Case Report

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Surgical Exposure of Turner's Tooth and CBCT Representation of Inverted Tooth: A case report

Abstract:

Hypoplasia, or enamel deficiencies pertaining to thickness and quality, are caused by disruptions in the enamel matrix growth process. The topographic relationship between the primary tooth and the developing permanent tooth germ explains the range of potential developmental disruptions that can occur, from minor changes in enamel mineralization to severe sequestration of the tooth germ, dilations of the crown or root, duplication of the crown or root, and complete or partial arrest of root formation. Turner's hypoplastic types might have bright, rough, or pitted enamel. It is incredibly rare for trauma, odontomas, and nearby supernumerary teeth to cause permanent central incisor impaction and inversion. When incisors are impacted or moved in the opposite direction, root development is inhibited. To determine whether enamel thickening and inversion are present or absent, dental radiographs and CBCT are utilized. Not only is this data useful for diagnosis, but it also helps create the most effective treatment strategy. Thus, a 9-year-old girl with maxillary lateral incisor inversion and Turner's hypoplasia is described.

Key-words: MeSH- Turner's Hypoplasia, Tooth inversion, CBCT, RVG

Introduction:

According to histomorphology, hypoplasia is characterized as an outward abnormality involving the enamel's surface that is associated with decreased enamel thickness. It appears to be a quantitative enamel defect .The main objective of this article is to describe the consequences of permanent teeth that arise from the traumatic injuries sustained by their predecessors, which cause hypomineralization and inappropriate development of the enamel matrix. These modifications might have occurred at the accident scene or as a consequence of post-traumatic stress disorder.[1]

The enamel and dentin serve as kymographs during the formative and calcifying stages of their maturation, permanently recording any physiologic or pathologic changes in the organism's metabolism. Ameloblasts that have been active for longer periods of time may be more susceptible to disruption. This could explain the widely observed increased percentage of anomalies found in larger teeth.[2]

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When Turner's hypoplasia is observed in the anterior portion of the mouth, it is most often the result of a primary tooth damage. When the damaged tooth—typically a maxillary central incisor—is driven into the tooth that is developing beneath it, it affects the enamel-forming process. The area most likely affected by the permanent tooth's growth bud's location in respect to the primary tooth is its facial surface.[3] Turner's hypoplasia may also be accompanied by a discoloration that is yellow or white.

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Case Report:

A 9yr old female patient came to the department of Pediatric& Preventive Dentistry, of Sri Aurobindo College of Dentistry, Indore, with a chief complaint of unerupted tooth in her upper front region since 1-2 years as the other central incisor was already erupted. Patient had a history of trauma at age of 1yr. On oral examination an eruption buldge in the region of 11 was seen (Fig 1). Unerupted teeth in relation to 12,22 and exfoliated 53 were observed. A RVG was taken to check the current status of the unerupted 11. The radiograph revealed an open apex and loss of enamel around cervical region in relation to 11,unerupted 12 with open apex and inverted tooth in relation to 22 also could be seen(Fig2) .

To confirm the enamel loss and inversion both, the patient was sent for CBCT. The CBCT showed the presence of enamel loss at cervical third of the labial aspect of the tooth, thus confirming the case of turner's hypoplasia (Fig 3).

The treatment plan was made to surgically expose the unerupted 11 and further is planned to be treated endodontically followed by orthodontic management. Patient's consent was taken. Bleeding time and clotting time of the patient was investigated. The surgical exposure was done by conventional method i.e scalpel using 15# blade (Fig 4). After surgical exposure patientwas given post operative instructions. The patient has been kept on follow up every month until the tooth erupts completely. After one month of follow up the hypoplastic area was restored by composite to prevent it from decalcification so as to preserve it's vitality (Fig 5). The tooth 11 was seen to be palatally inclined and confirms to beerupting in crossbite position in future, hence once the tooth will completely erupt, orthodontic management will be done along with intentional endodontic treatment (RCT).

The CBCTalso revealed an inverted tooth bud of 22 adjacent to 21 (fig 6). The inversion of the tooth bud is believed to be due to traumatic injury in the past. Treatment plan will include extraction of 22 and placement of implant at appropriate age.

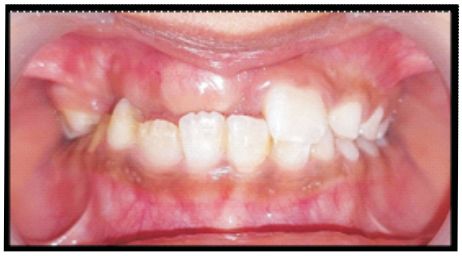

Fig1 Showing eruption buldge irt 11

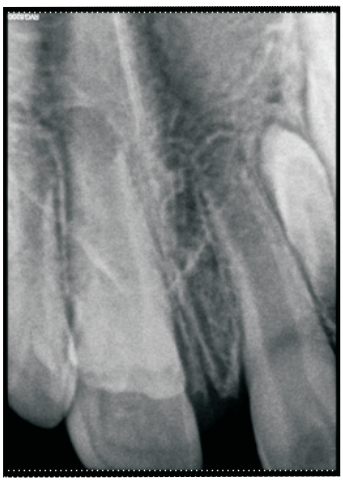

Fig 2 RVG irt 11 and 22

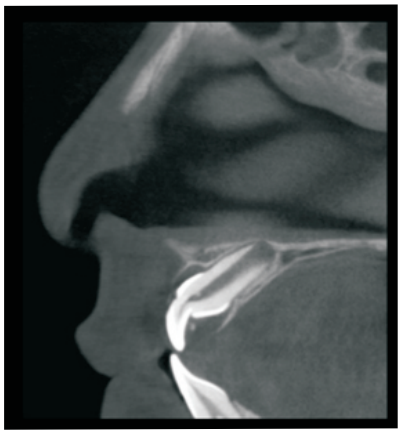

Fig 3 CBCT showing loss of enamel at cervical third of 11

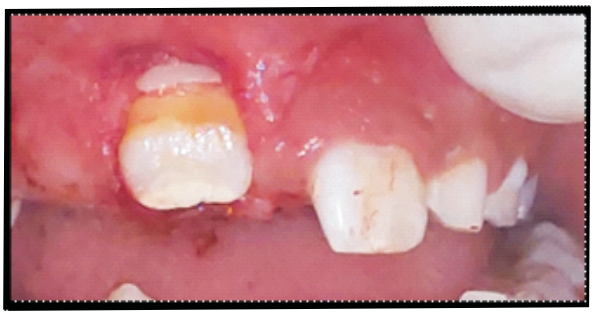

Fig 4 Surgical exposure of unerupted 11 showing hypoplastic region in cervical third

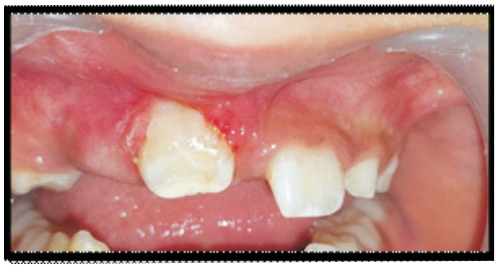

Fig 5 Showing restored hypoplastic area with composite

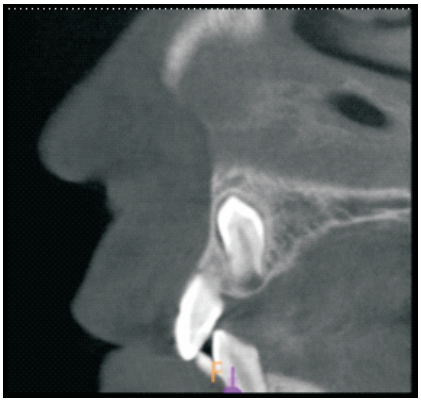

Fig 6 CBCT showing inverted tooth budirt 22

Discussion:

Damage to primary teeth might lead to issues with the permanent teeth's development. Turner's tooth, sometimes referred to as enamel hypoplasia, is the most prevalent abnormality. The enamel of a tooth may appear bright, rough, or pitted due to Turner's hypoplasia, which can affect the entire crown of the tooth or only a portion of it. This is a typical finding in maxillary incisors and molars alike. A complete medical history is necessary to properly diagnose discoloration, especially with reference to the tooth in question.[4]

When a trauma occurs prior to the third year of life, Turner's hypoplasia usually affects the enamel of teeth. After this, because the enamel has already calcified and hardened, injuries are less likely to result in enamel defects. Human deciduous teeth have fewer Wilson bands and are substantially less hypoplastic than their permanent counterparts.

Extended periods of time without interruption may make ameloblasts more vulnerable to disruption. The general pattern of larger teeth having a higher percentage of anomalies could be explained by this. The following categories of hypoplasia were established by Silberman et al.[5]

Type I hypoplasia: Enamel discoloration due to hypoplasia.

Type II hypoplasia: Abnormal coalescence due to hypoplasia.

Type III hypoplasia: Some parts of enamel missing due to hypoplasia.

Type IV hypoplasia: A combination of previous three types of hypoplasia.

In the present case, enamel hypoplasia occurred due to traumatic injury during developmental stages of permanent incisor. A permanent tooth's crown, root, or entire replacement may be affected by aftereffects of trauma to the primary dentition. The crown is affected by the structural alterations associated with yellow or brown pigmentation, dilatation of the crown, and enamel hypoplasia. Remnants and other sequelae may cause roots to become partially or totally displaced. When the entire bud of the permanent successor is impacted, changes in the eruption process, retention, or malformation of the permanent tooth may transpire.[6]

These teeth's structural defects not only make them weak, but they also provide the perfect setting for the colonization of bacteria. Hypoplasia makes permanent teeth seven times more susceptible to carious attack than teeth without it. Compared to normal teeth, hypoplastic primary teeth are twice as likely to acquire caries.[7] as a result, we filled the tooth with composite till more work was necessary to properly erupt it and safeguard it from any carious attack.

The inversion of regular teeth is unusual, with only a few documented cases, in contrast to the frequent reports of inverted supernumerary teeth. The upper lateral incisor has not been shown to be involved, as far as the authors are aware.

In some cases, a cause for the abnormal tooth position, such as local trauma, can be identified. as is in this case.

Inverted teeth appear to be less common in the mandible than in the maxilla (Mori et al., 1979). The mandible's second and third molars are the most commonly affected; reports of second premolars that have been successful in erupting have been made (Mori et al., 1979). The majority of inverted teeth, including the third molar, develop maxillary impacted teeth.[8]

Previous studies have suggested that inversion of teeth, or the deviation of teeth from their normal position, is caused by abnormal or unusual proliferation of odontogenic epithelium before the development of the tooth germ.[9]

Conclusion:

The need for close periodic examination and early detection of all possible developmental defects in the permanent dentition—as well as the importance of preventive measures—are necessary for maintaining the vitality of teeth.

Since information on the microstructural level of enamel hypoplasia and tooth inversion is still limited, further studies to be conducted to better understand the mechanisms behind non-vitality.

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