Developmental Disturbances of a Maxillary Central Incisor due to Trauma to Its Predecessor: A Case Report

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Introduction

Although dental injuries may occur at any age, one of the more likely times is from the age of 2–5 years, probably because at this age, children’s coordination and judgement are not properly developed and falls are common. Consequently, many of these children suffer injuries to their primary teeth, generally fractured or displaced primary incisors [1, 2]. Because of the close anatomic relationship between the apices of primary teeth and the germs of permanent successors, any trauma to the primary teeth may easily disturb the permanent dentition. Traumas may interfere with further odontogenesis, and, depending on the site and extent of the injury, different malformations may occur, ranging from a slight disturbance in the mineralization of enamel to a sequestration of the entire tooth germ [1, 2]. In treating traumatic injuries to primary teeth, the objective is to deal with pain and to prevent sequelae in the underlying permanent tooth germ; however, there is no agreement on the ideal treatment [3]. We report the effects of a primary tooth trauma on the underlying permanent tooth germ.
Clinical Presentation and Intervention

A 12-year-old girl was referred to our clinic with a complaint of poor aesthetic appearance. The patient revealed a history of trauma at the age of 4 years. There was no record of the incident, but the girl’s mother affirmed that the patient had fallen onto a sofa, and the primary maxillary left central tooth was dislocated from the socket with increased mobility. The girl was not seen by a dentist immediately after the trauma. Approximately 2 weeks later, when the tooth mobility increased, she was referred to a private dentist and the tooth was extracted. It was reported that the permanent maxillary left central tooth had erupted at 7 years of age with yellow-brown stains. The patient stated that initially there was no pain associated with this tooth, but she currently suffered from sensitivity to cold and sweets.

Discussion

Discoloration, alteration and hypoplasia of the enamel are the most frequent sequelae in successors after trauma to the primary tooth. Other sequelae that may occur less often are dilaceration of the crown and root, sequestering of the germ of the permanent tooth and even root duplication. The severity of sequelae is associated with different factors, such as age at the time of the accident, the degree of root resorption of the injured primary tooth, the type and extent of the traumatic lesion and the stage of development of the permanent tooth germ [1, 2, 4].

In the present case, there were both enamel hyperplasia and hypoplasia with yellowish-brown discoloration of the permanent maxillary left central incisor. Ameloblastic activity interrupted by the trauma contributed to the formation of areas of irregular and imperfect enamel on the buccal side of the permanent maxillary left central tooth, which probably caused the formation of hyperplastic enamel after the injury [4]. Yellow-brown discolorations are caused by the incorporation of breakdown products of hemoglobin from bleeding in the periapical area. The breakdown products of hemoglobin from bleeding can be incorporated into the tooth during tooth formation, even after the ameloblast activity is arrested [2].

Dilaceration of teeth is assumed to be a disturbance in the growth of the epithelial root sheath of Hertwig, and its true cause is unknown. Acute trauma, scar formation and primary tooth germ developmental anomalies may be contributing factors [5]. It has been reported that crown dilaceration occurs in cases with injury at an age between 1.5 and 3.5 years, and root malformation with injury between 4 and 5 years of age. The trauma in the
present case, which occurred at 4 years of age, could have caused a direct impact on the root angulation [2].

Severe dental malformation resulting from injury to the primary dentition has been described previously [6]. However, most of the cases presented are the result of intrusive injuries to the primary teeth [6–8]. As our patient was not seen by a dentist at the time of injury, her mother’s statements were taken into consideration, and it was thought that the primary tooth was extrusively luxated. No other case reports were found in the literature reporting the sequelae of extrusive luxation of primary teeth to their successors.

In the present case, multiple abnormalities in a permanent tooth following trauma to its predecessor are reported. It is a rare case demonstrating enamel hypoplasia, hypoplasia, discoloration and root dilaceration in the same tooth as sequelae after traumatic injury. Tewari and Pandey [9] reported similar multiple sequelae but not in the same tooth, rather in teeth in the same quadrant of the jaw.

Arikan et al. [10] reported that in the absence of acute symptoms, parents tend not to visit a dental clinic for children’s dental injuries, especially those affecting primary teeth. Consistent with their findings, in the present case the parents did not visit a dentist immediately. Traumatic injury to the primary teeth may directly affect the underlying permanent tooth germ; moreover, subsequent local infection may also hinder the regular formation of tooth tissues. When regular follow-up is carried out appropriately after a precise diagnosis, the chances of sequelae can be minimized.

Conclusion

In the present case, sequelae of a primary tooth trauma on the permanent tooth were restored. We recommend that parents should be aware of the consequences of untreated trauma to a primary tooth. Educational and preventive programmes on dental trauma are required to educate parents about emergency knowledge and sequelae of dental trauma.

References