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Dens Invaginatus (Tooth Within Tooth). A Review Of The Literature And Diagnostic And Management Guidelines For Practicing Dentists.

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Abstract
Dens invaginatus is a maldevelopment of the dental germ which occurs as a result of the invagination of the enamel organ. These cases may present difficulties with respect to its diagnosis and treatment because of canal morphology.It frequently leads to caries, pulpal and periodontal involvement with necrosis and loss of attachment. The knowledge of classification and anatomical variations of teeth with dens invaginatus are of great importance for correct treatment Due to the complexity of the malformation, treatment options in former days were limited. This article presents a profound review of the literature regarding etiology, epidemiology and histology. It discusses clinical appearance and diagnosis and it provides guidelines for decision-making and treatment of invaginated teeth.

Key Words
Dens invaginatus, aetiology,classification,diagnosis, management.

Introduction

Dens invaginatus is a developmental anomaly resulting from the invaginations of the enamel organ into the dental papilla during the soft tissue stage of development. As the hard tissues are formed, the invaginated enamel organ produces a small tooth within the future pulp chamber. This kind of tooth malformation was described first by Ploquet in 1794 who discovered this anomaly in a whale's tooth[1].

Dens invagination in a human tooth was first described by a dentist named 'Socrates' in 1856 [2]. Synonyms for this malformation are: Dens in dente, invaginated odontome, dilated gestant odontome, dilated gestant odontome, dilated composite odontome, tooth inclusion, dentoid in dente, gestant odontome, dents telescopes.

Aetiology

The aetiology of dens invaginatus malformation is controversial and remains unclear. Over the last decades several theories have been proposed to explain the aetiology of dental coronal invaginations:

- Kronfeld (1934) suggested that the invagination results from a focal failure of growth of the internal enamel epithelium while the surrounding normal epithelium continues to proliferateand engulf the static area[3].
- Rushton (1937) proposed that the invagination is a result of rapid and aggressive proliferation of a part of the internal enamel epithelium invading the dental papilla. He regarded this as benign neoplasma of limited growth[4].
- Oehlers (1957) considered that distortion of the enamel organ during tooth development and subsequent protrusion of a part of the enamel organ will lead to the formation of an enamel-lined channel ending at the cingulum or occasionally at the incisal tip. The latter might be associated with irregular crown form[5,6].
- Atkinson (1943) suggested that the problem was the result of external forces exerting an effect on the tooth germ during development[7]. Such forces could be from adjacent tooth germs, e.g. the central incisor or canine which develop at least 6 months prior to the lateral incisor[8] whilst other external factors such as trauma[9] and infection[10] have also been suggested as a cause.
- During tooth development the ectomesenchymal signalling systems that occur between the dental papilla and the internal enamel epithelium affect tooth morphogenesis[11]. These signals have specific roles such as the regulation of growth and the folding of the enamel organ[12]. The absence of certain molecules can result in abnormally shaped teeth as well as defects in the developing tooth germ[13]. For this reason the proposal that genetic factors may be the cause of dens invaginatus has some credibility[14-16]. Support for this possible cause also comes from a reported case of an individual lacking chromosome 7q32 who presented with dens invaginatus in addition to other dental abnormalities such as hypodontia[17]. There is further support from a clinical study of 3020 Swedish children that reported 2.7% of patients with dens invaginatus, 43%

of their parents and 32% of siblings also had evidence of the condition[14].

Classification

The first documented attempt to classify dens invaginatus was by Hallet[18] (1953) who suggested the existence of four types of invagination based on both clinical and radiographic criteria. Other classifications have also been described involving a variety of criteria and standards[19,20]. For example, Schulze & Brand[21] (1972) suggested an assessment based on twelve possible variations in clinical and radiographic appearance of the invagination. However, the system described by Oehlers[5] (1957a) appears to be the most widely used, possibly because of its simple nomenclature and ease of application. This system categorizes invaginations into three classes as determined by how far they extend radiographically from the crown into the root **(Figure 1)**.

Type I: The invagination is minimal and enamel-lined, it is confined within the crown of the tooth and does not extend beyond the level of the external amelo-cemental junction.

Type II: The invagination is enamel-lined and extends into the pulp chamber but remains within the root canal with no communication with the periodontal ligament.

Type IIIA: The invagination extends through the root and communicates laterally with the periodontal ligament space through a pseudo-foramen. There is usually no communication with the pulp, which lies compressed within the root.

Type IIIB: The invagination extends through the root and communicates with the periodontal ligament at the apical foramen. There is usually no communication with the pulp.

In Type III lesions, any infection within the invagination can lead to an inflammatory response within the periodontal tissues giving rise to a 'peri-invagination periodontitis'.

The limitations associated with the use of conventional radiography in the classification and management of dens invaginatus may be overcome in the future with the increasing availability of computerized 3D imaging[22,23]. Currently, such clinical techniques do not provide images of sufficient quality to fully evaluate the morphology of an invagination in situ although for extracted teeth sufficient detail can be obtained.

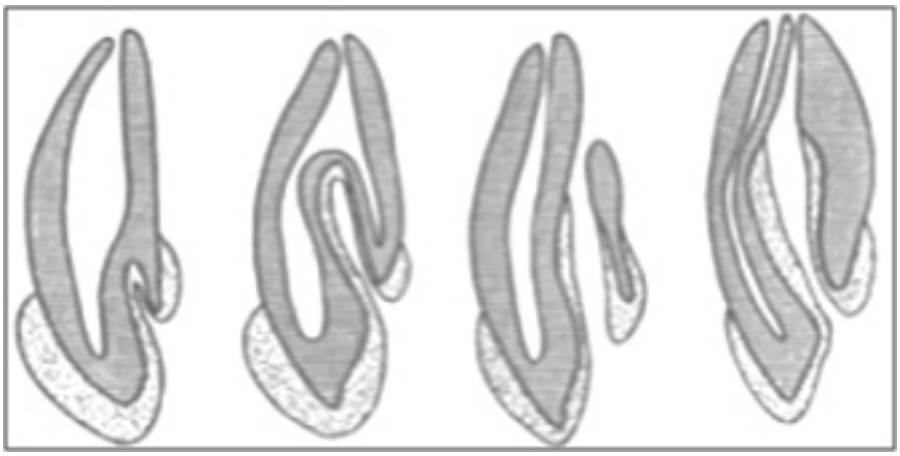


Figure 1: Oehler's classification of dens invaginatus (Coronal types)

Prevalence and Distribution

The reported prevalence of adult teeth affected with dens invaginatus is between 0.3% and 10% with the problem observed in 0.25% to 26.1% of individuals examined **(Table 1)**. The wide variation in reported prevalence may be explained by the different cohorts studied, identification criteria used and diagnostic difficulties However, more recent studies[24,25] have utilized a recognized classification system.

The permanent maxillary lateral incisor appears to be the most frequently affected tooth[26] with posterior teeth less likely to be affected[27,28]. This is supported by Hamasha & Al-Omari[29] (2004). A number of case reports have identified dens invaginatus in mandibular teeth[30-34].There is also some evidence that the problem may be symmetrical[7,14,24]. However, conflicting opinions exist with Swanson & McCarthy[35] (1947) . There have also been case reports of dens invaginatus occurring in the primary dentition[36-39].

However, all the documented case reports are of males which, if a true reflection, contrasts to the permanent dentition where females appear to be

Table 1: Prevalence Studies On Dens Invaginatus

Muhlreiter 41	1873	500 maxillary lateral incisors	2.80%
Atkinson 7	1943	500 maxillary lateral incisors	10% of teeth
Boyne 42	1952	1000 maxillary incisors	8%
Shafer 43	1953	2542 Full-mouth surveys	1.3% of patients
Hallet 18	1953	586 Full-mouth surveys	6.6% of lateral incisor0.5% of central incisors
Amos 44	1955	1000 Full-mouth surveys	5.1% of patients
Amos44	1955	203 Full-mouth surveys	6.9% of students ofdentistry
Grahnen et al14	1959	3020 right maxillary incisors	2.7% of patients
Ulmansky & Hermel 19	1964	500 Full-mouth surveys	2% of patients
Poyton & Morgan 45	1966	5000 Full-mouth surveys	0.25% of patient
Miyoshi et al. 46	1971	Extracted maxillary lateral incisors	38.5% of teeth
Fujiki et al 47	1974	2126 Lateral maxillary incisors	4.2% of teeth
Thomas 48	1974	1886 Full-mouth survey	7.74% of patients
Gotoh et al49	1979	766 Maxillary lateral incisors	9.66% of teeth
Ruprecht et al 50	1986	1581 Full-mouth surveys	1.7% of patients
Ruprecht et al 51	1987	300 Full-mouth surveys	10% of patients
Thongudomporn and Freer 52	1998	111 Full-mouth surveys	26.1% of patients
Backman& Wahlin 53	2001	739 Full-mouth surveys	6.8% of patients
Hamasha& Al-Omari 29	2004	1660 Full-mouth survey	2.95% of patients and 0.65% of teeth
Ezoddini et al. 54	2007	480 Dental panoramic Tomograph	0.80%
Cakici et al. 55	2010	1012 Full-mouth surveys	1.30%

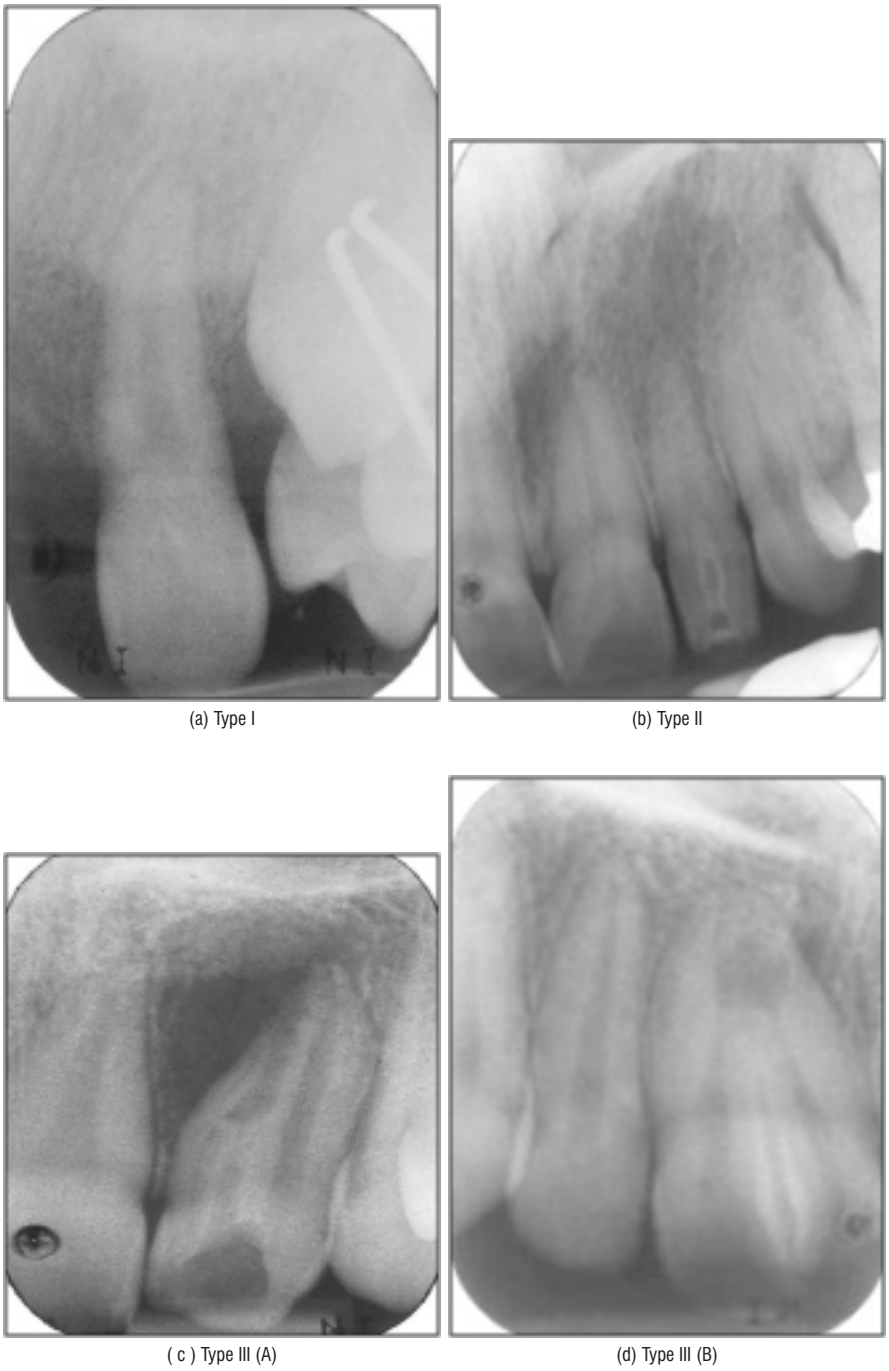
more at risk[40] or there is no gender difference reported[24]. The dental literature on dens invaginatus malformations contains several case reports presenting invaginated teeth coincident with other dental anomalies, malformations and even dental or medical syndromes.[26] **(Table 1)**

Nature of Invagination

The invagination may be associated with changes in the morphology of the root canal itself. One study that examined an extracted root filled invaginated tooth observed that the root canal was irregular in cross-section, with wave-like constrictions and dilatations[56]. There have also been reports of multiple root canals being present in association with the invagination[57-59]. Investigations into the histological, microscopically and radiographical nature of dens invaginatus have provided conflicting results. In some studies, the invaginated surface has been described as being uniform and regular with no communication with the pulp[60-63]. In contrast, others have described interruptions in the invaginated surface which could potentially act as a portal for irritants to the pulp [3,5,9,61,63,64]. Beynon [65] reported hypomineralized enamel at the base of the invagination where as Morfis [66] in a chemical analysis, detected up to eight times more phosphate and calcium compared with the outer enamel, but in his analysis magnesium was missing completely. Bloch -Zupan et al[67] found differences in structure and composition between the external and internal enamel. The internal enamel exhibited a typical and more complex rod shapes and its surface presented the typical honey comb pattern but no perikymata which however was observed on the outer surface of tooth.The structure of the dentine surrounding the invagination has also been reported as being irregular with connective tissue inclusions and communications towards the pulp[7,60,62,65]. The variation in findings of the nature of the invagination can possibly be explained by the different techniques used in each study, namely, histological[62] chemical analyses[66], SEM[67,68] and microradiographic[65] and micro X-ray diffraction[60] techniques employed.

Diagnosis of Dens Invaginatus

In most cases a dens invaginatus is detected by chance on the radiograph



(a) Type I (b) Type II

(c) Type III (A) (d) Type III (B)

Figure 2: Radiographic appearance of different types of dens invaginatus (a) Type I DI, Note the deep fissuring pointing towards the pulp. (b) Type II DI, Note the tearshaped ribbon-like formation of the invagination cavity. (c) Type III (A) DI, Note the invagination opening into the periodontal ligament creating an apical radiolucency. Type III (B) DI, Note the invagination opening into the apical portion, which has blunder-buss morphology

(Figure 2). Clinically, unusual crown morphology ('dilated', 'peg-shaped', 'barrel-shaped') or a deep foramen coecum may be important hints, but affected teeth also may show no clinical signs of the malformation. As pulpal involvement of teeth with coronal invaginations may occur a short time after tooth eruption early diagnosis is mandatory to instigate preventive treatment.

The invagination allows entry of irritants into an area which is separated from pulpal tissue 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. Channels may also exist between the invagination and the pulp[3,69]. Therefore, pulp necrosis often occurs rather early, within a few years of eruption, sometimes even before root end closure[19,35,70]. Other reported sequelae of undiagnosed and untreated coronal invaginations are abscess formation [71-73], retention of neighbouring teeth [74,75,] cysts [27,71] and internal resorption [76]

Normal conventional radiograph can not provide detailed structural information about this malformation.

A latest radiographic technology, spiral computed tomography, have been introduced by Reddy et al which is not only helpful in diagnosis of dens invaginatus but also provide3-dimensional image of variations in root canal anatomy. [77]

Management of dens invaginatus

The treatments options are;

1) Prophylactic or preventive sealing of the invagination [78,79]
2) Root canal treatment [34,80]
3) Endodontic apical surgery [81,82]
4) Intentional replantation [83]
5) Extraction [43,84]

Type I and Type II

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. [85,86] 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.[84] 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.[85]

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).[34,87,88] When simple endodontic therapy fails then combination of nonsurgical and surgical treatment could be performed.[81]

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.[89] A combined endodontic and surgical therapy in a vital tooth has also been reported.[82] 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.[90,91] In some cases ultrasonic can be used for the complete removal of invagination to facilitate endodontic therapy.[84]

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.[85,92] The role of intentional replantation has also been reported in very complex forms of type III dens invaginatus.[93,94] 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.[95]

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