Dens Invaginatus: Review, Relevance, and Report of 3 Cases

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ABSTRACT

Dens invaginatus is a rare developmental morphoanatomical variation resulting from the infolding of the dental papilla before biological mineralization that allows the invagination of inner dental epithelium. Permanent maxillary lateral incisors are most commonly affected, and the condition is frequently bilateral, but it may also prevail in permanent maxillary central incisors. The purpose of this paper was to provide an overview of the etiopathogenesis, frequency of occurrence, and clinical and radiographic features and to discuss 3 dens invaginatus cases. (J Dent Child 2012;79(3):143-53)

Received December 12, 2010; Last Revision April 14, 2011; Revision Accepted April 15, 2011.

KEYWORDS: CT SCAN, DENS INVAGINATUS, ROOT CANAL TREATMENT

Dens invaginatus (**DI**) is a rare developmental morphoanatomical variation resulting from the infolding of the dental papilla before biological mineralization that allows the invagination of inner dental epithelium.[1] DI was first described by a dentist in 1856,[2] as a "tooth within a tooth" by Salter in 1955,[3] and as "anomalous cavities in human teeth" by Muhlreiter in 1873.[1] This kind of malformation, however, was first described in a whale's tooth in 1794 by Plaquet.[4,5] Tomes reported a case of coronal DI in 1859,[6] and in their article, Swanson and McCarthy[7] discussed the features of bilateral DI.

The purpose of this paper was to provide an overview of etiopathogenesis, frequency of occurrence, categorization, and clinical and radiographic features and discuss 3 cases of DI.

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LITERATURE REVIEW

Among the various names for this anomalous defect (Table 1),[1, 8-12] the term "dens invaginatus" appears to be most appropriate, as it reflects the invagination of the outer portion (enamel) into the inner portion (dentin) with the formation of a pocket or dead space, which generally ocurs before crown calcifications.[13,14] It also represents the range of presentations rather than other descriptions that appear more applicable to specific variations fundamental to the condition.[14] Although the permanent maxillary lateral incisor is the most commonly involved tooth, it may occur in primary teeth[15] as well as in the maxillary and mandibular arches. In most cases, it appears to simply represent an accentuation in the development of a lingual pit.[16]

Table 1. Suggested Synonyms for Dens Invaginatus

1.	"Dens in dente" owing to the radiographic appearance of a tooth within a tooth[8]
2.	"Dilated composite odontome" deduces an abnormal dilation of the dental papilla[9]
3.	"Invaginated odontome"[10]
4.	"Dilated gestant odontome"[10]
5.	"Telescope teeth"[11]
6.	"Tooth inclusion"[1]
7.	"Dentoid in dente"[1]
8.	"Warty tooth"[12]

Table 2. Theories and Conditions Attributed to DI

Year	Author	Theories or attributed association.
1934	Kronfeld[25]	Invagination results from a focal failure of growth of the internal enamel epithelium while the surrounding normal epithelium continues to proliferate and engulfs the static area.
1936 1937	Fischer[26] Sprawson[27]	Infection is considered responsible for this malformation.
1937	Rushton[28]	Benign neoplasm of limited growth.
1939 1943	Euler[29] Atkinson[30]	Advocated the condition to growth pressure of the dental arch resulting in buckling of the enamel organ.
1978	Casamassimo et al.[32]	Reported a familial association of microdontia, taurodontism, and dens invaginatus inherited as an X-linked recessive trait in a 12-year-old boy.
1950	Bruszt[31]	Gave twin-theories and suggested a fusion of 2 tooth germs.
1950	Gustafson and Sundberg[33]	Attributed trauma as a causative factor for the anomaly.
1957	Oehlers[34,35]	Distortion of the enamel organ during tooth development and subsequent protrusion of a part of the enamel organ may lead to the formation of an enamel-lined channel ending at the cingulum or occasionally at the incisal tip, which might be associated with irregular crown form.
1959	Grahnen et al.[36]	Reported multiple DI in 2 brothers in a clinical, and genetic study of maxillary lateral incisors.
1970	Schultze[2]	Suggested DI as a deep folding of the foramen coecum during tooth development.
1971 1987 1997 2009	Rantanen,[37] Ireland et al.[38] Jimenez-Rubio et al.[39] Kirzioglu and Ceyhan [40]	Advocated that dental anomalies such as DI, dens evaginatus, fusion, germination, and agenesis occurred because of the degeneration or hyperactivity of dental lamina and is more frequently seen in the anterior region.
1973 1990	Burzynski[41] Nazif and Laughlin[42]	Reported concomitant occurrence of gemination and DI.
1974	Ekman-Westborg and Julin[43]	Reported a case of macrodontia, mutituberculism, central cusps, and pulp invaginations in the permanent dentition with no involvement of hereditary factors.
1987	Ireland et al.[38]	Showed association of short roots, taurodontia, and multiple dens invaginatus.
1990	Mann et al.[44]	Reported in the skull of a 5-year-old Native American who displayed macrodontia, shovel-shaped maxillary central incisors, 3-rooted primary mandibular molars, DI, agenesis of permanent maxillary canines, and crenulated occlusal surfaces of permanent first and second primary molars discovered as part of an archaeological exploration.
1994	Pokala and Acs[45]	Suggested that an individual lacking chromosome 7q32 had DI and hypodontia along with other dental abnormalities.
1995 2005	Oncag et al.[46] Hibbert[47]	Suggested the association of DI with Williams and Nance-Horan syndromes.
1996	Hosey and Bedi[48]	Reported possible role of genetic factors in DI.
1999	de Souza et al.[49]	Reported DI in both maxillary and mandibular central incisors.
2000 2004	Kettunen et al.[50] Ohazama et al.[51]	Described specific roles of ectomesenchymal signaling systems in the regulation of growth and the infolding of the enamel organ during tooth development.
2000	Dassule et al.[52]	Suggested that the absence of certain substances can result in abnormally shaped teeth as well as defects in the developing tooth germ.
2002	Goncalves et al.[13]	An association between the invagination and talon cusp.
2003 2004 2004 2008	Lorena et al.[53] Mupparapu et al.[54] Tikku et al.[55] Anthonoppa et al.[56]	Reported rare occurrence of DI and dens evaginatus (or talon cusp).
2005	Suprabha[57]	Described a case of double dens in dente in a patient with a feature of albinism.
2006	Desai et al.[58]	Reported a peculiar combination of all the traits associated with short root anomaly, including generalized microdontia, taurodontism, multiple dens invaginatus, periapical radiolucency, obliteration of pulp chambers, and increased tooth mobility leading to spontaneous exfoliation in a 20-year-old male.
2007	Sannomiya et al.[59]	Described rare associations of DI and mesiodens in an 8-year-old female and a 16-year-old male patient.
2008	Anegundi et al.[12]	Reported a case of coronal double DI occurring in an impacted molariform supernumerary tooth in a 14-year-old boy.
2009	Sedano et al.[60]	5 genes—MSX1, DLX1, DLX2, PAX9, and PITX2 could be responsible for or coparticipators in some dental anomalies present in their patient.[60]

Oehlers[17] further suggested that this condition may affect the posterior dentition; occasionally, an analogous form occurs in tooth roots. Bhatt and Dholakia pointed out that the radicular invagination usually results from an infolding of Hertwig's sheath and takes its origin within the root after development is complete. Radicular dens invaginatus was also reported by other authors.[19-22] Some have described it an "incipient dens in dente" which represents a deep palatal or lingual pit completely lined by enamel with no communication to the pulp.[23,24]

ETIOPATHOGENESIS

Numerous theories and conditions have been suggested for DI however, its exact etiology remains unclear (Table 2).[1,2,12,13,25-60] Besides being an isolated variant of the normal population, the following conditions have been found to be reportedly associated with DI[61]: microdontia, macrodontia, hypodontia, oligodontia, taurodontism, gemination, fusion, supernumerary teeth, dentinogenesis imperfecta, odontomes, coronal agenesis, shovel-shaped incisors, mesiodens, obliterated pulp chambers, C-shaped canal configuration, palatoradicular groove defect, short root anomaly, dilacerations, albinism, periodontal abscess, multiple root canals, cranial suture syndromes, unicystic ameloblastoma, and coronal fractures. Also, multiple dental abnormalities were detected more frequently in patients with chromosomal disparities.[62]

Table 3. Oehler's Classification of DI[34,35]

Type	Description
1	An enamel-lined cavity confined to the crown and not extending beyond the cementoenamel junction
2	An enamel-lined cavity extending into the root beyond the cementoenamel junction ending in a blind sac; there may/may not be communication with pulp
3	An invagination extending beyond the cementoenamel junction perforating laterally (type 3a) or apically (type 3b) at a foramen; usually, there is no communication with the pulp, sometimes it may be completely lined by enamel, and sometimes cementum will be found lining the invagination[34]

Since these malformations are highly reproducible in shape, they show a predilection for some racial groups and often occur together; hence, they seem more likely to be determined genetically.[53]

CATEGORIZATION

Hallet[63] suggested the existence of 4 types of invagination based on clinical and radiographic features. Although Hallet was credited with proposing the first classification of DI the most commonly used was given by Oehlers.[34,35] Oehlers classified DI into 3 categories, according to the depth of penetration and communication with the periodontal ligament or periapical tissue, determined radiographically from the crown into the root (Table 3).

Schulze and Brand[64] suggested an assessment based on 12 possible variations in clinical and radiographic appearance of the invagination, and proposed a more detailed classification, including invaginations and dysmorphic root configurations.[1,14] Two variations (A4, B4) were reported in the classification in which the invagination starts laterally and near the incisal edge and continues attached to the external outline of the affected tooth mesially or distally.[65]

FREQUENCY OF OCCURRENCE

Differences in the study methodology and diagnostic criteria result in a wide variation in reported occurrence of DI ranging between 0.04%[49,66] and approximately 26%.[14,67] Although permanent maxillary lateral incisors are most commonly affected (47%) and the condition is frequently bilateral (43%),[45,68] it may also prevail in permanent maxillary central incisors.[16] Full-mouth surveys conducted in different populations revealed a variable prevalence ofDI (Table 4).[8,24,40,69-79] Type 1 dens invaginatus is most commonly seen (94%), followed by type 3 (33%), and type 2 (4%).[40]

The mandibular occurrence of this anomaly is rare. There are to date 2 cases each in the central incisors (1 bilateral)[80,81] and lateral incisors,[54,82,83] 1 in canines,[84]

Table 4. Frequency of Occurrence of Dens Invaginatus in Various in Various Populations

Year	Author	Distribution of dens invaginatus in population or group
1966	Poyton and Block[69]	Full-mouth surveys conducted revealed that the prevalence of dens invaginatus is approximately 0.25% in the Canadian population.[69]
1952 1955 1974 1977	Boyne[70] Amos[71] Thomas[8] Hovland and Block[72]	Approximately 1%[72] to 8% of the population,[8] 7% of dental students,[71] and <1% of maxillary incisors in the United States.[70]
1971 1974	Miyoshi et al.[73] Fujiki et al.[74]	Approximately 4%[74] to 39% of maxillary lateral incisors in the Japanese population.[73]
1974	Vincent-Townend[24]	2% in the Israeli population.[24]
1986, 1987	Ruprecht[75,76]	Approximately 2% to 10% in Saudi Arabian patients.[75,76]
2001	Backman and Wahlin[77]	Approximately 7% of 7-year-old Swedish children and 3% of maxillary right incisors have been shown to possess dens invaginatus.[77]
2004	Hamasha and Al-Omari[78]	Showed that approximately 3% of patients and <1% of teeth examined have dens invaginatus; among them, approximately 9% were maxillary lateral incisors, <1% were maxillary first premolars, and <1% were maxillary first and third molars in the Jordanian population.
2007	Ezoddini et al.[79]	Approximately 1% in the Iranian population.[79]
2009	Kirzioglu and Ceyhan[40]	12% of the Turkey population shows this anomalous variation; among them, 82% of cases are bilateral.[40]

5 in premolars (2 bilateral),[85-89] and 1 in mandibular third molars.[90] In the primary dentition, second molars,[47,91] maxillary canines,[92] mandibular canines,[15] and maxillary central incisors[93] have been shown to manifest DI. Data records revealed more cases in males and no cases of primary lateral incisor involvement[1] compared to a gender predilection for females and lateral incisor involvement observed in the permanent dentition.[14]

CLINICAL REPORTS

CASE 1

A 13-year-old rural Indian female with non-contributory systemic and family histories reported to a pediatric dentist with the chief complaint of a mobile, excessively large and rotated maxillary incisor with increased interdental spacing, giving her an unpleasant appearance (Figure 1). A detailed history revealed intermittent pain for the last 1.5 years in the same area, for which she took medication from a local rural medical practitioner. The tooth in question had an irregular enamel surface in the cervical third region circumferentially, along with a bulky cingulum.

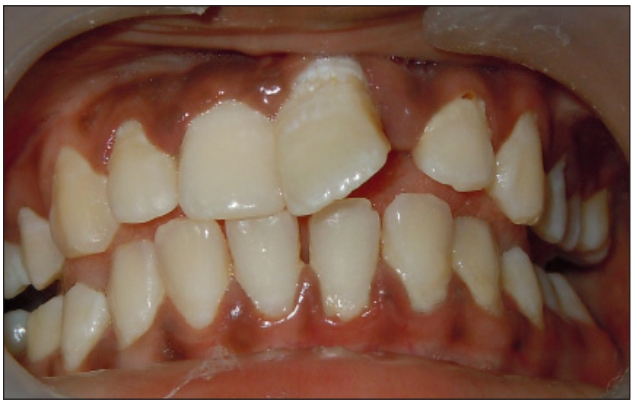

Figure 1. A large and rotated permanent maxillary left central incisor.

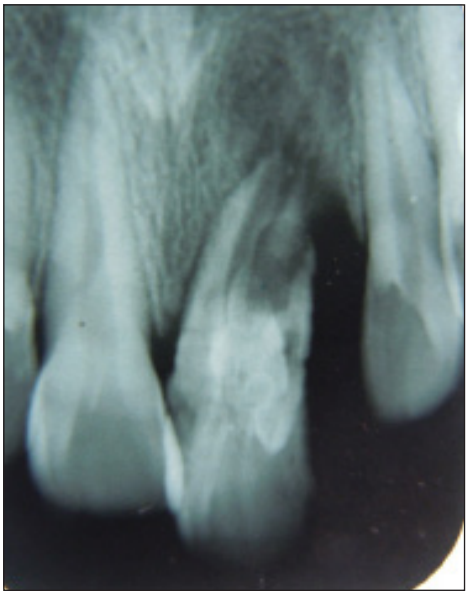

Figure 2. Radiograph of the permanent maxillary left central incisor showing Oehlers type 3 DI.

A periodontal screening showed grade 3 mobility and an 11-mm deep periodontal probing depth on the distal surface of an infant maxillary incisor.

The tooth was sensitive to percussion and nonresponsive to both thermal and electrical pulp testing. A radiographic examination revealed a bizarre morphologic crown and root along with a periapical radiolucency that extended throughout the distal aspect of the root (Figure 2). For accurate information of the morphological variation, eccentric radiographs were taken. An unusual morphology with radiopaque projection attached to the crown, probably lined with radiopaque enamel, appeared as an invagination extending from the crown to the apex that opened on the distal surface. No other associated morphological variations were found on the panoramic radiographs. Careful interpretation of the history and clinical and radiographic features led to the diagnosis of DI type 3 with pulp necrosis.

The case was evaluated by an endodontist, periodontist, and restorative dentist. Due to the poor periodontal condition, complex root canal morphology, large anatomical crown, and smaller anatomical root, the tooth was deemed not restorable per Ante's law (ie, the root surface area of the abutment teeth should be equal to or greater than that of the teeth being replaced with pontics). The patient was advised to undergo extraction with simultaneous socket augmentation for future implant placement. The tooth was extracted on the next visit and the shallow socket was immediately embeded with a sponge form of fisiograft up to its brim and covered with a periodontal flap, suture, and COE-PAK (GC America Inc., Alsip, Ill., USA) for 10 days. Following extraction, the tooth's bizarre anatomy inspired the authors to explore further and understand its morphoanatomical disparity. After 10 days, healing was uneventful and the patient was seen for maintenance visits every 3 to 4 weeks initially for 2 months, increasing to once a month over the next 6 months.

An examination of the extracted tooth revealed a crown-to-root ratio of approximately 2:1, 2 perforations on the palatal side of the 3-mm root, and a few necrosed patches. The tooth was mounted horizontally on a modeling wax sheet and scanned using a 64 Slice computed tomography scanner (Brilliance CT, Philips Medical System Nederlands BV, Best, The Netherlands) to assess pulp chamber morphology; it was viewed using MxLite View DICOM Viewer, (Version 1.24.0.0, Philips Medical Systems, Cleveland), in cross-sectional and longitudinal sections with a constant thickness of 0.1 mm/slice (Figure 3).

CASE 2

A 12-year-old Indian male without any medical and family contributory histories reported to a pediatric dentist with the chief complaint of an unpleasant looking maxillary anterior region. There was no history of pain or discomfort in the area. A clinical examination revealed severe hypo-

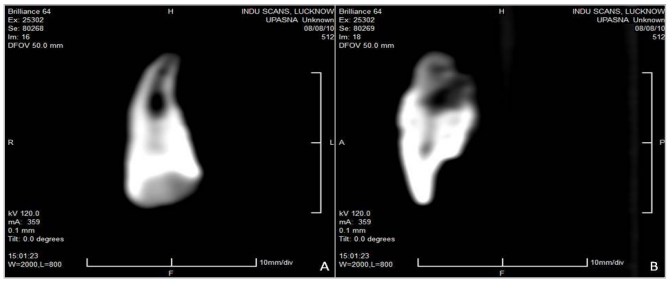

Figure 3. (A) Computed tomography image of the permanent maxillary left central incisor in the sagittal plane showing a bizarre pulp space morphology. (B) Longitudinal section showing 2 openings in the apical region.

plastic defects along with dark brown-black discoloration on the labial surface of the permanent maxillary left central incisor and a slightly wider permanent maxillary left lateral incisor. The permanent maxillary right central incisor was in crossbite with a mesially inclined crown, and a rudimentary permanent maxillary right lateral incisor present. Panoramic and periapical radiographs revealed "pear-shaped" radiopaque enamel invaginations, which extended beyond the cementoenameljunction in the right central incisor and left lateral incisor (Oehlers type 2), and several radiolucent patches present on the crown of left central incisor (Figure 4). Additionally, a missing permanent maxillary right canine and an ectopically erupting permanent maxillary left canine were evident.No other radiographic abnormality was visible either in the root or periapical area.

A comprehensive treatment protocol was made for full-mouth rehabilitation along with a prophylactic sealing of the palatal enamel invaginations of the right central incisor and left lateral incisor. The patient and his parents, however, were reluctant to start a comprehensive rehabilitation procedure that required a multi-

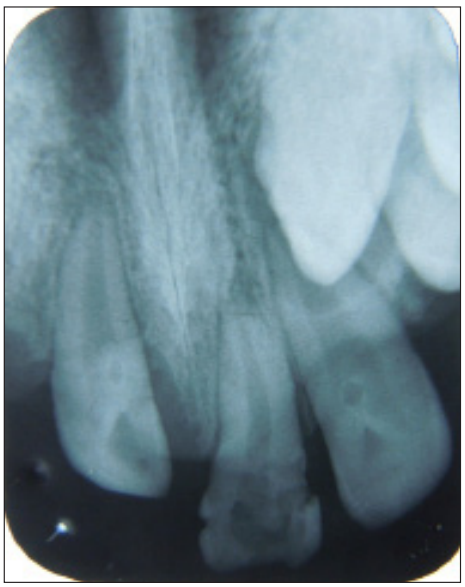

Figure 4. Radiograph showing a pear-shaped radiopacity in the permanent maxillary right central incisor and left lateral incisor.

disciplinary approach and numerous visits for completion of the treatment; they deferred the procedure and did not return for the further treatment.

CASE 3

A 32-year-old Indian female with non-contributory medical and familial histories reported to an endodontist for evaluation and management of continuous dull pain in the permanent maxillary right central incisor that was aggravated by hot and cold subsiding only after taking medication. Her dental history revealed that the right central incisor had endodontic treatment, followed by a porcelain fused to metal crwon. That tooth was asymptomatic. The patient did not have any previous traumatic injury to the dentition. Upon clinical examination, the right lateral incisor was found to be narrower mesiodistally, and periodontal probing revealed a normal intact periodontium.

Careful examination of the tooth surface exposed a deep pit on the lingual surface with no evidence of caries. The tooth in question was not sensitive to percussion and palpation, but responded abnormally to pulp testing; pain lingered after thermal and electrical stimuli in comparison to the contralateral tooth. Radiographic analysis showed a treated root canal in the right central incisor and a pearshaped radiopaque lining, which was inconsistent with the enamel opacity, giving an appearance of enamel invagination with a narrow constriction that opened near the surface of the right central incisor (Figure 5). Eccentric intra-oral periapical and panoramic radiographs taken revealed no further anomalies. A thorough interpretation of the history, clinical and radiographic examination, and pulp investigations suggested the diagnosis of the type 2 DI along with irreversible pulpitis in the right central incisor.

Nonsurgical endodontic therapy was planned for the latter. After routine local anesthesia and isolation with a rubber dam, access was made into the tooth with an Endoaccess bur (Dentsply, Maillefer). Initial exploration with a no. 15 K-file revealed a stony hard obstruction in the pulp space. Gates Glidden burs along with subsequently larger sized K-files and H-files were used to completely remove the dens and to obtain a buccolingually wider canal. The canal was copiously irrigated with 17% EDTA and 3% sodium hypochlorite to remove all organic and inorganic debris and was prepared to ISO size 45. Calcium hydroxide was used as an intracanal medicament and the access cavity was temporarily sealed using an intermediate restorative material. In the subsequent visit after 7 days, the patient was asymptomatic and the canal was laterally compacted with gutta-percha and AH-Plus sealer (Figure 6). The access cavity was restored using light-cured composite r esin. The patient remained asymptomatic when she last reported 6 months after the treatment and continues to be under active follow-up.

DISCUSSION AND TREATMENT OPTIONS

DI often a clandestine finding on radiographs, characteristically presents as a deep invagination in the lingual pit area that may not be recognized clinically.[61] To clarify the point that enamel is located centrally and dentin peripherally due to invagination, Hallet[63] introduced the term "dens invaginatus." Morphologically, a deep invaginated lingual pit leaves the tooth more susceptible to caries, with potential for pulpitis, necrosis, and a periapical manifestation, as constant microbial invasion may occur via the deep pit into the pulp.[94] Other reported sequelae of undiagnosed and untreated coronal invaginations include abscess formation, retention of neighboring teeth, displacement of teeth, cysts, and internal resorption.[55]

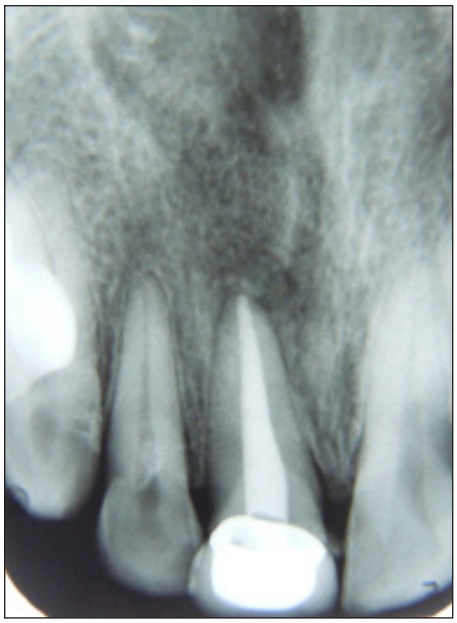

Figure 5. Radiograph showing a pear–shaped radiopacity in the permanent maxillary right central incisor and left lateral incisor.

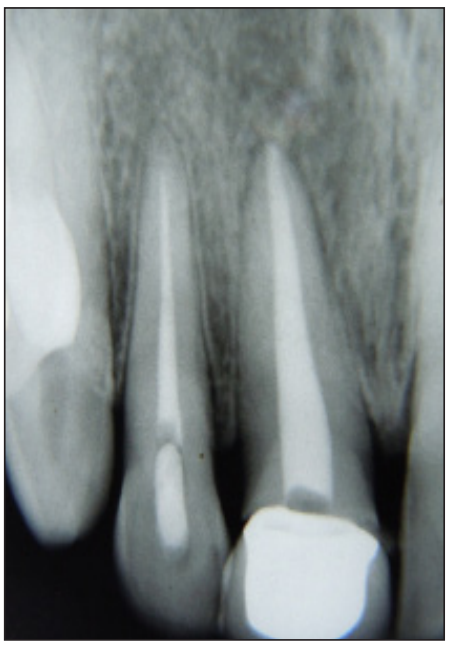

Figure 6. Postobturative radiograph of the permanent maxillary right later incisor.

Teeth affected by DI often have various shape changes and may have the following characteristics: increased labiolingual or mesiodistal diameter, incisal notching in association with a labial groove, bifid cingulum,[61] barrel-shape,[40] peg or conical morphology and presence of an exaggerated palatal cingulum or talon cusp.[40,61] Dilated crown and root and the consequential rotated position have been reported by Holtzman and Lezion.[95] These present clinical reports are suggestive of variations in DI. Case 1 showed an anomalously bulbous coronal portion and small root (less the cementum portion) in a central incisor, and a single lateral incisor was involved in Case 3, whereas the maxillary left lateral incisor and right central incisors revealed type 2 DI in Case 2. As in Case 1, Oehlers type 3 invagination often allows the entry of irritants into the periapical area, resulting in pathosis. When communication between the invagination and the pulp space causes pulp pathosis, and in cases where no communication with the invagination exists, pulp pathosis by retrograde infection should be suspected.[96] Pulp necrosis of the involved tooth, in combination with the location of the malformed structure, has been reported to provoke severe periodontal destruc-tion in the area.[65]

As discussed earlier, because of the possible presence of other associated dental anomalies or malformations and a symmetrical pattern for DI, a thorough clinical and radiographic examination with emphasis on the contralateral tooth is advisable whenever DI has been identified on one side.[61] Additionally, the presence of an invagination should be suspected when the tooth shows a history of signs and symptoms traditionally associated with pulpal disease. In the absence of the obvious cause (eg, trauma, caries, etc) and in case of an obscure invagination, application of methylene blue dye to the tooth's palatal portion may be useful in detection of DI. Application of radiopaque markers also helps illustrate the extent and shape of the invagination.[61] Pandey and Pandey[20] used forcibly injected radiopaque dye and radiovisiograph imaging to analyze the morphology of radicular DI in an extracted maxillary right first premolar and to establish the communicability of all 3 canals with a large sigmoid pulp chamber in the center.

Eccentric radiographs using Clark's same lingual, opposite buccal (SLOB) rule may be advisable for knowing the exact extent and identifying the associated anomalous conditions.[59] Radiographically, invagination varies from a slender and undilated fissure to a classical pear shape, as in Cases 2 and 3, or a teardrop-shaped loop pointing toward the pulp's main body. It may appear as a radiolucent pocket surrounded by a radiopaque

enamel border.[61] In some, however, it may be completely separate from the pulp and manifest as a deep enamel-lined fissure with its own opening into a periodontal ligament which has been described as a pseudocanal.[61,97]

Other manifestations may include the blunting of the pulp outline and associated atypical butterfly shaped apical radiolucency or a blunderbuss opening of the invagination into the periodontal ligament in Oehlers type 3 DI.[61] A foramen cecum may be visible as a radiolucency on the crown.[97] Arrested root development may be apparent on the radiograph over time if tooth vitality is lost soon after eruption.[61] Owing to its 2-dimensional basis, Oehlers' system may underestimate the true extent and complexity of the invagination[14]; in this respect, a radiographically appearing type 2 DI might actually be a type 3.[40] Also, overlapping cervical vertebrae make DI difficult to diagnose on rotational panoramic radiographs, especially in their minor forms.[68] These limitations associated with the use of conventional radiography in the classification and management of dens invaginatus may be overcome using a CT scan[14,98,99]; consequently, 3-dimensional reconstruction of the affected tooth is possible.[100]

Reuben et al.,[101] concluded that spiral CT not only helps in understanding the root canal system, but it can also give an accurate measurement for the morphology of the root canal space.[102] Its usage in practice may be impractical, however, due to associated setup costs. In our case, patients also were reluctant to bear the "added-cost" when comparing it's "cost-therapeutic benefit ratio" as a diagnostic aid. Sindeaux et al.[103] argued that, inmost cases, plain radiographs give enough information notonly in the initial assessment of dental anomalies, but also for after-treatment follow-up. Such circumstances are particularly relevant in developing countries where other imaging modalities for diagnosis are not always available. Another important point by Bayrak et al.[102] was that, while CT offers more useful information for diagnosis and treatment planning than conventional radiographs, it also involves high levels of radiation exposure. In cases where a cone-beam CT system is unavailable, the risk-benefit ratio of a conventional CT must be carefully considered on a case-by-case basis; hence, CT should be limited to cases where conventional radiography poorly depicts the actual status.

A thorough preoperative evaluation of the severity and complexity of the invagination, as well as the importance of the tooth, may influence the treatment decisions of teeth with dens invaginatus.[100] Thus, management may include conservative procedures such as root canal treatment or extraction, followed by intentional reimplantation or implant replacement therapy.[104]

Fissure sealing and composite or amalgam before carious destruction occurs, along with strict periodic observation, is recommended in teeth with deep palatal or incisal invaginations. No treatment is required in the absence of any detected entrance to the invagination and when visible clinical and radiographic signs of pathosis are missing. Root invagination treatment or root canal treatment of only the invagination was first recommended by Grossman[105] and Creaven[106]; prior to that, extraction was the only preferred treatment. Root canal treatment of the invaginaton is indicated in cases where the invagination has a separate apical or lateral foramen. In certain cases, invaginations must be treated as a separate root canal, whereas sometimes cutting through the invagination to access the apical foramen is required.[1] Greenfeld and Cambruzzi[107] and Ikeda et al.,[108] reported successful conservative root canal treatment of the invaginated canal withouttreatment of the main canal. Complete removal of the invagination to eliminate all communicating channels between the invaginated cavity and the pulp space has been reported by Verma et al.[109] and Jaramillo et al.[110] A surgical operating microscope may reveal an aberrant canal, which emphasizes its benefit during the treatment of a DI.[111-113]

Shaping and cleaning may be difficult because of the complex morphology associated with invaginations, including the large and irregular volume of the root canal system. NiTi instrumentation with greater flexibility, along with ultrasonic cleaning of the pulp space, may be a proficient means of disinfection.[1,114] Warm gutta-percha techniques (vertical condensation[109] or thermoplastic filling techniques) have been recommended for obturation of such teeth, as they compact the softened material into major irregularities within the root canal system.[1,109,115-117] Because the DI was not very large in Case 3, its removal did not enlarge the pulp canal significantly. Thus, the canal space was obturated satisfactorily using the lateral gutta percha condensation technique. There is a dilemma of unpredictable long-term prognosis of conventional root canal treatment in teeth with DI. Obstruction by the invagination often occurs because of inaccessibility, compromised debridement, and partially obturated sealer filled canal space beneath the invagination.[95]

In cases of endodontic failures after nonsurgical treatments because of bizarre anatomy and failure to access the root canal system completely, surgical treatment, including periapical debridement and/or root-end filling using mineral trioxide aggregate (MTA, DENTSPLY Tulsa Dental, Tulsa, OK), intermediate restorative material (IRM, DENTSPLY Caulk, Milford, DE), and ethoxy benzoic acid (Super EBA Harry J. Bosworth Co., Skokie, Ill., USA), should be considered. [97,109,118] Additionally, intentional replantation for otherwise hopeless cases has been proposed.[1] In cases involving endodontic and periodontal lesions, where there is a of lack of buccal cortical plate in the area, the best healing results may be obtained with the use of a barrier membrane technique during surgical management to prevent the invsion into the bony defect of competing nonosteogenic cells from the overlying soft

tissues.[65] Regardless of the size of a periradicular lesion, surgical treatment should be performed only when nonsurgical treatment has failed or is not permissible.[96] As suggested by Pai et al.[119] and Steffen and Splieth,[120] the sixe of the periradicular lesion's size should not dictate the treatment procedure.

It can be concluded that DI is undoubtedly an endodontic challenge. Early detection and management are recommended before the development of pulpal and periodontal pathosis that further complicates the prognosis. Postoperative follow-up subsequent to treatment, however, is essential for effective long-term clinical management, and a comprehensive multidisciplinary treatment approach should definitely be used while managing severe DI cases.

REFERENCES

1. Hülsmann M. Dens invaginatus: Aetiology, classification, prevalence, diagnosis, and treatment considerations. Int Endod J 1997;30:79-90.
2. Schulze C. Development abnormalities of the teeth and the jaws. In. Gorlin O, Goldman H, eds. *Thomas' Oral Pathology*. St. Louis, Mo: Mosby; 1970:96-183.
3. Marwah N, Goenka P, Nigam AG Prathima GS. Combined surgical and non-surgical management of dens invaginatus. Type 3: A case report. Int J Clin Pediatr Dent 2009;2:43-7.
4. Westphal A. A rare item on the 1st "dens in dente." Zahnarztl Mitt 1965;55:1066-70.
5. Schaefer H. Clinical aspects of the dens in dente. Dtsch Zahnarztl Z 1955;10:988-95.
6. Tomes JA. A System of Dental Surgery. London, UK. Lindsey and Blackstron; 1859:266.
7. Swanson WF, McCarthy FM. Bilateral dens in dente. J Dent Res 1947;26:167-71.
8. Thomas JG. A study of dens in dente. Oral Surg Oral Med Oral Pathol 1974;38:653-5.
9. Hunter HA. Dilated composite odontome: Report of two cases, one bilateral and one radicular. Oral Surg Oral Med Oral Pathol 1951;4:668-73.
10. Colby RA. Color Atlas of Oral Pathology. Philadelphia, Pa: JB Lippincott Co; 1956:34-5.
11. Arslan A, Göker K, Olgaç V, "Dens invaginatus: A case report." Oral Health and Dental Management in the Black Sea Countries 2004;3:52-5.
12. Anegundi RT, Kaveri H, Patil SB, Punnya A. Double dens invaginatus in an impacted molariform supernumerary tooth: An unique case. J Indian Pedod Prev Dent 2008;26:S26-8.
13. Goncalves A, Goncalves M, Oliveria DP, Goncalves N. Dens invaginatus type III: Report of a case and 10-year radiographic follow-up. Int Endod J 2002; 35:873-9.
14. Alani A, Bishop K. Dens invaginatus. Part 1: Classification, prevalence, and etiology. Int Endod J 2008;41:1123-36.
15. Holan G. Dens invaginatus in a primary canine: A case report. Int J Paediatr Dent 1998;8:61-4.
16. Rajendran R. Developmental disturbances of oral and paraoral structures. In: Shafer's Textbook of Oral Pathology. Rajendran R, Shivpathasundram B, eds. 5th ed. New Delhi, India: Elsevier; 2007:57-9.
17. Oehlers FA. A case of multiple supernumerary teeth. Br Dent J 1951;90:211-2.
18. Bhatt AP, Dholakia HM. Radicular variety of double dens invaginatus. Oral Surg Oral Med Oral Pathol 1975;39:284-7.
19. Oehlers FA. The radicular variety of dens invaginatus. Oral Surg Oral Med Oral Pathol 1958;11: 1251-60.
20. Pandey SC, Pandey RK. Radicular dens invaginatus: A case report. J Indian Soc Pedod Prev Dent 2005;23:151-2.
21. Payne M, Craig GT. A radicular dens invaginatus. Br Dent J 1990;169:94-5.
22. Soames JV, Kuyebi TA. A radicular dens invaginatus. Br Dent J 1982;152:308-9.
23. Ulmansky M, Hermel J. Double dens in dente in a single tooth: Report of a case and radiologic study of the incidence of small dens in dente. Oral Surg Oral Med Oral Pathol 1964;17:92-7.
24. Vincent-Townend J. Dens invaginatus. J Dent 1974;2:234-8.
25. Kronfeld R. Dens in dente. J Dent Res 1934;14: 49-66.
26. Fischer CH. Zur Frage des Dens in dente. Deutsche Zahn 1936;3:621-34.
27. Sprawson EC. Odontomes. Br Dent J 1937;62: 177-201.
28. Rushton MA. A collection of dilated composite odontomas. Br Dent J 1937;63:65-85.
29. Euler H. Die Anomalien, Fehlbildungen, und Verstummelungen der menschlichen Zahne. Munchen, Germany: Lehmann; 1939:62-7.
30. Atkinson SR. The permanent maxillary lateral incisor. Am J Orthod 1943;29:685-98.
31. Bruszt P. Etiology of dens in dente. SSO Schweiz Monatsschr Zahnheilkd 1950;60:534-42.
32. Casamassimo PS, Nowak AJ, Ettinger RL, Schlenker DJ. An unusual triad: Microdontia, taurodontia, and dens invaginatus. Oral Surg Oral Med Oral Pathol 1978;45:107-12.
33. Gustafson G, Sundberg S. Dens in dente. Br Dent J 1950;88: 83-88, 111-122, 144-6.
34. Oehlers FA. Dens invaginatus. I. Variations of the invagination process and associated anterior crown forms. Oral Surg Oral Med Oral Pathol 1957;10: 1204-18.
35. Oehlers FA. Dens invaginatus. II. Associated posterior crown forms and pathogenesis. Oral Surg Oral Med Oral Pathol 1957;10:1302-16.

36. Grahnen H, Lindahl B, Omnell K. Dens invaginatus. 1. A clinical, roentgenological, and genetically study of permanent maxillary lateral incisors. Odontologisk Revy 1959;10:115-37.
37. Rantanen AV. Talon cusp. Oral Surg Oral Med Oral Pathol 1971;32:398-400.
38. Ireland EJ, Black JP, Scures CC. Short roots, taurodontia, and multiple dens invaginatus. J Pedod 1987;11:164-75.
39. Jimenez-Rubio A, Segura JJ, Jimenez-Planas A, Llamas R. Multiple dens invaginatus affecting maxillary lateral incisors and a supernumeracy tooth. Endod Dental Traumatol 1997;13:196-8.
40. Kirzioglu Z, Ceyhan D. The prevalence of anterior teeth with dens invaginatus in the western Mediterranean region of Turkey. Int Endod J 2009; 42:727-34.
41. Burzynski NJ. Gemination and dens in dente. Oral Surg Oral Med Oral Pathol 1973;36:760-71.
42. Nazif MM, Laughlin DF. Dens invaginatus in a geminated central incisor: Case report. Pediatr Dent 1990;12:250-2.
43. Ekman-Westborg B, Julin P. Multiple anomalies in dental morphology: Macrodontia, multituberculism, central cusps, and pulp invaginations. Report of a case. Oral Surg Oral Med Oral Pathol 1974;38: 217-22.
44. Mann RW, Dahlberg AA, Stewart TD. Anomalous morphologic formation of primary and permanent teeth in a 5-year-old 15th century child: A variant of the Ekman-Westborg-Julin syndrome. Oral Surg Oral Med Oral Pathol 1990;70:90-4.
45. Pokala P, Acs G. A constellation of dental anomalies in a chromosomal deletion syndrome (7q32): Case report. Pediatr Dent 1994;16:306-9.
46. Oncag A, Gunbay S, Parlar A. William's syndrome. J Clin Pediatr Dent 1995;19:301-4.
47. Hibbert S. A previously unreported association between Nance-Horan syndrome and spontaneous dental abscesses. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99:207-11.
48. Hosey MT, Bedi R. Multiple dens invaginatus in two brothers. Endod Dental Traumatol 1996;12: 44-7.
49. de Sousa SM, Tavano SM, Bramante CM. Unusual case of bilateral talon cusp associated with dens invaginatus. Int Endod J 1999;32:494-8.
50. Kettunen P, Laurikala J, Itaranta P, Vainio S, Itoh N, Thesleff I. Associations of FGF-3 and FGF-10 with signaling networks regulating tooth morphogenesis. Dev Dyn 2000;219:322-32.
51. Ohazama A, Courtney JM, Sharpe PT. Opg, Rank, and Rankl in tooth development: Coordination of odontogenesis and osteogenesis. J Dent Res 2004;83:241-4.
52. Dassule HR, Lewis P, Bei M, Maas R, McMahon AP. Sonic hedgehog regulates growth and morphogenesis of the tooth. Development 2000;127:4775-85.
53. Lorena SC, Oliveira DT, Odellt EW. Multiple dental anomalies in the maxillary incisor region. J Oral Sci 2003;45:47-50.
54. Mupparapu M, Singer SR, Goodchild JH. Dens evaginatus and dens invaginatus in a maxillary lateral incisor: Report of a rare occurrence and review of literature. Aust Dent J 2004;49:201-3.
55. Tiku A, Nadkarni UM, Damle SG. Management of two unusual cases of dens invaginatus and talon cusp associated with other dental anomalies. J Indian Soc Pedod Prev Dent 2004;22:128-33.
56. Anthonappa RP, Yiu CK, King NM. A novel combination of dens evaginatus and dens invaginatus in a single tooth: Review of the literature and a case report. J Clin Pediatr Dent 2008;32:239-42.
57. Suprabha BS. Premolarized double dens in dente in albinism: A case report. J Indian Soc Pedod Prev Dent 2005;23:156-8.
58. Desai RS, Vanaki SS, Puranik RS, Rashmi GS, Nidawani P. An unusual combination of idiopathic generalized short-root anomaly associated with microdontia, taurodontia, multiple dens invaginatus, obliterated pulp chambers and infected cyst: A case report. J Oral Pathol Med 2006;35:407-9.
59. Sannomiya EK, Asaumi J, Kishi K, Dalben Gda S. Rare associations of dens invaginatus and mesiodens. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104:e41-e44.
60. Sedano HO, Ocampo-Acosta F, Naranjo-Corona RI, Torres-Arellano ME. Multiple dens invaginatus, mulberry molar, and conical teeth. Case report and genetic considerations. Med Oral Patol Oral Cir Bucal 2009;14:e69-e72.
61. Bishop K, Alani A. Dens invaginatus. Part 2: Clinical and radiographic features and management options. Int Endod J 2008;41:1137-54.
62. Gorlin RJ, Cohen MM, Levin LS. Syndromes of the Head and Neck. 3rd ed. New York, NY: Oxford University Press; 1990:472-4.
63. Hallet GE. The incidence, nature, and clinical significance of palatal invagination in the maxillary incisors teeth. Proc R Soc Med 1953;46:491-9.
64. Schulze C, Brand E. Uber den dens invaginatus. Zahnaztliche Welt Reform 1972;81:569-73, 613-20, 653-60, 699-703.
65. Chaniotis AM, Tzanetakis GN, Kontakiotis EG, Tosios KI. Combined endodontic and surgical management of a mandibular lateral incisor with a rare type of dens invaginatus. J Endod 2008;34:1255-60.
66. Rotstein I, Stabholz A, Friedman S. Endodontic therapy for dens invaginatus in a maxillary second premolar. Oral Surg Oral Med Oral Pathol 1987; 63:237-40.

67. Thongudomporn U, Freer TJ. Prevalence of dental anomalies in orthodontic patients. Aust Dent J 1998;43:395-8.
68. Hülsmann M, Hengen G. Severe dens invaginatus malformation: Report of two cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82:456-8.
69. Poyton HG, Morgan GA. Dens in dente. Dent Radiogr Photogr 1966;39:27-33.
70. Boyne PJ. Dens in dente: Report of three cases. J Am Dent Assoc 1952;45:208-9.
71. Amos ER. Incidence of the small dens in dente. J Am Dent Assoc 1955;51:31-3.
72. Hovland EJ, Block RM. Nonrecognition and subsequent endodontic treatment of dens invaginatus. J Endod 1977;3:360-2.
73. Fujiki Y, Tamaki N, Kawahara K, Nabae M. Clinical and radiographic observations of dens invaginatus. J Dentomaxillofac Radiol 1974;3:343-8.
74. Miyoshi S, Fujiwara J, Nakata T, Yamamoto K, Deguchi K. Dens invaginatus in Japanese incisors. Jpn J Oral Biol 1971;13:539-44.
75. Ruprecht A, Batniji S, Sastry KA, el-Neweihi E. The incidence of dental invagination. J Pedod 1986; 10:265-72.
76. Ruprecht A, Sastry KA, Batniji S, Lambourne A. The clinical significance of dental invagination. J Pedod 1987;11:176-81.
77. Backman B, Wahlin YB. Variations in number and morphology of permanent teeth in 7-year-old Swedish children. Int J Paedatr Dent 2001;11:11-7.
78. Hamasha AA, Al-Omari QD. Prevalence of dens invaginatus in Jordanian adults. Int Endod J 2004;37:307-10.
79. Ezoddini AF, Sheikhha MH, Ahmadi H. Prevalence of dental developmental anomalies: A radiographic study. Community Dent Health 2007;24:140-4.
80. Coklin WW. Bilateral dens invaginatus in the mandibular incisor region. Oral Surg Oral Med Oral Pathol 1978;45:905-8.
81. Olmez S, Uzamis M, Er N. Dens invaginatus of a mandibular central incisor: Surgical endodontic treatment. J Clin Pediatr Dent 1995;20:53-6.
82. Mupparapu M, Singer SR. A rare presentation of dens invaginatus in a mandibular lateral incisor occurring concurrently with bilateral maxillary dens invaginatus: Case report and review of literature. Aust Dent J 2004;49:90-3.
83. Khabbaz MG, Konstantaki MN, Sykaras SN. Dens invaginatus in a mandibular lateral incisor. Int Endod J 1995;28:303-5.
84. Goodman NJ, Stroud WE, Kuzma E. Dens in dente in a mandibular canine. Oral Surg Oral Med Oral Pathol 1976;41:267.
85. Banner H. Bilateral dens in dente in mandibular premolars. Oral Surg Oral Med Oral Pathol 1978;45:827-8.
86. Karaca I, Toller MO. Multiple bilateral dens in dente involving all the premolars. Case report. Aust Dent J 1992;37:449-52.
87. Bramante CM, de Sousa SM, Tavano SM. Dens invaginatus in a mandibular first premolar. Oral Surg Oral Med Oral Pathol 1993;76(3):389.
88. Tavano SM, de Sousa SM, Bramante CM. Dens invaginatus in first mandibular premolar. Endod Dent Traumatol 1994;10:27-9.
89. Hartup GR. Dens invaginatus type III in a mandibular premolar. Gen Dent 1997;45:584-7.
90. Kenezevic G. Dens invaginatus with ameloblastoma. Oral Surg Oral Med Oral Pathol 1980;49 (3):274.
91. Eden EK, Koca H, Sen BH. Dens invaginatus in a primary molar: report of case. J Dent Child 2002; 69:49-53.
92. Rabinowitch BZ. Dens in dente: Primary tooth. Report of a case. Oral Surg Oral Med Oral Pathol 1952;5:1312-4.
93. Kupietzky A. Detection of dens invaginatus in a one-year-old infant. Pediatr Dent 2000;22:148-50.
94. Chen YH, Tseng CC, Harn WM. Dens invagina tus: Review of formation and morphology with 2 case reports. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86:347-52.
95. Holtzman L, Lezion R. Endodontic treatment of maxillary canine with dens invaginatus and immature root. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82:452-5.
96. Lichota D, Lipski M, Woźniak K, Buczkowska-Radlińska J. Endodontic treatment of a maxil lary canine with type 3 dens invaginatus and large periradicular lesion: A case report. J Endod 2008;34:756-8.
97. Fregnani ER, Spinola LF, Sônego JR, Bueno CE, De Martin AS. Complex endodontic treatment of an immature type III dens invaginatus: A case report. Int Endod J 2008;41:913-9.
98. Vannier MW, Hildebolt CF, Conover G, Knapp RH, Yokoyama-Crothers N, Wang G. Three-dimen sional dental imaging by spiral CT: A progress report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84:561-70.
99. Sponchiado EC Jr, Ismail HA, Braga MR, de Carvalho FK, Simões CA. Maxillary central incisor with two root canals: A case report. J Endod 2006;32:1002-4.
100. Kristoffersen Ø, Nag OH, Fristad I. Dens invagina tus and treatment options based on a classification system: Report of a type II invagination. Int Endod J 2008;41:702-9.
101. Reuben J, Velmurugan N, Kandaswamy D. The evaluation of root canal morphology of the mandibu lar first molar in an Indian population using spiral computed tomography scan: An in vitro study. J Endod 2008;34:212-5.

102. Bayrak S, Dalci K, Sari S. Case report: Evaluation of supernumerary teeth with computerized tomog raphy. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100:e65-e69.
103. Sindeaux R, Leite AF, de Souza Figueiredo PT, et al. Proliferative periostitis associated with dens in dente. Brasilia Med 2009;46:295-9.
104. Altundal H, Kaptan F, Kayahan MB, Bayirli G. Se vere dens invaginatus in the maxillary central incisor. Oral Health and Dental Management in the Black Sea Countries 2003;4:45-8.
105. Grossman LI. Endodontic case reports. Den Clin North Am 1974;18:509-28.
106. Creaven J. Dens invaginatus-type malformation without pulpal involvement. J Endod 1975;1:79-80.
107. Greenfeld RS, Cambruzzi JV. Complexities of end odontic treatment of maxillary lateral incisors with anomalous root formation. Oral Surg Oral Med Oral Pathol 1986;62:82-8.
108. Ikeda H, Yoshioka T, Suda H. Importance of clinical examination and diagnosis: A case of dens invaginatus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79:88-91.
109. Verma P, Loomba K, Chandra A, Tikku AP, Wad hwani KK, Rhythm Surgical management of di lated gestant odontoma: A case report. J Indian Dent Assoc 2008;2:58-9.
110. Jaramillo A, Fernández R, Villa P. Endodontic treat ment of dens invaginatus: A 5-year follow-up. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101:e15-e21.
111. Girsch WJ, McClammy TV. Microscopic removal of dens invaginatus. J Endod 2002;28:336-9.
112. Jung M. Endodontic treatment of dens invaginatus type III with three root canals and open apical foramen. Int Endod J 2004;37:205-13.
113. Silberman A, Cohenca N, Simon JH. Anatomical redesign for the treatment of dens invaginatus type III with open apexes: A literature review and case presentation. J Am Dent Assoc 2006;137:180-5.
114. Gupta R, Tewari S. Nonsurgical management of two unusual cases of dens in dente. J Indian Soc Pedod Prev Dent 2005;23:190-2.
115. Budd CS, Weller RN, Kulild JC. A comparison of thermoplasticized injectable gutta-percha obturation techniques. J Endod 1991;17:260-4.
116. Gutmann JL. Adaptation of injected thermoplas ticized gutta-percha in the absence of the dentinal smear layer. Int Endod J 1993;26:87-92.
117. Tsurumachi T, Hayashi M, Takeichi O. Non-surgical root canal treatment of dens invaginatus type 2 in a maxillary lateral incisor. Int Endod J 2002;35:310-4.
118. Chong BS, Pitt Ford TR, Hudson MB. A prospec tive clinical study of mineral trioxide aggregate and IRM when used as root-end filling materials in endodontic surgery. Int Endod J 2003;36:520-6.
119. Pai SF, Yang SF, Lin LM. Nonsurgical endodontic treatment of dens invaginatus with large periradicu lar lesion: A case report. J Endod 2004;30:597-600.
120. Steffen H, Splieth C. Conventional treatment of dens invaginatus in maxillary lateral incisor with sinus tract: One year follow-up. J Endod 2005; 31:130-3.