endodontics, UNICAMP/Piracicaba.

⁴Full Professor of the Department of Endodontics, UNICAMP/Piracicaba.

Submitted: July 03, 2012 - Accepted: August 05, 2012.

Contact address: Jefferson José de Carvalho Marion
Rua Néo Alves Martins, 3176, 6º andar, Sala 64 – Zip code: 87.013-060
Maringá/PR, Brazil – E-mail: jefferson@jmarion.com.br

Introduction

During jaw formation some abnormalities may occur resulting in dental malformations such as *dens invaginatus* (DI).¹ Some authors describe DI as an alteration caused by the invagination of the enamel organ internal epithelium before its mineralization. In a specific moment during development, a relatively developed amelo-dental structure is formed inside the pulp.^{2,3,4}

Teeth affected by this anomaly presents, radiographically, an invagination of the enamel and dentin that can extend deep into the pulpal cavity, and into the canal, sometimes reaching the root apex.¹ The cavity that is formed, generally from the tooth's palatal aspect, advance towards the pulpal cavity, involving it, but keeping communication with the outside through a small opening on the surface of the crown, which may lead to the retention of food residues.⁵ This malformation may also be found in the literature as: Dens in dens, invaginated odontoma, dilated composed odontoma, telescopic tooth, dilated gestant odontoma, tooth inclusion. This wide range of names is the result of different theories on DI etiology.^{1,2,3} The dental elements mostly affected by this abnormality are the permanent upper lateral incisors, followed by the central incisors, canines, upper premolars, as well as the lower incisors, canines and premolars. The occurrence of DI in the posterior teeth is rare but, when it occurs, is more frequently seen in the cervical area.^{2,3,6,7}

Depending on the level of the tooth's involvement by the invagination, Oehlers⁸ classified DI under three different forms: Type I, small invagination lined with enamel restricted to the dental crown, not extending beyond the cemento-enamel junction; type II, moderate invagination lined by enamel, extending apically beyond the cemento-enamel junction, which may or not be in communication with the dental pulp, but confined inside the root as in a blind sac; Type III, invagination lined by enamel, extending beyond the cemento-enamel junction, penetrating along all the root's extension, normally without communication with the dental pulp and revealing a second lateral or apical foramen on the root's surface.

DI diagnostic may be performed clinically: The morphology of the affected crown may vary from normal to abnormal, depending on the size of the invagination,

with a relatively deep pit on the tooth's palatal region, and a cingulate accentuation.² However, the main tool for identifying this anomaly is the radiograph which can show an image similar to a tooth inside another. The radiolucent image in the interior of DI should not be interpreted as lack of pulpal tissue, but just as the invagination intimacy.⁹ Sometimes, DI may be recognized radiographically even before the tooth has erupted into the oral cavity.²

Several factors may influence in the choice of DI treatment. For instance, the patient's age, the patient's physical, psychological and financial situation, the type of invagination, the possibility of access, the form and the localization of the invagination opening in the crown, the root canal system configuration and the tooth's esthetical function.^{2,3} Sometimes, the affected tooth may present incomplete rizogenesis, in which case apexification is recommended.¹ Therefore, several treatment modalities have been proposed for DI management, including preventive restorative procedures, conventional endodontic therapy, paraendodontic surgery, intentional dental re-implant. The treatment is complex and no method may be absolutely proposed due to the variety of existing malformations. Thus, the treatment is based on the anomaly's signs and symptoms, and the prognosis is frequently questionable.^{2,7,10,11} Nevertheless, the literature has shown that, even in cases where endodontic therapy seems to be technically unsatisfactory, successful outcomes may be obtained.¹²

Therefore, due to the difficulties normally found in the endodontic management of DI, the objective of this paper is to report on the treatment of four cases of teeth presenting this developmental anomaly. All teeth presented periapical lesions and incomplete rizogenesis, contributing to the complexity in obtaining a satisfactory endodontic treatment. In addition to that, the paper presents the positive results obtained through the long-term clinical and radiographic preservation in two of these cases.

Case Reports

Case 1

A male patient, aged 31 years with a fistula in his left upper lateral incisor was referred to endodontic treatment in September 2010. There was no history of systemic diseases, and his last dental treatment had

taken place 4 years previously. A gutta-percha cone was used to trace the fistula. A periapical radiograph showed a radiopaque projection inside tooth 22, lined by enamel (Oehlers's type I DI), apical radiolucency and an open apex (Fig 1A). Graphic resources from Microsoft Office PowerPoint 2010 were used to better visualize the image's volume (Fig 1B). Moreover, Corel Draw X5 was also used to better define the figure's outline on the radiographic image (Fig 1C). The tooth did not respond to the pulpal sensitivity, percussion and palpation tests, suggesting the probable clinical diagnostic of chronic periapical abscess.

After the administration of local anesthesia, a rubber dam was placed and stabilized with cyanoacrylate and dental floss. The access cavity was performed on the tooth's incisal edge with a high speed diamond bur until the invagination had been totally removed. Biomechanical preparation was

achieved with manual endodontic files and 2.5% sodium hypochlorite solution, and the working length was confirmed (Fig 1D). The canal was dried with sterilized paper cones, and flooded with 17% EDTA trisodium solution for 3 minutes, after which the canal was dried once again. Calcium hydroxide paste with propylene glycol was placed inside the canal, which was replaced monthly to induce apexification, or the formation of a mineralized barrier on the apical foramen. After six months, the formation of an apical barrier was observed radiographically, which was detected with a gutta-percha cone as a probe. It was also possible to observe that the periapical healing was in the process of completion (Fig 1E). Final obturation was performed in March 2011 with a calcium hydroxide-based cement and gutta-percha by lateral condensation, followed by additional vertical condensation (Fig 1F).

Figure 1. **A)** Initial radiograph showing fistula tracing. **B)** graphic resources, showing the tooth's volume. **C)** graphic resource defining the tooth's anomaly contour. **D)** Confirmation of working length. **E)** Cone test showing apical barrier. **F)** Final radiograph.



Case 2

A 14-year-old female patient was referred to our private clinic in April 2001 with a periapical radiograph of tooth 12 showing an Oehlers's type II DI image associated to incomplete root formation, an open apex and a periapical radiolucent image, with the probable diagnostic of chronic periapical abscess (Fig 2A). In this case also, graphic resources were used as described previously for the same objectives (Fig 2B). The intraoral clinical examination showed the absence of edema, or pain on percussion and palpation, but there was a fistula which was traced with a gutta-percha cone (Fig 2C). There was no systemic diseases, but the patient reported to be a mouth breather and did not permit the placement of a rubber dam in any of the appointments. Hence, tooth's relative isolation was used only.

After local anesthesia and the tooth's isolation, the coronal access was performed through the incisal edge, because the crown was flared, and also to facilitate access to the canal. As it was an Oehlers's type II DI case, it was not possible to remove all the invagination, not even during the biomechanical preparation with manual endodontic files and 1% calcium hypochlorite solution. The working length was determined (Fig 2D). The canal was dried

with sterile paper points and flooded with 17% EDTA trisodium solution for 3 minutes, after which the canal was dried once again and filled with calcium hydroxide paste, propylene glycol and iodoform (Fig 2E). The dressing was changed whenever radiographs showed that the intracanal medication had been partially reabsorbed. Five months later, the canal was obturated as described for Case 1. At this moment, radiographs showed that the cervical and apical to distal grooves had been filled by the obturating cement, and there was the partial repair of the periapical lesion and apical sealing (Fig 2F).

Three months after the case had been concluded, the patient was called for her first follow-up radiograph, which showed the evolution of the repair and apical closure (Fig 2G). The second follow-up visit was one year later when repair was almost concluded and a radiolucent area appeared inside the canal due to the obturating cement reabsorption (Fig 2H). Five years and nine months later, the patient returned for her third follow-up examination. The radiograph showed that the periapical lesion had been completely repaired, the obturating cement in the canal's apical groove had been totally reabsorbed, and the crown had been definitely restored (Fig 2I).

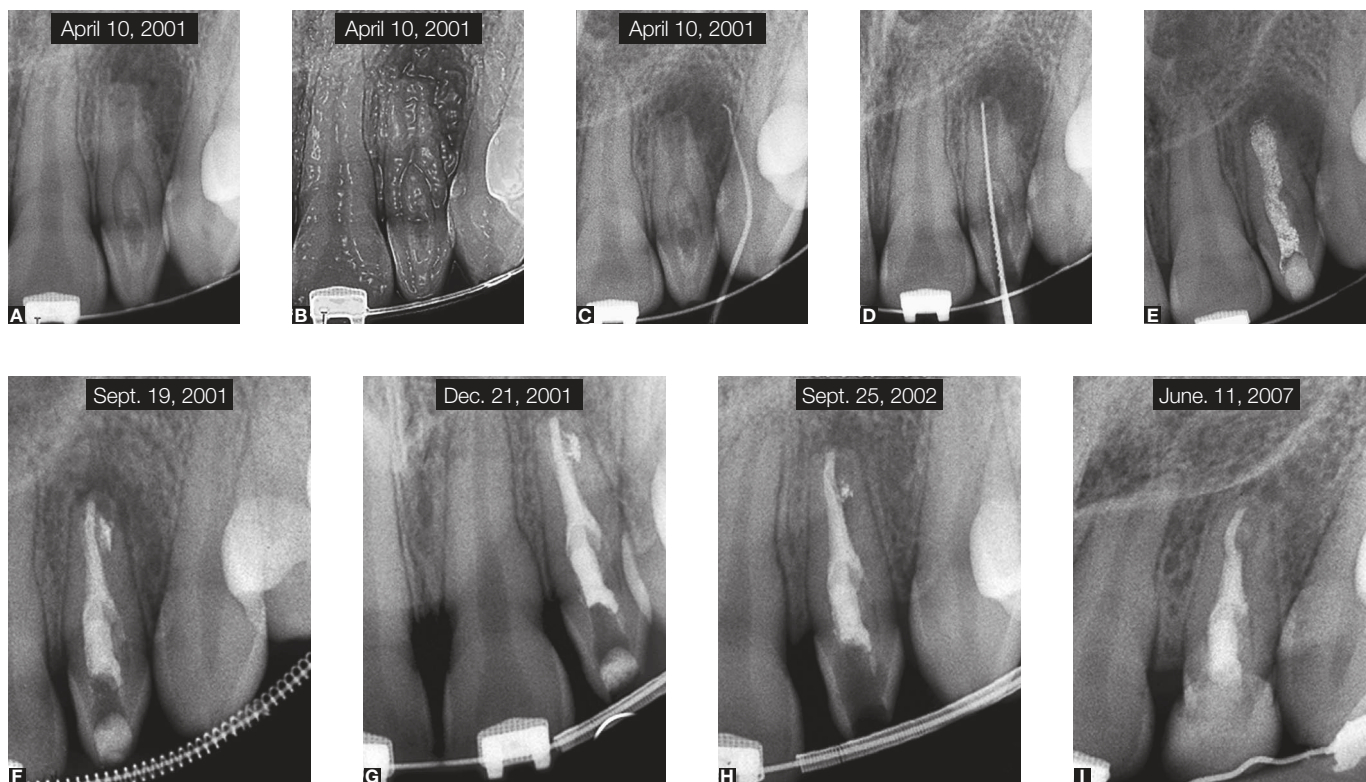


Figure 2. A) Initial radiograph; B) Graphic resource; C) Fistula tracing; D) Confirmation of working length; E) Intracanal dressing; F) Final radiograph; G) follow-up - 3 months; H) 1 year; I) 5 years and 9 months follow-up.

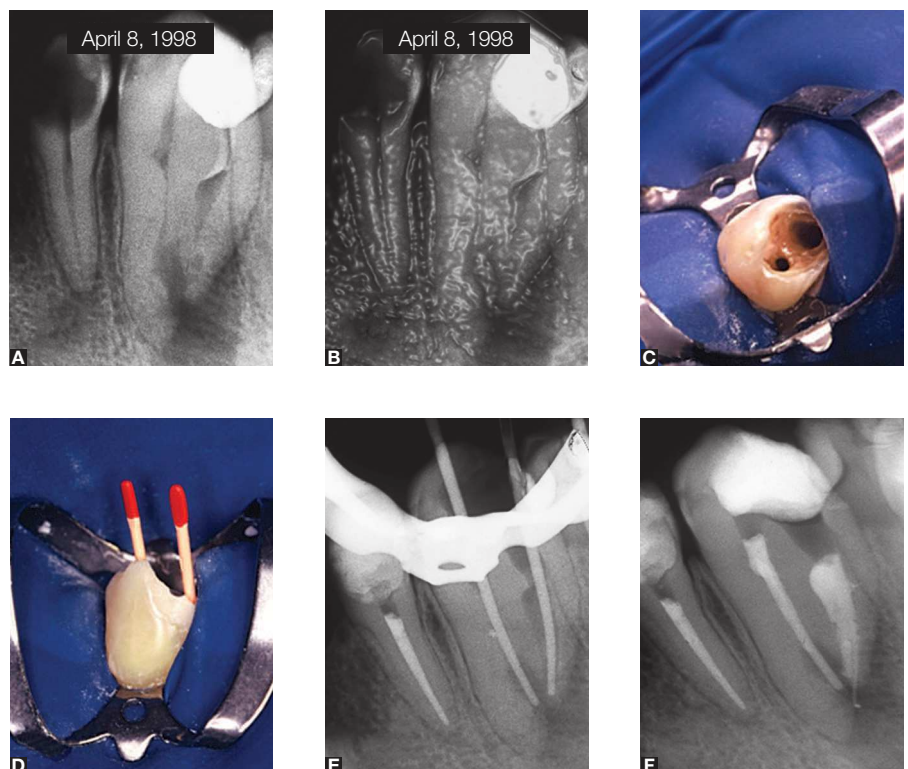
Case 3

A male patient, aged 32 years was referred to treatment in April 1998 and reported that about 30 days previously had felt intense pain in the right lower canine region. On that occasion, coronal access and the placement of an intracanal dressing only had been performed. A periapical radiograph of tooth 43 showed Oehlers's type III DI, with open apex, and radiolucent lesion, suggesting chronic periapical abscess. During emergency, the clinician had accessed just one canal (Fig 3A and 3B). Clinically the tooth did not respond to the pulpal sensitivity, percussion and palpation tests, and a large volume was present in the dental crown.

After local anesthesia, a rubber dam was placed and the canals were accessed (Fig 3C). Both the biomechanical preparation and the application of 17% EDTA tri-sodium solution followed the same technique described for case 2. To assist in the sanitizing of the root canal,

calcium hydroxide paste with propylene glycol was used as an intracanal dressing. As the patient was going to move away to another city, it was not possible to change the dressing in an attempt to induce mineralized tissue formation apically before the definitive obturation of the root canal. Hence, the gutta-percha cones test (Fig 3D and 3E) was carried out to verify their locking inside the canal and confirm the working length. After that, the canals were obturated as described in the previous cases. The obturation radiograph (Fig 3F) showed that a cone had exceeded the obturation limit in one of the canals, and there was a radiolucent area laterally to the cone in the other canal. It was proposed to the patient to endodontically re-treat the tooth, but he refused due to his urgency to have the treatment finished. Six months later, a contact was tried to invite the patient perform a follow-up radiograph. Unfortunately, it was not possible and thus, preservation was not performed.

Figure 3. **A)** Initial radiograph showing the presence of dens invaginatus type III; **B)** With graphic resource showing volume; **C)** Coronal access to two canals; **D)** Photograph showing the main cone test; **E)** Radiograph showing the main cone test; **F)** Final radiograph.



Case 4

A male patient was referred to endodontic treatment of tooth 22 in August 2001, due to the presence of a fistula. There was no systemic problems and, during the intraoral examination, the fistula was traced with a gutta-percha cone. A lateral radiograph (Fig 4A) confirmed the presence of an Oehlers's type III DI. The fistula was traced back to its origin, a possible root defect due to malformation. The radiographic images were manipulated with the same tools and objectives as in case 1 (Fig 4B and 4C). In order to correct the root defect, endodontic re-treatment complemented by a periapical surgery was scheduled.

After local anesthesia, a rubber dam was placed and the root canals were accessed. The obturating material was removed with Gattes Glidden burs, and the canals were shaped manually with endodontic files irrigated with 1% sodium hypochlorite solution. Then, the working length was confirmed (Fig 4D), and 17% EDTA tri-sodium solution was applied to provide better cleaning to the canals. The canals were dried with sterile paper points and filled with calcium hydroxide paste and propylene glycol. The dressing was changed just once, since this tooth would be referred to paraendodontic surgery. The canals were obturated in October 2001 with gutta-percha cones and a calcium hydroxide-based cement

with the intentional extravasation of the obturating material, so that it could serve as a guide during surgery, as well as to promote a better sealing (Fig 4E).

The paraendodontic surgery was carried out by lifting a total thickness flap through two vertical relaxing incisions, keeping the interdental papillae in place, and exposing the root portion and the extravasated obturating material (Fig 4F). This extravasated material was then removed, the root defect was smoothed (Fig 4G), and the root lodging was obturated with MTA, until it was completely filled (Fig 4H). Finally, the gingival tissue was repositioned and sutured with Ethicon 6-0 thread (Fig 4I). The final periapical radiograph showed that the extravasated sealing material had been removed and the radiolucent area had been filled with obturating material (Fig 4J). The sutures were removed one week after the intervention.

Six years and one month later, the first follow-up radiograph showed clinical repair, characterized by the presence of a continuous lamina dura surrounding the whole periapical region, as well as the absence of a fistula and symptoms (Fig 4K). Nine years and six months after treatment, the patient returned for a second follow-up radiograph that showed the same characteristics as before, and the final prosthetic treatment given to the crown (Fig 4L).

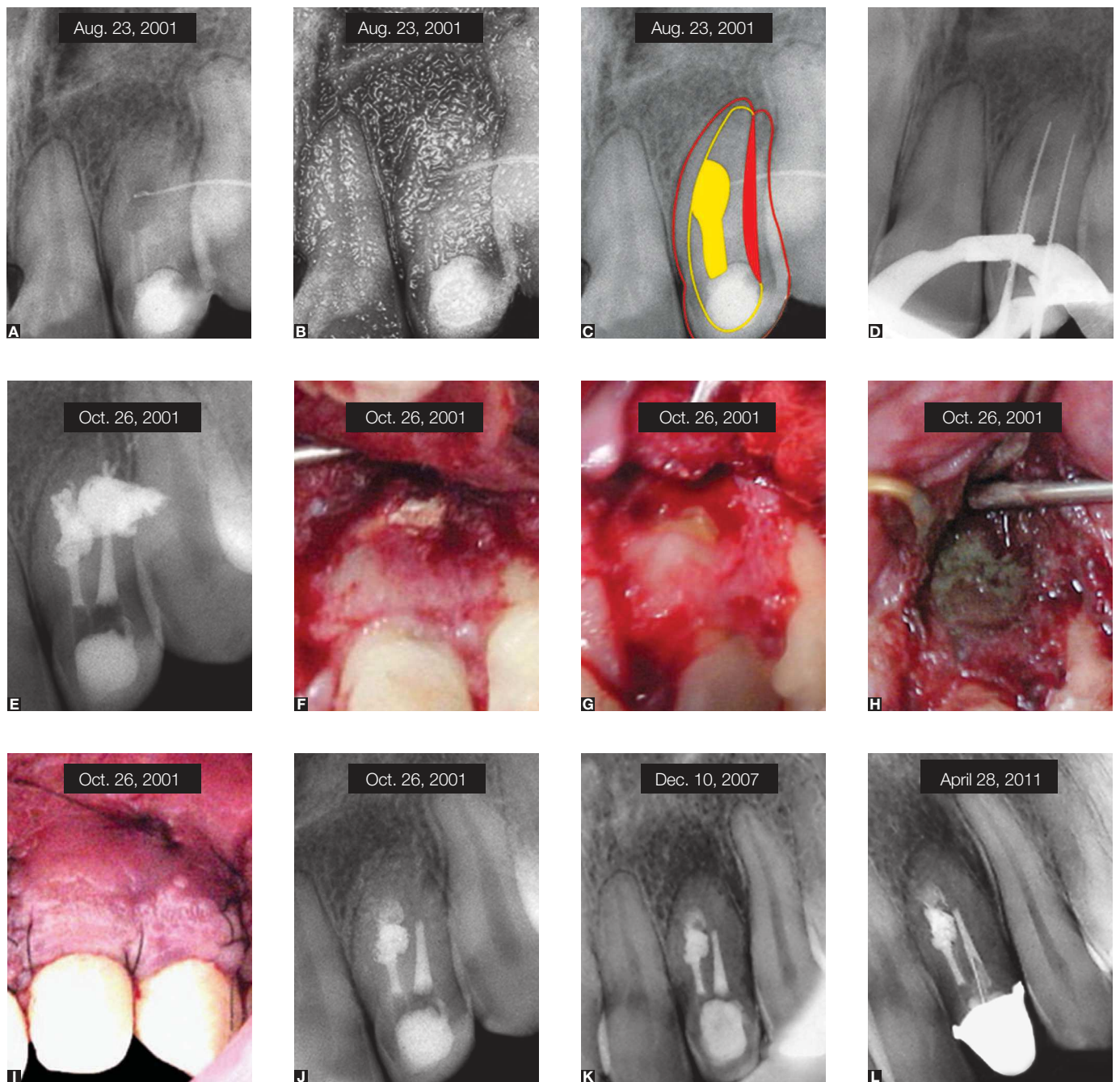


Figure 4. A) Initial radiograph showing fistula tracing; B) Graphic resource showing volume; C) Graphic resource defining the tooth's anomaly outline; D) Confirmation of working length; E) Extravasated obturating material; F) Transoperative photograph showing the root portion with extravasated material; G) After extravasated material removal; H) MTA inserted in the root defect; I) Tissue repositioning; J) Final radiograph; K) Follow-up radiograph; L) Follow-up radiograph.

Discussion

Previous studies report on a high incidence of DI in permanent upper lateral incisors,^{2,3,6,7,10} which is in agreement with this report (three out of four cases). The radiographic images of this anomaly also showed rarefaction in the periapical area as a result of the evolution of the infectious process, and apexes with incomplete rizogenesis, as reported in the literature.^{1,2,3,9} Therefore, after careful analysis, we opted for the initial endodontic intervention to stimulate the regression of signs and symptoms, since pulpal pathology was verified and endodontic therapy was indicated.¹¹

The cases 1, 2, and 3 correspond to Oehlers's type I, II, III DI, respectively, and all presented chronic periapical lesions and incompletely formed apexes. In these conditions, endodontic therapy is faced with a complex root anatomy that hampers treatment. That is the reason why initial sanitizing, with the removal of necrotic tissue and the combat against bacterial infection with abundant irrigation with a sodium hypochlorite solution, was carried out.^{13,14} Dressing with calcium hydroxide paste was used between sessions to complete disinfection and stimulate the deposition of mineralized tissue.^{1,4,10,15,16} These procedures led to the clinical success in three cases without the need for periapical surgery.¹⁷ These data corroborate the reports of other authors that demonstrated the influence of calcium hydroxide as an intracanal dressing, and the obturation with a calcium hydroxide-based cement for the repair of extensive periapical lesions in teeth with complete apexes.^{16,18,19,20}

The frequency of pulpal necrosis in DI cases is explained by its coronal invagination, which makes teeth more susceptible to dental caries and pulpal infection, due to the structural defect existing at the bottom of the pit. Besides, the hypomineralization makes the internal enamel layer more fragile, or even absent, which facilitates the exposition of the dental pulp by fracture during chewing, or even by the natural microexposition.^{2,3,11}

The case 4 is an Oehlers's type III DI. This tooth had been referred to endodontic re-treatment due to failure in the previous treatment. As in this type of anomaly the invagination may extend through the root in the shape of a cleft in the apical or lateral region without

communication with the pulp, as shown by tracing the fistula with a gutta-percha cone (Fig 4A), endodontic as well as periodontal treatment was recommended.^{21,22}

Therefore, paraendodontic surgery was planned for this case as a complement to the endodontic treatment. The literature states that this complementary approach is aimed at overcoming DI root canal irregularities, because the apical abnormality does not provide a good environment for the correct root canal debridement and obturation. In addition to that, the majority of cases treated between 1977 and 1994 used as a complement to endodontic treatment, the periapical surgery to seal the space existing between the root canal and the periapical tissues.²³⁻²⁹

Figures 4K and 4L illustrate a radiolucent image in the tooth's periapical region which is possible to observe the continuity of the lamina dura around the tooth, clinically indicating repair. This image suggests that it is a scar tissue since, according to Melcher²⁸ and Bosshardt and Sculean,²⁹ if the bone defect is not separated from the connective and/or epithelial tissue by a physical barrier, it will be filled by epithelial or connective cells that proliferates faster than bone and periodontal ligament cells, preventing the defect to be completely repaired, and generating this kind of radiographic image.

This work is in agreement with the work of Girsch and McClammy,⁹ who admit that the prognosis of DI endodontic treatment is doubtful, specifically because of the complex morphology and communication among root canals, which makes the adequate cleaning and shaping of root canal systems more difficult. However, in cases 2 and 4, where clinical and radiographic preservation was possible, the treated teeth remained in the dental arch, keeping their esthetical and functional characteristics.

The four cases reported here demonstrate that DI is a challenge for the endodontic treatment, since this abnormality points to a complex root canal system. Moreover, the endodontic treatment of teeth with DI, although difficult to execute, can be successfully accomplished when supported correct diagnostic and planning and, if necessary, it may be complemented by surgical intervention.

References

1. Hülsmann M. Dens invaginatus: a etiology, classification, prevalence, diagnosis, and treatment considerations. *Int Endod J*. 1997;30(2):79-90.
2. Sousa SMG, Bramante CM. Dens invaginatus: treatment choices. *Endod Dent Traumatol*. 1998;14(4):152-8.
3. Beltes P. Endodontic treatment in three of dens invaginatus. *J Endod*. 1997;23(6):399-402.
4. Sauveur G, Sobel M, Boucher Y. Surgical treatment of lateroradicular lesion on an invaginated lateral incisor (dens in dente). *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1997;83(6):703-6.
5. Yeh SC, Lin YT, Lu SY. Dens invaginatus in the maxillary lateral incisor Treatment of 3 cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1999;87(5):628-31.
6. Fristad I, Molvn O. Root resorption and apical breakdown during orthodontic treatment of a maxillary lateral incisor with dens invaginatus. *Endod Dent Traumatol*. 1998;14(5):241-4.
7. Chen YH, Tseng CC, Harn WM. Dens Invaginatus: review of formation and morphology with 2 case reports. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1998;86(3):347-52.
8. Oehlers FA. Dens invaginatus (dilated composite odontome). I. Variations of the invagination process and associated anterior crown forms. *Oral Surg Oral Med Oral Pathol*. 1957;10(11):1204-18.
9. Girsch WJ, McClammy TV. Microscopic removal of dens invaginatus. *J Endod*. 2002;28(4):336-9.
10. Nedley MP, Powers GK. Intentional extraction and reimplantation of an immature invaginated central incisor. *ASDC J Dent Child*. 1997;64(6):417-20.
11. Holtzman L. Conservative treatment of supernumerary maxillary incisor with dens invaginatus. *J Endod*. 1997;24(5):378-80.
12. Holland R, Souza V, Marion JJC, Anjos Neto DA, Borlina SC, Murata SS. Endodontic treatment of dens invaginatus. *Rev Assoc Paul Cir Dent*. 2008;62(6):476-80.
13. Abou-Rass M, Oglesby SW. The effects of temperature, concentration and type on the solvent ability of sodium hypochlorite. *J Endod* 1981;7(8):376-7.
14. Shih M, Marshall FJ, Rosen S. The bactericidal efficiency of sodium hypochlorite as an endodontic irrigant. *Oral Surg Oral Med Oral Pathol*. 1970; 29(4):613-9.
15. Holland R, Souza V, Tagliavini RL, Milanezi LA. Healing process of teeth with open apices. Histological study. *Bull Tokyo Dent Coll*. 1971;12(4):333-8.
16. Holland R, Souza V, Nery MJ, Mello W, Bernabé PFE, Otoboni Filho JA. Effect of the dressing in root canal treatment with calcium hydroxide. *Rev Fac Odontol Araçatuba*. 1978;7(1):39-45.
17. Er K, Kustarci A, Özcan U, Tasdemir T. Nonsurgical endodontic treatment of dens invaginatus in a mandibular premolar with large periradicular lesion: a case report. *J Endod*. 2007;33(3):322-4.
18. Holland R, Souza V. Ability of a new calcium hydroxide root canal filling material to induce hard tissue formation. *J Endod*. 1985;11(12):535-43.
19. Katebzadeh N, Sigurdsson A, Trope M. Radiographic evaluation of periapical healing after obturation of infected root canals: an in vivo study. *Int Endod J*. 2000;33(1):60-6.
20. Sonat B, Dalat D, Günhan O. Periapical tissue reaction to root fillings with Sealapex. *Int Endod J*. 1990;23(1):46-52.
21. Cecilia MS, Lara VS, Moraes IG. The palato-gingival groove. A cause of failure in root canal treatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1998 Jan;85(1):94-8.
22. Costa WF, Sousa Neto MD, Pécora JD. Upper Molar Dens In dente – Case Report. *Braz Dent J*. 1990;1(1):45-9.
23. Schmitz MS, Montagner F, Flores CB, Morari VHC, Quesada GAT, Gomes BPPFA. Management of Dens Invaginatus Type I and Open Apex: Report of Three Cases. *J Endod*. 2010;36(6):1079–85.
24. Hata G, Toda T. Treatment of dens invaginatus by endodontic therapy, apicocurettage, and retrofilling. *J Endod*. 1989;13(9):469-72.
25. Rotstein I, Stabholz A, Heling I, Friedman S Clinical considerations in the treatment of dens invaginatus. *Endod Dent Traumatol*. 1987;3(5):249-54.
26. Kulild JC, Weller N. Treatment considerations in dens invaginatus. *J Endod*. 1989;15(8):381-4.
27. Benenati FW. Complex treatment of a maxillary lateral incisor with dens invaginatus and associated aberrant morphology. *J Endod*. 1994;20(4):180-2.
28. Melcher AH. On the repair potential of periodontal tissues. *J Periodontol*. 1976;47(5):256-60.
29. Bosshardt DD, Sculean A. Does periodontal tissue regeneration really work? *Periodontol* 2000. 2009;51:208-19.