DENS INVAGINATUS: AETIOLOGY, CLASSIFICATION, PREVALENCE, DIAGNOSIS AND TREATMENT CONSIDERATIONS

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SUMMARY

Dens invaginatus is a developmental malformation of teeth. Affected teeth show a deep infolding of enamel and dentine starting from the foramen caecum or even the tip of the cusps and which may extend deep into the root. Teeth most affected are maxillary lateral incisors.

The malformation shows a broad spectrum of morphologic variations and frequently results in early pulp necrosis. Root canal therapy may present severe problems because of complex anatomy of teeth. Aetiology, classification, prevalence, diagnosis and treatment considerations are reviewed.

Key words: Dens invaginatus (DI), malformation

INTRODUCTION

Dens invaginatus is a developmental malformation resulting from an invagination of enamel organ into the dental papilla, beginning at the crown and sometimes extending into the root before calcification occurs.^{1.4}This condition most commonly occurs in permanent maxillary lateral incisors, followed by maxillary central incisors,premolars,canines and less frequently in the molars.^{5.9} Cases of bilateraland multiple occurrence have also been reported.^{6,10} The presence of double dens invaginatus is extremely rare.² Involvement of deciduous teeth have also been reported.¹¹

This kind of malformation was first described by 'Ploquet' in 1794 in whale's tooth.³ DI was first describedas "a tooth within a tooth" by Salter in 1855.⁹DI in human tooth was first described by a dentist named Socrates in 1856.³

A number of different terms have been used to describe this condition; such as Busch in 1897 first suggested the use of 'dens in dente' which implies the radiographic appearance of "tooth within a tooth".¹² Hunter suggested the term "dilated composite odon-tome".¹³ Of the various terms 'dens invaginatus' would appear to be the most appropriate as it reflects the infolding of the outer portion (enamel) into the inner portion (dentin) with the formation of a pocket and dead space.¹

Synonyms for this malformation are: Dens in dente, invaginated odontome, dilated gestant odontome, dilated composite odontome, tooth inclusion and dens telescope.^{3, 4, 6}

Actiology of dens invaginatus

Over the last few decades, several theories have been proposed to explain the aetiology of this malformation but it is still unclear.

- Growth pressure of dental arch resulting in buckling of enamel organ.¹⁴
- Kronfeld suggested that it results from a focal failure of growth of internal enamel epithe-lium.¹⁵
- Rushton proposed that the invagination is a result of rapid and aggressive proliferation of a part of internal enamel epithelium invading the dental papilla.¹⁶
- Oehlers considered that distortion of the enamel organ during tooth development and subsequent protrusion of a part of the enamel organ will lead to

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the formation of an enamel-lined channel ending at the cingulum or occasionally at the incisal tip.¹⁷

- The 'twin-theorie' suggested a fusion of two toothgerms.¹⁸
- Infection was considered to be responsible for the malformation.¹⁹
- Gustafson and Sundbergdiscussed trauma as a causative factor.²⁰
- Genetic factor cannot be excluded.^{21,22}
- It may result from a deep infolding of foramen caecum during tooth development which in some cases may result in a second apical foramen.²³
- Ectomesenchymal signaling system between dental papilla and the internal enamel epithelium can affect tooth morphogenesis.²⁴ These signals have specific roles such as tooth morphogenesis and the folding of enamel organ.²⁵

Classification of dens invaginatus

The first classification of invaginated teeth was published by 'Hallet' in 1953.^{1, 26} Most commonly used classification was proposed by Oehlers in 1957 is shown in Figure 1.¹⁷ He described the anomaly occurring in three forms (coronal invaginations);

Type I: An enamel-lined minor form occurring within the confines of the crown not extending beyond the cemento-enamel junction.^{1,2}(Fig 1A)

Type II: An enamel-lined form which invades the root but remains confined as a blind sac. It may or may not communicate with the dental pulp.^{1,2} (Fig 1B)

Type III A: A form which penetrates through the root and communicates laterally with the periodontal ligament space through a pseudo-foramen. There is usually no communication with the pulp, which lies compressed within the root. ^{1, 2} (Fig1C) **TYPE III B**: A form which penetrates through the root and perforating at the apical area through a pseudoforamen. The invagination may be completely lined by enamel, but frequently cementum will be found lining the invagination.^{1,2}(Fig 1D)

In 1958, Oehlers also described the radicular form of invagination.²⁷ This type is rare and is thought to arise secondary to a proliferation of Hertwig's root sheath and radiographically, the affected tooth demonstrates an enlargement of the root.²⁸

In 1972, Schulze & Brand proposed a more detailed classification, including invaginations starting at the incisal edge or the top of the crown and also describing dysmorphic root configuration.²⁹ (Figure 2)

Prevalence of dens invaginatus

The prevalence of dens invaginatus ranges from 0.04%-10%.^{4,6} The prevalence studies are given in table 1. The teeth most affected are permanent maxillary lateral incisors and bilateral occurrence is not uncommon and occurs in 43% of all cases.^{3,7}

Cakici et al. reported that DI was detected in only maxillary lateral incisors with no gender difference and the most commonly observed was type I dens invaginatus (81.25%).⁴⁷In another study, most common type was also type I.⁴⁸

The permanent maxillary lateral incisor appears to be the most frequently affected tooth with posterior teeth less likely to be affected.⁴⁹Evidenc of DI in mandibular teeth have also been reported.^{6,50}Swanson & McCarthy explained about bilateral invagination.⁵¹Primary dentition involvement have also been reported in in different studies.^{52,53,54}

Histological findings

The dentin below the invagination may be intact without irregularities.^{55, 56}It may also contains strains







Fig 2:Classification of dens invaginatus by Schulze & Brand(1972)

of vital connective tissue or even fine canals with communication to the dental pulp. $^{\rm 14,\,57,58}$

The enamel was described as irregularly structured.^{14,59}Beynon reported hypomineralized enamel at the base of the invagination whereas Morfis, in a chemical analysis, detected up to eight times more phosphate and calcium compared with the outer enamel.^{59,60}Bloch-Zupanet al. described that internal enamel exhibited atypical and more complex rod shapes and its surface presented the typical honeycomb pattern but no perikymata,which, however, were observed on the outer surface of the tooth.⁶¹

Diagnosis

In most cases a DI is detected by chance on the radiograph.³Clinically, unusual crown morphology ('dilated', 'peg-shaped', 'barrel-shaped') or a deep foramen caecum may be important hints. Maxillary lateral incisors are the teeth most susceptible to coronal invaginations so these teeth should be investigated thoroughly clinically and radiographically, at least, in all cases with a deep pit at the foramen caecum at the lingual aspect of incisor teeth. However, in cases with peg shaped lateral incisors usually exhibit pit at the tip of the conical crown.^{3, 4, 62}

Normal conventional radiograph can not provide detailed structural information about this malformation. A latest radiographic technology, spiral computed tomography, have been introduced by Robinson et al and Sponchiado et al which is not only helpful in diagnosis of dens invaginatus but also provide3-dimensional image of variations in root canal anatomy.^{4,63}

Clinical features

The invagination allows entry of irritants into an area which is separated from pulpaltissue by only a thin

layer of enamel and dentine and presents a predisposition for the development of dental caries. In some cases, the enamel-lining is incomplete and channels may also exist between the invagination and the pulp.^{57,58} Therefore, pulp necrosis often occurs rather early, within a few years of eruption, sometimes even before root end closure.^{53,60}

Other reported sequelae of undiagnosed and untreated coronal invaginations are abscess formation, retention of neighboring teeth, displacement of teeth, cysts, internal resorption and facial cellulitis recently diagnosed.⁶⁵⁻⁷³

Clinical identification of the invagination entrance can be difficult as entrances can be unremarkable and be similar to normal fissures To aid in the identification process, the use of methylene blue dye can be utilized.⁷⁴ (Fig 3)

General features of teeth with DI include pegshaped formation, incisal notching, increased labiolingual and mesio-distal diameter, conical morphology and the presence of an enlarged palatal cingulum or cusp.^{3,74} (Fig 4(a,b)& 5)

The dental literature on dens invaginatus malformations contains several case reports presenting invaginated teeth coincident with other dental anomalies, malformations and even dental or medical syndromes. (Table 2)

Radiographic features

General radiographic features are;

Type I and Type II

In general both Types I and II DI begin coronally with a narrow undilated fissure. This then dilates into a uniglobular mass that either ends within the coronal

AUTHORS	YEAR	SAMPLE	FREQUENCY
Muhlreiter ³⁰	1873	500 maxillary lateral incisors	2.8%
Atkinson ¹⁴	1943	500 maxillary lateral incisors	10% of teeth
Boyne ³¹	1952	1000 maxillary incisors	8%
Shafer ³²	1953	$2542\mathrm{Full} ext{-mouth surveys}$	1.3% of patients
$\mathrm{Hallet}^{ 26}$	1953	586 Full-mouth surveys	6.6% of lateral incisor0.5% of central incisors
Amos ³³	1955	1000 Full-mouth surveys	5.1% of patients
Amos ³³	1955	203 Full-mouth surveys	6.9% of students of dentistry
Grahnen et al. ³⁴	1959	3020 right maxillary incisors	2.7% of patients
Ulmansky& Hermel ³⁵	1964	500 Full-mouth surveys	2% of patients
Poyton& Morgan ³⁶	1966	5000 Full-mouth surveys	0.25% of patient
Miyoshi et al. ³⁷	1971	Extracted maxillary lateral incisors	38.5% of teeth
Fujiki et al. ³⁸	1974	2126 Lateral maxillary incisors	4.2% of teeth
$ m Thomas^{39}$	1974	1886 Full-mouth survey	7.74% of patients
Gotoh et al. ⁴⁰	1979	766 Maxillary lateral incisors	9.66% of teeth
Ruprecht et al. ⁴¹	1986	1581 Full-mouth surveys	1.7% of patients
Ruprecht et al. 42	1987	300 Full-mouth surveys	10% of patients
Thongudomporn and Freer ⁴³	1998	111 Full-mouth surveys	26.1% of patients
Backman & Wahlin ⁴⁴	2001	739 Full-mouth surveys	6.8% of patients
Hamasha&Al-Omari 45	2004	1660 Full-mouth survey	2.95% of patients and $0.65%$ of teeth
Ezoddini et al.46	2007	480 Dental panoramic Tomographs	0.8%
Cakici et al.47	2010	1012 Full-mouth surveys	1.3%

TABLE 1: PREVALENCE STUDIES ON DENS INVAGINATUS

portion (Type I) or invades the radicular portion (Type II). As the invagination invades the coronal and radicular portion, the outline of the pulp space can change resulting in 'blunting' of the pulp horns. The defect may vary in size and shape from a loop like, pear-shaped or slightly radiolucent structure to a severe form resembling tooth within a tooth. The outline of the invagina-





Fig 3: Methylene blue dye applied under rubber dam isolation, revealing staining of the distal fissure. This tooth required root canal treatment secondary to loss of vitality due to an invagination

tion is generally well defined with an opaque layer of enamel. $^{1\cdot3,\,38,\,74}(Figures\,7\,\&\,8)$

Type III

Type IIIa presents as a deep fissuring of the tooth that exits on the lateral surface of the root. The root canal adjacent to the invagination may be undulating and abnormal. Bacterial ingress down the invagination





Fig 4 (a,b): Tooth having DI with conical crown Fig 5:maxillary lateral having DI enlarged cingulum



Fig 5: Maxillary lateral incisor having DI with enlarged cingulum

can result in a peri-invagination periodontitis. The nature of the peri-invagination is wide and present in a blunder-buss formation.^{1, 3,74}As a result of intricate communications between the invagination and the root canal; the latter will almost definitely lose vitality if the invagination is infected.^{74, 88}

In contrast, Type IIIb DI is more difficult to identify and fully locate, as it is superimposed on the root canal system exiting apically from within the root canal. This apical formation can present with an immature apex and in most cases presents with a well-established periapical lesion (Figure 8 & 9).⁷⁴

Management of dens invaginatus

The treatments options are;

- 1) Prophylactic or preventive sealing of the invagination 89
- 2) Root canal treatment^{6,7}
- 3) Endodontic apical surgery ^{90,91}
- 4) Intentional replantation ⁹²
- 5) Extraction^{93,94}



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Fig 6: Type I Dens invaginatus

Fig 7: Type II Dens invaginatus



Fig 8 & 9: Type IIIbDens invaginatus. Enamel lined fissure opening up into the apical portion, which has blunder-buss morphology.



Fig 10 A



Fig 10 B





Fig 10 C

Fig 10 D

- Fig 10A. The access opening prior to removal of the central hard-tissue core in type III demonstrating the main dens invaginatus(note alternate openings)
- B. Length determination and lateral canals
- C. Radiograph taken after the completeremoval of the invaginatus
- **D**. MTA plug was formed then themoplasticised gutta percha obturation done and coronal seal was done with composite resin

DENTALANOMALIES	AUTHORS	
Microdontia	Casamassimoet al. ⁷⁵ Desai et al. ⁷⁸	
Macrodontia	$Ekman-Westberg \& Julin.^{76}$	
Hypodontia	Hulsmann ⁷⁷	
Oligodontia	Conklin ⁴⁹ , Ruprecht et al. ⁴¹	
Taurodontism	${ m Casamassimo}{ m et}{ m al.}^{75}$	
Germination and Fusion	Ruprecht et al. ⁴¹	
Supernumerary teeth	$\mathrm{Petz}^{79}\mathrm{Beynon},^{59}\mathrm{Morfis}^{60}$	
Amelogenesisimperfect	Kerebel et al. 80	
Invagination in an odontome	Hitchin & McHugh ⁵⁸	
Multiple odontomes	Robbins & Keene ⁸¹	
Coronal agenesis	Hicks & Flaitz ⁸²	
William's syndrome	Oncaget al. ⁸³	
Mesiodens	Sannomiya et al. ⁸⁴	
Talon cusp	Tiku et al. ⁸⁵	
Densevaginatus	${ m Anthonappaetal.}^{ m 86}$	
Crouzon and Apert syndromes	Melero et al. ⁸⁷	

TABLE 2: DENTAL ANOMALIES ASSOCIATED WITH DENS INVAGINATUS

TypeI and TypeII

Once an invagination has been identified, base-line vitality test readings should be taken to get current pulpal status. Where pathological disease is absent the instigation of appropriate prophylactic measures should commence as soon as possible. There have been a wide variety of techniques advocated for prophylactic treatment. This has included the preparation of the invagination entrance and the placement of an amalgam restoration whilst more contemporary techniques have included the use of fissure sealant.^{74,88}

Where possible the use of magnification to open up the invagination entrance using a combination of fast hand piece burs and ultrasonic instruments should be considered to gain full access to the invagination.⁹⁴ Once the invagination is fully exposed, MTA can be used to obturate the invagination and access cavity will be sealed with composite The tooth should be regularly monitored for vitality loss. If this arises, appropriate root canal treatment should be instigated.⁷⁴

Antiseptic control is usually achieved by intracanal medicaments such as calcium hydroxide most commonly used but recently introduced triple antibiotic(ciprofloxacin,metronidazole,minocycline) paste has also been used and open apex problem is overcome by forming apical plug either using calcium hydroxide or by MTA(mineral trioxide aggregate).^{6,95,100} When simple endodontic therapy fails then combination of nonsurgical and surgical treatment could be performed.⁹⁰

Type III

In a vital tooth associated with periradicular inflammation, the invagination should be treated endodontically in the same way as anormal root canal to preserve the pulp vitality.⁹⁶A combined endodontic and surgical therapy in a vital tooth has also been reported.⁹¹ Pulp necrosis occurring in a tooth with the usual form of type III invagination is often successfully managed by endodontic therapy of root canal and the invagination alone, or by a combined endodontic and surgical therapy.^{97, 98} In some cases ultrasonic can be used for the complete removal of invagination to facilitate endodontic therapy.⁹⁴

As the morphology of these teeth is complex, if root canal treatment is not possible then extraction is usually recommended and prosthetic replacement is done.^{8,74}

The role of intentional replantation has also been reported in very complex forms of type III dens invaginatus. 99,73

The use of MTA in combination with gutta percha may be considered, where the post preparation apical portions are wide and blunder-buss in their formation.⁹ (Figures 10A, B, C & D)

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