Dens Invaginatus: History, Etiology, Classification, Clinical Feature and Management

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Abstract: Dens invaginatus is a tooth malformation resulting from an infolding of the dental papilla at the time of tooth development. The deep infolding of enamel and dentin are seen in the affected tooth. Maxillary lateral incisors are commonly affected tooth and bilateral occurrence is rare. It shows a broad spectrum of morphologic variations and it usually results in pulpal necrosis. Root canal therapy may undergo severe problems due to its complex anatomy of teeth. This review addresses the etiology, clinical and radiographic features of taurodontism, its association with various anomalies, as well as important considerations in various areas of expertise dental treatments of such teeth.

Keywords: Malformation, Anomalies, Root canal therapy, Apical surgery

INTRODUCTION

Dens invaginatus is a malformation of developing tooth due to invaginations of the enamel organ into the dental papilla, involving the crown and sometimes extending into the root before calcification occurs [1]. During hard tissue development, the invaginated enamel organ produces a small tooth within the future pulp chamber. The affected teeth on radiograph show an infolding of enamel and dentine which may elongate deep into the pulp cavity and into the root and sometimes even reach the root apex [2]. Both crown and root structures may show variations in size and form. This condition frequently seen in permanent maxillary lateral incisors, followed by maxillary central incisors, premolars, canines and less commonly in the molars [3]. Cases of bilateral and multiple occurrences have also been reported. The presence of double dens invaginatus is unusual. Involvements of deciduous teeth have also been reported [3, 4].

Synonyms for this malformation are: Dens in dente, dentoid in dente, dilated gestant odontoma, invaginated odontome, deep foramen caecum, , dilated composite odontoma, tooth inclusion, gestant odontome, and dents telescopes [2-4].

HISTORY

It was first described by ‘Ploquet’ in 1794 in whale’s tooth.3 DI was first described as “a tooth within a tooth” by Salter in 1855. DI in human tooth was first described by a dentist named Socrates in1856. In 1873 Mühlreiter reported on ‘anomalous cavities in human teeth’. 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 imply the radiographic appearance of “tooth within a tooth”. Hunter suggested the term “dilated composite odontome”. 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 [5].

ETIOLOGY

Over the last few decades, several proposals for theories to explain about the etiology of dens invaginatus, but it is still unclear:

1. Kronfeld [6] suggested that the invagination results from a focal failure of growth of the internal enamel epithelium while the surrounding normal epithelium continues to proliferate and engulf the static area.

2. Infection was considered to be responsible for the malformation by Fischer in 1936 and Sprawson in 1937.

3. Rushton [7] 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 benign neoplasm of limited growth.
4. Oehler [8] 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. Growth Pressure of the dental arch results in buckling of the enamel organ [9].

6. The “twin-theorie” suggested a fusion of two tooth-germs [10].

7. Gustafson & Sundberg [11] discussed trauma as a causative factor, but could not sufficiently explain why just maxillary lateral incisors were affected and not central incisors.

8. Some author suggests that the ectomesenchymal signaling system between dental papilla and the internal enamel epithelium can affect tooth morphogenesis [12]. These signals have specific roles such as tooth morphogenesis and the folding of enamel organ [13].

9. Most authors say that it may result from a deep infolding of foramen caecum during tooth development which in some cases may result in a second apical foramen [14].

10. Genetic factor cannot be excluded [15].

CLASSIFICATION OF DENS INVAGINATUS

The first classification of invaginated teeth was published by ‘Hallet’ in 1953. Most commonly used classification was proposed by Oehlers in 1957 [8] shown in Fig: 1. 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, 16].

**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, 16].

**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, 16].

**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, 16].

In 1958, Oehler also described the radicular form of invagination. 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 [8].

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 [16].
CLINICAL FEATURES

Due to the deep invagination, which allows entry of irritants into an area which is separated from pulpal tissue by only a thin layer of enamel and dentine, it is more susceptible for the development of dental caries. In some cases, the enamel-lining is deficient and channels may also exist between the invagination and the pulp. Hence, pulp necrosis often occurs at early stages, within a few years of eruption, sometimes even before root end closure [17].

Other reported consequences of undiagnosed and untreated coronal invaginations are abscess formation, displacement of teeth, retention of neighboring teeth, cysts, internal resorption and facial cellulitis recently diagnosed [18]. Clinically, dens invagination entrance can be difficult to identify as it may be similar to normal fissures. Methylene blue dye can be used to detect the condition [17]. General features of teeth with Dens Invaginatus include peg shape, conical morphology, incisal notching, increased mesio-distal and labiolingual diameter, and the presence of an enlarged palatal cingulum or cusp [16].

The dental literature on dens invaginatus malformations contains several case reports presenting invaginated teeth coincident with other malformations, dental anomalies and even medical or dental syndromes [17] (Table 1).

Table 1: Dental Anomalies Associated With Dens Invaginatus [19, 20]

<table>
<thead>
<tr>
<th>S. No</th>
<th>Dental Anomalies</th>
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<td>1</td>
<td>Microdontia</td>
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<td>2</td>
<td>Macrodontia</td>
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<td>3</td>
<td>Hypodontia</td>
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<td>Oligodontia</td>
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<td>5</td>
<td>Taurodontism</td>
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<td>6</td>
<td>Germination And Fusion</td>
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<td>7</td>
<td>Supernumerary Teeth</td>
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<td>8</td>
<td>Amelogenesis Imperfect</td>
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<td>9</td>
<td>Invagination In An Odontome</td>
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<tr>
<td>10</td>
<td>Multiple Odontomes</td>
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<tr>
<td>11</td>
<td>Coronal Agenesis</td>
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<td>12</td>
<td>William’s Syndrome</td>
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<td>13</td>
<td>Mesiodens</td>
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<td>14</td>
<td>Talon Cusp</td>
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<tr>
<td>15</td>
<td>Dens Evaginatus</td>
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<tr>
<td>16</td>
<td>Crouzon And Apert Syndromes</td>
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RADIOGRAPHIC FEATURES

General radiographic features are;

Type I and Type II

In general both Types I and II DI initiates coronally with a narrow undilated fissure. This then dilates into a uniglobular mass that either limits itself within the coronal portion (Type I) or invades the radicular portion (Type II). As it invaginates the coronal and radicular portion, the pulpal outline may change resulting in ‘blunting’ of the pulp horns. The defect may differ in shape and size from a loop like, pear-shaped or slightly radiolucent structure to a severe type resembling tooth within a tooth. The outline of the invagination is generally well defined with an opaque layer of enamel [18, 21].

In contrast, Type IIIb DI is superimposed on the root canal system exiting apically from within the root canal. Therefore, it is more difficult to identify and fully locate. It exhibits an immature apex and in most cases presents with a well-established periapical lesion [18].

Histological findings

The dentin below the invagination may be intact and reliable. It contains strains of vital connective tissue or even fine canals with communication to the dental pulp. Irregularly structured enamel will be seen. 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. Bloch-Zupan et 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 [16, 19].

**MANAGEMENT**

The treatment options are as follows:
1. Preventive and restorative treatment
2. Root canal treatment
3. Surgical treatment
4. Extraction

**Preventive and restorative treatment**

Teeth with deep palatal or incisal invaginations or foramina coecae should be managed by application of adequate fissure sealant before a caries process is initiated. A composite restoration and a compulsory periodical review are recommended. If the entrance of the invagination is undetectable without any clinical or radiographical signs of pathosis, then no treatment is advised, but should be in observation of the condition [22].

**Root canal treatment**

When the invagination has a separate apical or lateral foramen, root canal treatment of the invagination is indicated. Due to large and irregular volume of the root canal system, convenient shaping and cleaning may be difficult [3]. Irrigation along with ultrasonic cleaning of the root canal system has been suggested to be an efficient means of disinfection and hence therefore been recommended for cleaning of the complex morphology of the root canal system in teeth with dens invaginatus [4]. Warm gutta-percha techniques including vertical condensation or thermoplastic filling techniques have been recommended for obturation of such teeth. When pulp necrosis occurs before root-end closure, apexification with calcium hydroxide may be necessary [3, 4].

**Surgical treatment**

Surgical treatment should be considered in cases of tooth:
- With endodontic failure
- Which cannot be treated non-surgically
- With anatomical problems
- With failure to gain access to all parts of the root canal system [23].

**Extraction**

Extraction is indicated only in:
- Teeth with severe anatomical irregularities that cannot be treated non-surgically or by apical surgery
- Supernumerary teeth
- Teeth with abnormal crown morphology present aesthetic or functional problems [23].

**CONCLUSION**

It occurs frequently with permanent teeth and it is a very rare developmental anomaly of deciduous dentition. In general, the treatment options were very limited to extraction but early diagnosis and intervention can help to prevent the pulp from necrosis and potential loss of tooth. With an eye to the future, the best treatment modalities available should be implemented soon. Keeping this in mind, innovative and better outcome techniques are only as far away as the imagination of the researcher.

**REFERENCES**


