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Case Report

Dens in dente: A review of literature with a rare case report

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ABSTRACT

Background: Dens invaginatus (DI) (dens in dente) is a developmental anomaly resulting from an invagination in part of variable depth of the enamel organ into the dental papilla in the surface of the crown before calcification of the dental tissues. They are a target for caries leading to pulp necrosis and apical pathosis, so the diagnosis and early prevention measures are important. When treatment is necessary, conservative, endodontic, and periodontal possibilities are very effective. Surgical interventions are only rarely indicated.

Case Presentation: This article presents a unique case report of unilateral DI of Type III (Ohlers) involving the maxillary left central incisor as well as a comprehensive review of literature on history, aetiology and classification.

Conclusion: 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 is essential for effective long-term clinical management.

1. Dens Invaginatus

Dens invaginatus (DI) (dens in dente) is a developmental anomaly resulting from an invagination in part of variable depth of the enamel organ into the dental papilla in the surface of the crown before calcification of the dental tissues. This anomaly usually involves the upper lateral incisors. These teeth are frequently expressed by an unusual form of crown and accentuation in the cingulum or a cusp. They are a target for caries leading to pulp necrosis and apical pathosis, so the diagnosis and early prevention measures are important. When treatment is necessary, conservative, endodontic, and periodontal possibilities are very effective. Surgical interventions are only rarely indicated. This article presents a unique case of unilateral DI of Tupe III (Ohlers) involving the maxillary left central incisor.

2. Background

Dens invaginatus or dens in dente, is a type of congenital maldevelopment which can occur to any dental component in oral cavity involving permanent, deciduous and supernumerary, with a greater prevelance in maxillary lateral incisors.[1] This congenital change has been given many designations by different authors such as odontoma invaginatus, dilated composite odontoma, tooth inclusion, dentoid in dente, gestant anomaly, tooth in tooth, and telescopic tooth.[2–4] Dens invaginatus is a common condition that can be easily neglected due to the absence of any substantial clinical indications of the defect. This is problematic because having an invagination increases the risk of caries, pulpal pathosis, and periodontal inflammation.[5]

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2.1. History

Dens Invaginatus was described by Ploquet in 1794 in a whale's tooth and was identified as such in a human tooth in 1856 by Socrates who documented his findings in Dentists Register.[2] It was Hallet, who first introduced the term dens invaginatus and gave the first classification of it.[6] This anomaly has been given different label at different period of time by different authors such as Busch first proposed the term "dens in dente," which refers to the radiographic appearance of a tooth within a tooth, in 1897.[7]

However, Hunter (1951) proposed the name "dilated composite odontome," which denotes an aberrant dilatation of the dental papilla,[8] whilst Colby (1956) suggested the word "gestant anomaly." Out of all the designations given dens invaginatus is the most appropriate one as it aptly describes the infolding of the outer portion (enamel) into the inner portion (dentine) with the formation of a pocket or dead area.[5]

2.2. Etiology

The anomaly is caused by infolding of the enamel organ into the dental papilla preceding the calcification of the dental tissue during morpho differentiation; later, as the hard tissues are forming, the infolded enamel organ produces a small denticle within the future pulp chamber.[5]

One of the earliest theories was given by Kronfeld in 1934 where he considered that the problem was caused by the slowing of a specific set of cells, while the surrounding cells continued to proliferate normally.[9] Rushton in 1937 on the other hand stated that the cause of anomaly was embryological with the stimulation and subsequent proliferation and ingrowth of cells of the enamel organ into the dental papilla during development.[10] However, Atkinson in 1943 proposed that DI was caused by external forces acting on the tooth germ during development.[11]

Ohler in 1975 contemplated that deformation of the enamel organ during tooth development, followed by protrusion of a portion of the enamel organ, would result in the creation of an enamel-lined tube that terminates at the cingulum or infrequently at the incisal tip. The latter could be linked to an uneven crown form.[12] Other factors responsible for invagination includes growth pressure from the dental arch, trauma, ectomesenchymal signalling system between dental papilla and the internal enamel epithelium, deep infolding of foramen caecum and genetic factors.[13]

2.3. Prevelance

The reported prevalence of adult teeth afflicted by dens invaginatus is between 0.3% and 10%, with the condition identified in 0.25% to 26.1% of individuals studied. The vast variance in reported prevalence may be explained by the diverse cohorts analysed, the identification criteria used, and the diagnostic challenges. The permanent maxillary lateral incisor appears to be the most frequently affected tooth with posterior teeth less likely to be affected and bilateral occurrence is not uncommon.[14]

2.4. Classification

Oehler provided the most frequently accepted and basic classification of DI, identifying three types of dens invaginatus based on the extent and coronal or radicular position of the invagination as follows:[12]

Class I: The invagination is limited and enamel-lined, restricted within the crown of the tooth, and does not extend beyond the external cementoenamel junction.

Class II: The invagination is enamel-lined and extends into the pulp chamber, but it remains inside the root canal and does not communicate with the periodontal ligament.

Class III a: The invagination continues into the root and interacts with the periodontal ligament area laterally via a pseudo-foramen. Normally, there is no communication with the pulp, which is compacted within the root.

Class III b: The invagination spreads through the root and interacts with the periodontal ligament through the apical foramen. Typically, there is no communication with the pulp.

In 1958 Oehlers also described radicular invagination. This type is uncommon and is assumed to be caused by a proliferation of Hertwig's root sheath, and radiographically, the affected tooth shows root expansion.

Early detection and accurate diagnosis of dens invaginatus are crucial for optimal management and preventing potential complications. Depending on the classification, treatment options range from simple filling of the invaginated enamel area to root canal therapy with or without retrograde surgery, purposeful re-implantation, or extraction of the damaged tooth.[5]

2.5. Clinical features

1. In a few cases, the enamel-lining is inadequate, and communication between the invagination and the pulp may exist.[9,10,12]
2. The invagination permits irritants to enter an area separated from pulpal tissue by only a thin layer of enamel and dentine, posing a risk for the development of dental caries.
3. As a result, pulp necrosis frequently develops within a few years of eruption, sometimes even before root end closure.[15,16] Other cases have documented misdiagnosed and untreated coronal invaginations causing abscesses, tooth retention and displacement, cysts, internal resorption, and face cellulitis.[17–20]
4. Peg-shaped development, incisal notching, increased labiolingual and mesiodistal diameter, conical morphology, and the presence of an expanded palatal cingulum or cusp are all characteristics of teeth with

dens invaginatus.[21]

5. In deciduous teeth, there have only been a limited number of well-documented case reports, one of which described an invagination located on the incisal edge of the tooth. This is in contrast to permanent incisors and other anterior teeth where the entrance to the defect invariably begins palatally.[21]

2.6. Radiographic features

The morphology of the invagination varies from a thin and undilated fissure to a tear-shaped loop heading towards the main body of the pulp.[22]

2.6.1. Type I and Type II

In general, both type I and type II initiates coronally as a thin fissure further dilating into uniglobular mass which is either restricted to coronal portion or descends into radicular portion, changing the pulpal outline and resulting in blunt pulp horns. The defect can range in size and shape from a loop-like, pear-shaped, or faintly radiolucent structure to a severe variant that looks like a tooth within a tooth.[2,5]

2.6.2. Type III

Type IIIa is identified by a deep fissure exiting from the lateral surface of the root. The root invagination is referred to as peri invagination, and it is wide and blunderbuss in nature, permitting bacterial entry and leading to peri invagination periodontitis.[2,5,21]

Type IIIb, on the other hand, is superimposed on the root canal system emerging apically from within the root canal. As a result, identifying and fully locating it is more challenging. It has an undeveloped apex and, in most cases, a well-established periapical lesion.[21]

2.7. Histologic Features

Investigations into the histological, microscopical and radiographical nature of dens invaginatus have provided conflicting results.[5] Kramer in 1953 emphasized the inadequacy of enamel in the invagination exposing the intact dentin leading to bacterial contamination of dentin tubules and pulp infection.[23] Beynon in 1982 observed that the enamel and dentine surrounding an invagination were hypomineralized.[24] In contrast to this study Morfis in 1993 conducted a SEM microanalysis in which he concluded that there was absence of magnesium, although there was an increase in phosphate and calcium ions in comparison to the normal coronal enamel present.[25] According to several authors the dentin surrounding the invagination was irregular with connective tissue and pulp connection.

Bloch-Zupan et al. reported that internal enamel had uncommon and more complex rod forms, and its surface had the typical honeycomb pattern but no perikymata, which were found on the tooth's exterior surface.[26]

Thus, presenting a unique case of unilateral DI of Tupe III (Ohlers) involving the maxillary left central incisor.

3. Case Presentation

A 12-year-old male patient reported the Department of Paediatric and Preventive dentistry of KD dental college and hospital with the chief complain of irregular teeth. The patient was healthy with no significant medical history and the past history of patient revealed that he has undergone trauma several years back as hit by a cow. The extraoral examination revealed no notable findings.

On detailed intraoral examination, there was discolouration of maxillary left central incisor and hyperplasia of gingiva on the palatal side. The tooth showed no tooth mobility and insensitivity to percussion and the findings of a pulp viability test on a tooth were also negative.

Initial panoramic radiographs examination was requested for evaluation, diagnosis, and treatment plan. Examination of a panoramic radiograph showed the presence of dens invaginatus of the maxillary left central incisor with an apical radiolucent area. A diagnosis of type III (Oehlers) dens invaginatus with periapical abscess was made. For further investigation a maxillary occlusal radiograph was advised which confirmed our diagnosis of dens in dente.

Endodontic treatment was planned for maxillary left central incisors After administration of local anaesthesia hyperplastic tissue on the palatal side was removed by electrocautery. The access cavity preparation was made using a diamond round burr and the invagination orifice was located, the primary root canal was discovered in a more buccal position. Invagination orifice was enlarged & radiograph with files in the root canal was obtained, working length were established and noted. It did not appear to be any communication between the primary root canal & the invagination. The canal system was debrided thoroughly using 2.5% of NaOCL followed by 2.5% saline & was prepared by step back technique to a size of 40. The root canals were dried with paper points & calcium hydroxide root canal medicament was placed in root canal and the tooth was temporarily sealed with GIC.

The patient was recalled after 7 days, he presented with symptoms of swelling & sinus followed by hematoma formation. So, the calcium hydroxide was removed from the canal, analgesic (Combiflam) for 7 days. Patient was recalled after 7 days. On patient 3^{rd} visit, the sinus formation was there and on IOPAR the teeth were evident with periapical radiolucency. All these leads to poor prognosis of the tooth. Hence it was concluded to extract the tooth.

Figure 1: Front profile of patient

Figure 2: Side profile of patient

Figure 3: Intraoral photograph depicting discoloured maxillary left incisor.

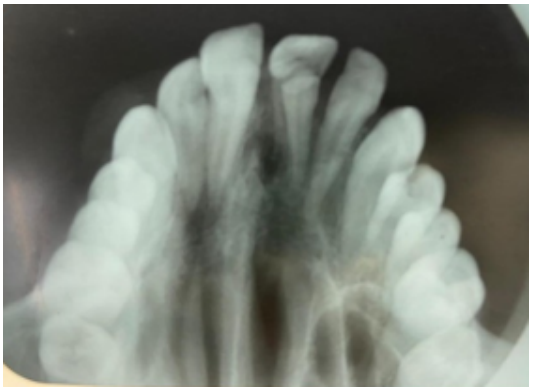

Figure 4: Intraoral photograph depicting discoloured maxillary left incisor.

Figure 5: OPG showing dens in dente

Figure 6: Occlusal radiograph showing Dens in dente

Figure 7: IOPAR in relation to 21

Figure 8: IOPAR depicting the working length determination.

Figure 9: IOPAR showing the canals filled with calcium hydroxide.

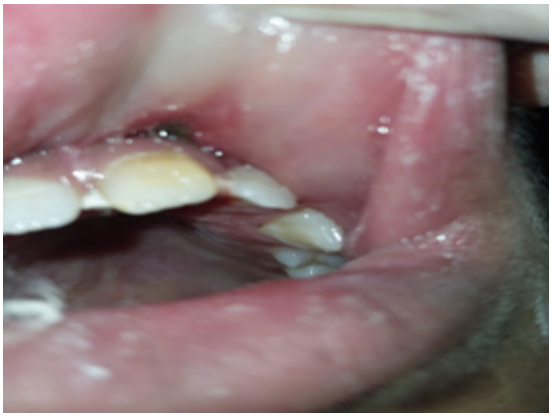

Figure 10: Hematoma seen in relation to maxillary left incisor

Figure 11: After the extraction of maxillary left incisor

Figure 12: Extracted maxillary left incisor.

4. Discussion

Initially, the teeth should be clinically evaluated, the pulp vitality measured, and a suitable treatment regime is advised based on the pulpal status, radiographic degree of root development, and the amount of the invagination. If the invagination is severe then the benefit of maintaining the tooth must be assessed while keeping the general condition of the mouth and potential future treatment needs in mind. For example, in young patient if the orthodontic treatment is required irrespective of e presence of invaginated tooth, extraction of the tooth will be more acceptable. However, if the goal is to keep an invaginated tooth, the following treatment options may be considered.[21]

4.1. Minimal invaginations (Oehlers' type I)

If the invagination is limited to the crown of the tooth and there is no sign of pulpal illness, preventative therapy should be initiated as soon as reliable moisture control can be accomplished. The entrance to invagination is sealed with Acid-etched fissure sealant or flowable composite resin material. The teeth should subsequently be checked on a regular basis for signs of pulp disease or degradation of the restoration.[27]

In cases of pulpal disease root canal treatment is included in the treatment regimen. A vital pulpotomy may be considered in cases where the inflammation is thought to be restricted, particularly in developing teeth. If the condition is severe, a more traditional method should be sought. The invagination should be adequately debrided and cleaned by incorporating it into access by extending the cavity. The use of gates gladden burs or ultrasonic alloy tips of appropriate size is beneficial in such cases followed by canal filling using thermoplastic gutta percha and final restoration using glass–ionomer or a flowable composite material overlaid with an acid-etched traditional composite.[21]

4.2. Moderate invagination (Oehlers' type II)

Prophylactic treatment is not of a much success in these cases as there is increased extent of invagination which may lead to void or dead space. Because it is impossible to rule out the possibility of pulpal communication even under magnification, it seems reasonable to treat all such invaginations as involving the pulp. If significant caries is present, it can be removed either using long necked round burs or ultrasonic tips or pulpotomy can be performed keeping in mind that Type II are wider and reach further apically than type I.[12] After preparing access cavity with tungsten carbide bur the lumen should be cleaned and irrigated with chlorhexidine or 1% Naocl prior to the invagination being filled and sealed. In view of properties of MTA, it appears that it is a material of choice in such scenarios. Following the removal of sufficient moisture, vertical compaction of MTA into the lumen can be accomplished with typical endodontic pluggers of appropriate size.[21]

4.3. Severe invagination (Oehlers' type III)

If there is no evidence of pulp disease in these more intricate and widespread lesions, simple prophylactic approach should be taken rather than aggressive one as it might lead to pulp exposure. In cases where peri invagination periodontitis is present the objective should be to treat the invagination in isolation to the root canal which is practically only possible in the cases of type IIIa since in type IIIb there is close proximation of invagination and root canal. Microscopy and methylene blue can help identify the entrance of invagination and aid in instrumentation followed by placing the gutta percha or file to ensure correct orientation. Because the surface is largely covered in enamel and has an irregular shape, ultrasonic tips are more useful in cleaning and shaping the invagination than stainless steel or nickel titanium endodontic instruments as they may fracture in the invagination.[21]

In cases where the pulp is necrotic, the canal and invagination may be treated individually. In practise, however, the proximity of the root canal to the invagination, as well as the complex and invariable relationships between them, makes it practically unavoidable that they will need to be joined. This can be accomplished by using ultrasonic instruments or long-shanked round burs on the root canal side of the split between the root canal and the invagination. The root canal and invagination should be kept distinct in the apical area since merging the canal in this location may result in apical constriction damage. Filling with gutta-percha is permissible once the preparation is complete. However, if the apical morphology of the root canal makes traditional filling impossible, MTA may be the chosen material. Once root canal treatment is completed, these teeth should be monitored in the same manner as other root canal-treated teeth.[21]

4.4. Necrotic pulps in immature teeth

The thin root walls and open apices associated with invaginated immature teeth with necrotic pulps complicate management of these teeth. As a result, even if early therapy is successful, the long-term viability of such teeth is regarded low. Treatment regimen for such teeth should be devised in accordance with the orthodontist to balance the occlusal requirements retaining the tooth as 'space maintainer' until the occlusion is fully established.[21]

If the tooth is retained, treatment plan indicated for type II and type III is appropriate, but with filling achieved using MTA in a similar method as described for non-invaginated teeth.[27]

The possible course of treatment ranges from preventive approach in minimal invagination to extraction in severe cases. Hence several authors have suggested extraction of the involved tooth as an option of treatment depending on the clinical status of the teeth. In this case the involved tooth was extracted since the endodontic treatment led to the complication of hematoma sinus and swelling making extraction a better treatment alternative. According to Wissal Kabbassi et al extraction can be the treatment of choice when the invaginated tooth causes an aesthetic or functional disturbance.[28] Third molars and mesiodens are examples of such situations. A similar treatment choice was made by Ulkem Aydin et al while treating the case of dens invaginatus due to mobility, excessive gingival recession and periapical inflammation.[29]

5. Conclusion

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.

6. List of Abbreviations

DI – Dens Invaginatus; SEM- Scanning Electron Microscopy; Naocl- Sodium hypochlorite; GIC- Glass Ionomer Cement; MTA- Mineral Trioxide Aggregate.

7. Source of Funding

None.

8. Conflict of Interest

None,

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