

Hypoplasia of a Permanent Incisor Produced by Primary Incisor Intrusion: A Case Report

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SOMMAIRE

Les traumatismes buccofaciaux sont de graves problèmes qui ont une incidence sur la santé buccodentaire et l'état de santé général et qui peuvent avoir des conséquences médicales, esthétiques et psychologiques chez les enfants et leurs parents. Lorsque la racine de la dent primaire se trouve près de la dent permanente qui n'a pas fait éruption, le traumatisme causé à la dent primaire peut perturber le développement de la dent permanente et causer des réactions pulpaires. Nous présentons un cas inhabituel où un traumatisme de la dent primaire a altéré le développement de la couronne et la formation de structures calcifiées dans la chambre pulpaire de la dent permanente. La malformation localisée de la couronne et l'hypoplasie de l'émail ont été traitées au moyen d'une restauration en résine composite photopolymérisée. Nous discutons également de la calcification de la pulpe et du traitement endodontique requis.

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Injuries to primary dentition are among the most common traumas that occur in the maxillofacial region; 30%–40% of all children injure at least one of their primary teeth.^{1,2} Consequences of such trauma include colour changes, pulp necrosis, obliteration of the pulp canal, gingival retraction, tooth displacement, pathological root resorption, alterations in the process of normal root resorption and premature loss of the primary tooth.^{3,4}

Sequelae in the permanent dentition after trauma to primary dentition are usually related to intrusive injury; either the coronal or root region, or the entire permanent tooth germ may be affected.^{5,6} An intrusive injury occurs when the impact of an axial force displaces the tooth within the socket. Between 18% and 69% of intrusive injuries to the primary dentition cause anomalous development of the permanent

teeth.^{5,7} Such alterations in dental pathology can include white or yellow-brown discoloration, or circular enamel hypoplasia; crown dilaceration; root duplication; vestibular or lateral root angulation or dilaceration; partial or complete arrest of root formation; sequestration of the permanent tooth germ; and disturbed eruption.⁸ Depending on the age of the child at the time of injury and the direction and severity of the trauma, force transmitted from the affected primary tooth may result in similar consequences to the underlying unerupted permanent tooth.⁷

Pulpal reaction to dental trauma varies. The most common complications are calcification and obliteration of the pulp.⁹ Calcification can vary from a small denticle to total obstruction of the pulpal canal. In the following report, we present the case of a permanent tooth in which localized crown malformation, enamel



Figure 1: Intraoral view shows crown malformation and enamel hypoplasia of the permanent maxillary left central incisor.



Figure 2: Initial radiograph of the injured teeth displayed radiopaque structures in the pulp chamber.



Figure 3: Intraoral view of the patient at the 15-month follow-up visit.

hypoplasia and calcified structures in the pulp of the permanent tooth were caused by trauma to its preceding primary tooth.

Case Report

A 7-year-old boy was referred to the Gülhane Medical Academy in Ankara, Turkey, for a complaint about the esthetics of his left central incisor. His medical history revealed that at 14 months of age he had injured his primary maxillary central incisors during play at home. After the injury, he had emergency treatment that involved suturing the lip and antibiotics at a hospital, but no professional dental treatment. Other medical records revealed that he had no general pathologic condition.

Clinical examination revealed that the patient had an unerupted permanent maxillary right central incisor, crown malformation and enamel hypoplasia of the permanent maxillary left central incisor, grade II gingival overgrowth,¹⁰ and yellow discoloration of the enamel (**Fig. 1**). Radiographic examination showed calcified tissue that resembled pulp stones in the pulp chamber of the permanent maxillary left central incisor (**Fig. 2**). No periapical condition or tooth fractures were observed.

The patient's parents were told that the excessive gingival tissue over the left central incisor could be removed to improve the appearance of the tooth, but they did not agree to a surgical procedure.

Routine follow-up visits every 3 months showed that the overall eruption patterns of the patient's central incisors were within normal limits. After 15 months, the right central incisor erupted in its normal position, but the left central incisor erupted more slowly than the right (**Fig. 3**). Additional brown lines and yellow discoloration were ob-

served on the left central incisor, and the rounded calcified structures were enlarged.

The treatment plan developed included instructions to improve oral hygiene, a gingivectomy to excise the gingival overgrowth, removal of the hypoplastic enamel and restoration of the region. At the 2-week follow-up visit, oral hygiene had greatly reduced the edematous gingiva, but not the gingival overgrowth. The patient's parents now agreed to the entire treatment plan, and the patient underwent gingivectomy. Local anesthesia was administered, and a pocket marker was used to make pinpoint perforations to indicate periodontal pocket depth. A beveled incision was made apical to the perforations to include the 2 adjacent teeth. Gingival tissue was excised, and gingival debridement done to eliminate deposits over the root surfaces. After surgical debridement, the area was covered with a surgical pack. Healing after the procedure was uneventful. No recurrent gingival overgrowth was observed during the patient's follow-up visits. The maxillary left central incisor was restored with a strip crown and light-cured composite resin (TPH; Dentsply Caulk, Milford, Del.). No problems occurred during the bonding procedure (**Fig. 4**), and occlusal adjustment was done. Because of the positive vitality test, endodontic treatment was not recommended. After the restoration, a periapical radiograph was taken, and the patient was followed up every 3 months. One year after the restoration, his left incisor responded positively to a vitality test, and a periapical radiograph showed normal root development (**Fig. 5**). The patient had no clinical or radiographic evidence of any pathologic condition during 2 years of follow-up. His periodontal structures were healthy and showed no signs of overgrowth or any other pathologic condition.



Figure 4: Intraoral view after restoration of the permanent maxillary left central incisor with a light-cured composite resin.



Figure 5: Periapical radiograph taken 1 year after the restoration.

Discussion

An injury to a young child's teeth can be physically and emotionally traumatic. The dentist must take time to carefully examine and analyze not only the damage itself, but also the possibilities of sequelae to the permanent tooth germ and the overall health of the child. For this reason, treatment of trauma in primary dentition must include long-term follow-up of sequelae in the permanent dentition.¹¹

The type of sequelae noted in permanent dentition can be explained in part by the age at which the trauma to the primary dentition occurred. Because the majority of traumatic injuries to primary teeth occur when children are between 1 and 3 years of age, developmental disturbances involving the crown of the permanent teeth are reported more frequently than developmental disturbances in the roots and in the eruption of permanent teeth.¹²⁻¹⁶ In the case reported here, developmental disturbances were observed in the crown only.

Formation of the permanent upper central incisor germ takes place at 20 weeks of gestation, and calcification begins when the child is 3 to 4 months of age. Depending on the severity of intrusion, intruded primary teeth can invade the follicle of the permanent germ and destroy the enamel matrix.¹⁴ Because ameloblasts are irreplaceable and no further cell division occurs after the completed formation of the enamel, trauma will likely arrest localized development of the crown.¹⁴ In the case reported here, the intrusive orofacial trauma to the primary left incisor that occurred when the patient was 14 months of age likely disturbed the crown formation and enamel matrix of the underlying permanent tooth, and caused changes in its colour and shape.

The literature^{7,17-19} contains a number of descriptions of the relationship between primary tooth trauma and permanent tooth hypoplasia. White discoloration is caused by the accelerated mineral deposition that results from trauma during the maturation stage of enamel development, whereas yellow-brown discoloration is caused by the incorporation of hemoglobin products from bleeding in the periapical area and enamel hypoplasia is caused by the destruction of ameloblasts in the active enamel epithelium.⁷

Pulp tissue is apt to form calcified structures as a result of traumatic force.²⁰ Although calcified structures and obliterations frequently occur in both primary and permanent teeth that have been directly traumatized, these problems occur very rarely in permanent teeth that underlie injured primary teeth. In a case similar to the one reported here, Katz-Sagi and others²¹ found unusual obliteration of the pulp canal in a maxillary central incisor

and crown malformation in the adjacent unerupted central incisor after trauma to the associated primary tooth. Bassiouny and others²² also reported a case of total and partial pulp obliteration of the maxillary central incisors after trauma to their associated primary teeth.

Conclusions

The case we report here stresses the importance of traumatic injuries to primary dentition because of their effects on the permanent tooth germ. Injured teeth should be followed up periodically for possible periapical infections and pulp necrosis. In addition, special care may be necessary in the restoration of injured teeth because their reaction patterns may differ from those of nontraumatized teeth. ♦

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