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Dilaceration of maxillary central incisor: a literature review

REVIEW ARTICLE

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Abstract – The main purpose of this review is to present the aetiological factors and the mechanism that cause dilaceration of the maxillary central incisors. In early developmental stages, the permanent tooth germ of the maxillary incisor is situated palatally and superiorly to the apex of the primary incisor and gradually changes direction in a labial direction with its crown coming closer to the resorbing primary root. For reasons of this close relationship between the permanent tooth germ and the apex of the primary incisor, it is believed that an acute trauma to the primary predecessor can cause dilaceration of the long axis of the permanent successor. Clinically, dilaceration can be revealed by palpation high in the labial sulcus or in the hard palate, while its radiographic view is characteristic. The therapeutic approach to the dilacerated maxillary central incisors has to be carefully planned and needs the cooperation of several specialities to attain the final objective.

The definition of root dilaceration varies in the relevant literature and depends on the criteria set by each author. For example, certain authors consider a tooth dilacerated when there is a mesial or distal tilt of the root and the angle is equal or exceeds 90° in relation to the tooth or root axis (1). Others consider a tooth dilacerated when its apical deviation is equal or exceeds 20° in relation to the normal tooth axis (2).

The term dilaceration was first coined in 1848 by Tomes (3), who defined the phenomenon as the forcible separation of the cap of the developed dentine from the pulp in which the development of the dentine is still progressing. Later, it was defined as an angulation or deviation or sharp bend or curve in the linear relationship of the crown of a tooth to its root (4–6). According to the glossary of dental terms (7), dilaceration is defined as the deformity of a tooth due to a disturbance between the unmineralized and mineralized portions of the developing tooth germ.

Andreasen et al., in 1971, defined dilaceration as the abrupt deviation of the long axis of the crown or root portion of the tooth, which is due to a traumatic non-axial displacement of already formed hard tissue in relation to the developing soft tissue (8, 9). The same (8, 9) and other authors (6) distinguish dilaceration from angulation, which is described as a bend of the root due to the gradual change in the direction of development, when no abrupt shift of the tooth germ has taken place during odontogenesis.

Aetiology

The aetiology of dilaceration is not fully understood without any consensus among researchers. There are two main explanations about the causes of dilaceration: The first suggestion, which is the most acceptable hypothesis, proposes that an acute mechanical injury to the primary predecessor tooth causes leads to the dilaceration of the underlying developing succedaneous permanent tooth. The hypothesis in question is supported by the fact that when the dental history of a case is taken, it is most often reported that there had been an injury at an earlier age. The calcified part of the permanent tooth germ is shifted in such a manner that the rest, non-calcified part of the permanent tooth germ forms an angle (10–15). However, the incidence of permanent successor teeth dilaceration is very low and disproportionate to the rates of primary teeth injuries (11, 16).

In 1978, Stewart (17) studied the phenomenon in 41 cases of dilacerated incisors and found that only in 22% (nine patients) of the cases, this was due to injury. Therefore, he concluded that the cause lay in the ectopic development of the tooth germ (17). This view was supported by Howe (18), who claims that an injury transmitted to the crown of the non-erupted tooth cannot lead to usual curvature orientation of maxillary central incisors, with the crown facing upwards and labially, unless the tooth germ of the permanent successor had already been displaced before the traumatic injury. McNamara et al. (15) and Singh and Sharma (19) report and add that injury has been proposed as an aetiological factor with traumatic injuries recorded in the developing dentition period, but there are also reports of dilaceration among patients without a history of injury. Furthermore, sometimes, history of trauma is not reported because such dental injuries of early childhood may go undetected or merely forgotten by parents. Others suggest that the injury of a primary predecessor

tooth is not the exclusive aetiological factor of dilaceration as it is usually only one tooth that presents dilaceration, whereas, if injury was the only aetiological factor, then adjacent teeth should be involved in the dilaceration more often (15, 19).

The second explanation proposes that idiopathic developmental disturbances lie at the cause of dilacerations, mainly in cases where there is no clear sign or history of traumatic injury (8, 10, 20, 21) (Fig. 1). Supporters of this theory maintain that an injury to a primary tooth sometimes leads to intrusion or avulsion, an event that normally occurs before the age of four. At this age, the formation of the root of the succedaneous permanent tooth does not start and hence it is reasonable to question the extent to which dilacerated teeth are due to injury of primary predecessor teeth. Therefore, it is supported that injury is not the main aetiological factor of dilaceration and that this disorder is caused by ectopic tooth germ development (8, 20–23). Furthermore, some researchers support this theory because dilaceration is observed more frequently in posterior teeth, which are less susceptible to traumatic injury (1).

Other possible synergistic factors mentioned in the literature include the formation of scar tissue, developmental disorder in the primary tooth germ, facial clefting (24), advanced infection of root canals (25), ectopic tooth germ development (8, 17, 26), lack of space (8, 17, 26) and the effect of anatomical structures, for example, the cortical bone of the sinus, the mandibular canal and the nasal fossa, which may shift the epithelial diaphragm (27). Orotracheal intubation and laryngoscopy have also been blamed causes for the dilaceration of primary maxillary central incisors (28–30), as well as the presence of cysts, tumours, odontogenic hamartoma (17, 28, 31–33), mechanical interference during eruption, such as an ankylotic primary tooth the roots of which are non-resorbed (30), tooth transplantation (34), extraction of a primary tooth (35) and hereditary factors (36–38). Certain syndromes and developmental disorders have also been associated with root dilaceration, such as the Smith-Magenis syndrome (39), the hypermobility type of Ehlers-Danlos syndrome (40), the Axenfeld-Rieger syndrome (41) and congenital ichthyosis (42).

Mechanism causing dilaceration

The hard tissue between a primary maxillary central incisor and its permanent successor is < 3 mm in thickness and mainly consists of fibrous connective tissue (43, 44). The close anatomical relationship between the developing permanent tooth germ and the root of the primary central incisor explains the severe development disorders observed in its permanent successor, following strong mechanical injury to its primary predecessor (43, 45–47). One of these disorders is the dilaceration of the root of the permanent central incisor, the position of which depends on the developmental stage of the tooth at the time of the injury (9, 48–50).

The vertical direction of the impact force is transferred in the direction of the longitudinal axis of the primary incisor and it may be transferred further up, through the apex, to the non-calcified or partially calcified tooth germ of the permanent successor (11).

Normally at the age of 2–3 years, the tooth germ of the permanent maxillary incisor lies in a palatal position, above the apex of the primary incisor (Fig. 2). If the child suffers an injury at this age, the potential consequence on the successor tooth would affect the buccal surface of its crown (45). Gradually, at the age of 4–5 years, the tooth germ of the permanent incisor develops in a labial direction, coming closer to the resorbing root of the primary tooth (46, 51) (Fig. 3). If the child is injured at this critical age, when the crown of the permanent tooth is

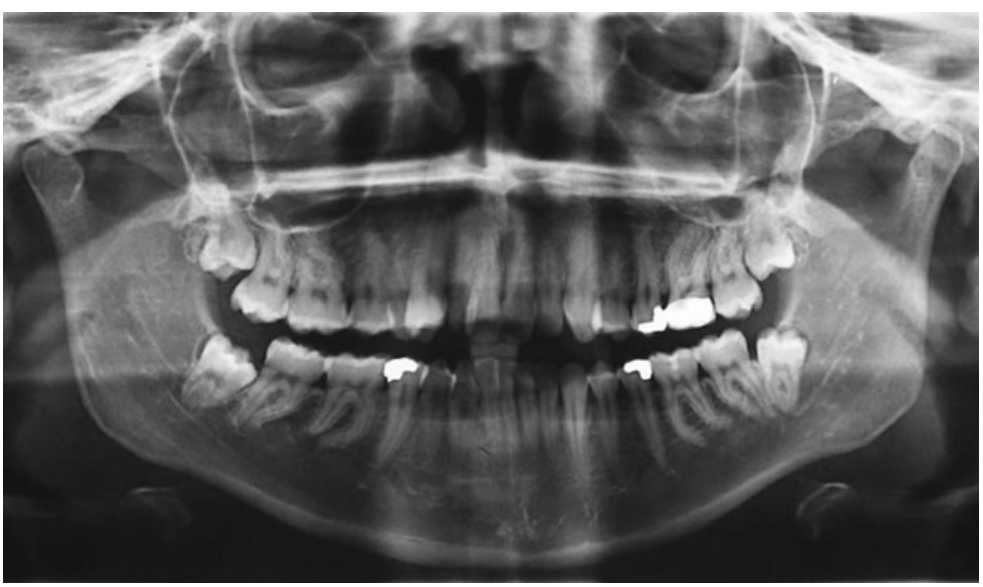

Fig. 1. Panoramic radiographic image of a patient whose roots were presented with dilacerations. They are attributed to idiopathic developmental disturbances.

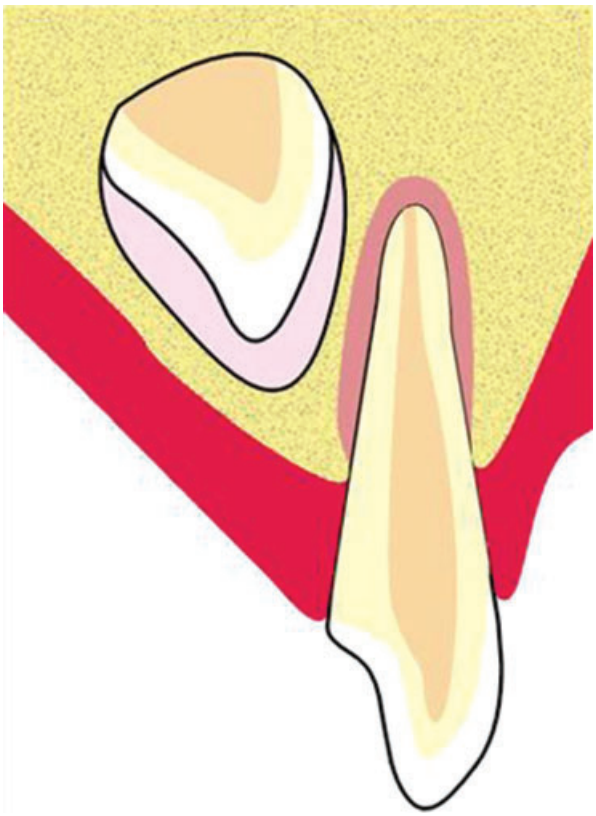

Fig. 2. Close anatomical relationship between the maxillary deciduous incisor and permanent successor tooth germ at 2–3 years of age.

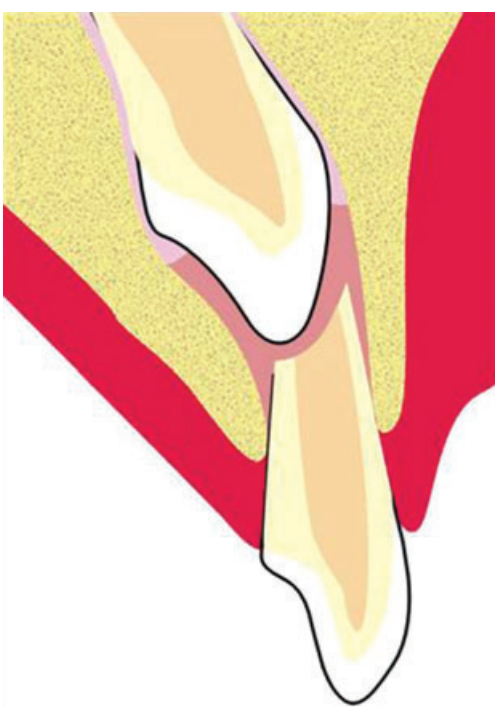

Fig. 3. Close anatomical relationship between the maxillary deciduous incisor and permanent successor dental germ at 4–5 years of age.

in direct relationship with the resorbed root of the primary predecessor, the impact force will be transferred along an imaginary oblique line that goes through the incisal edge of the permanent incisor and a point on the labial aspect of its newly formed root (49) (Fig. 4). It is estimated that the direction of this force may be more significant than its magnitude. As the impact force is directly transferred to the cells of Hertwig's epithelial root sheath, through the sharp end of the non-formed root of the permanent tooth, it is possible for serious damage to be caused despite the relatively mild forces involved. The resorbing apex of the primary incisor creates an impact point with the incisal edge of the crown of the permanent incisor and causes this crown to turn upwards into its tooth follicle (9, 11, 46, 49). As the permanent incisor root has not been fully developed at the moment of injury, the part of the root already formed will rotate along with the crown. However, further root development, following the injury, usually continues in the same direction it was following before the injury. This creates an unusual angle between the pre- and post-traumatic parts of the tooth, which results in local curvature of the longitudinal axis of the permanent central incisor and causes dilaceration (49).

As the injured Hertwig's epithelial root sheath continues to produce dentine at the same rate as before the injury, the final root shape of the permanent maxillary central incisor will be formed in a continuous labial curve, until apex formation has been completed (9, 49). Furthermore, as the Hertwig's epithelial root sheath remains in its place within the alveolar process against the eruptive forces of the developing tooth and guides the orientation of root development, the crown of the permanent central incisor appears to be moving labially

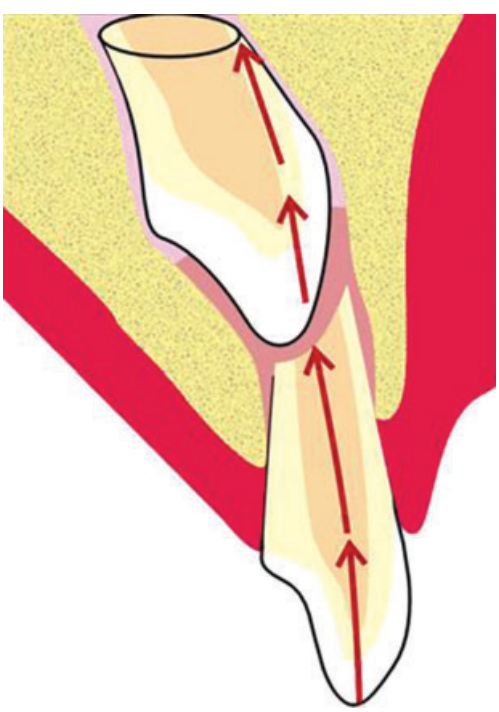

Fig. 4. Direction of force transmission following traumatic injury of the maxillary deciduous incisor.

and upwards for as long as this asymmetric calcification of the root continues (8, 9, 49) (Fig. 5).

Epidemiology

Dilaceration might occur anywhere along the length of the tooth, i.e. the crown, the cement-enamel junction, along the root or the root apex (4, 6, 15, 26). Crown dilaceration of a permanent tooth constitutes 3% of all traumatic injuries to developing teeth and is habitually due to intrusion or avulsion of their primary predecessors (8, 52). It usually involves maxillary or mandibular central incisors (8, 52) and more often maxillary incisors than their mandibular counterparts (14). Approximately, 50% of teeth with crown dilaceration become impacted with the remainder erupting normally or in a labiolingual direction (9, 52). The clinical appearance of this deformity in succedaneous permanent tooth depends on the developing stage at which the injury occurred (53).

Dilaceration may appear in both permanent and primary teeth, yet at much lower prevalence in the latter case (10, 15, 19, 54). While some studies report no gender preference for dilaceration (55), others report a male to female ratio of 1:6 (15, 17). In Malčić et al. (26) reported a prevalence rate of 1.2% or 0.53% for maxillary central incisors on the basis of periapical and panoramic radiographs respectively. Hamasha et al. (1) examined 4655 teeth on periapical radiographs and found that 176 (3.78%) presented dilaceration. Maxillary central and lateral incisors had rates of 0.4% and 1.2% respectively (1).

Fig. 5. The crown of the maxillary permanent central incisor has shifted labially and upward following traumatic injury, while Hertwig's epithelial root sheath remains in position despite the trauma impact.

In 2005, Malčić et al. (26) reported that dilaceration is observed in the apical third of the roots of incisor, canines and premolars, while the middle third is more often affected in molars and finally, the cervical third in third molars. These authors also reported that premolars and maxillary anterior teeth present a higher total prevalence (4.6%) as compared with the rate affecting the corresponding region of the mandible (1.3%).

No bilateral dilacerated teeth have been observed in the same patient (56, 57), while the presence of dilacerated teeth in both the maxillary and mandibular dental arches in the same patient is quite a rare phenomenon (37).

The most common type of dilaceration is that of a tooth root angulation combined with a reversal crown direction. The palatal aspect of the crown faces the labial side and the tooth is usually impacted (15, 17, 19, 58, 59). Crown dilaceration of permanent maxillary incisors usually presents with palatal angulation, while permanent mandibular incisors usually present crown dilaceration with labial angulation (8, 60). Dilaceration is more often observed in the root of the affected tooth and most commonly in permanent maxillary incisors. This is likely due to their close topographic relationship with primary teeth, which are often injured (61).

Clinical features of dilaceration

Clinical features of dilaceration usually include the non-eruption of the responsible tooth, the longer retention of the primary predecessor tooth, possible apical fenestration of the buccal or labial cortical plate, or it may be asymptomatic (10, 28, 54, 62). The presence of dilaceration in an impacted maxillary central incisor may be diagnosed clinically through palpation at two locations. The first lies high on the labial side of the alveolar ridge in the vestibular sulcus. The upper middle line is normally on the same line as the projection of the anterior nasal spine with a shallow depression on either side. In cases of dilaceration of the permanent central incisor when the palatal surface of the crown has rotated anteriorly, there is pronounced swelling in the region in place of the shallow depression. When the upper lip is pulled upwards, the oral mucosa moves freely above the protruding region which indicates the outline of the crown of the impacted dilacerated central incisor. The importance of such a palpation should not be underestimated, because if it is not thorough, the opportunity for an important diagnosis might be missed (49). The second palpation area lies in the palate. If there is an abnormal position, such as when the crown has rotated upwards and labially, the root continues to develop along a more palatally tilted axis. Therefore, at the final stages of incisor root formation when the apex is closed, the apex may be palpated in the palate as a small hard nodule (49).

Radiographic features of dilaceration

White and Pharoah (21) reported that the mesial or distal root curvature of dilacerated roots is clearly discernible on the periapical radiograph (26). However, if the

curvature lies in a labial-buccal direction, the central x-ray beam passes almost parallel to the deviating part of the root. Therefore, the deviating root portion appears at the end of the non-deviating portion as a circular radiopaque region with a dark central radioluscent spot, which represents the apical foramen and a part of the root canal as well. This radiographic image is known as a Bull's eye (21, 63, 64) (Fig. 6). The periodontal ligament around the deviating part of the root appears as a black region (radiolucent halo). The deviating portion of the root appears more radiopaque as compared with the rest of the root because the x-ray beam passes through a higher osseous density portion of the root (21, 63, 64).

In the cases of labial-buccal and lingual-palatal root dilaceration, the use of a panoramic radiograph as the only means of detection is insufficient and it is necessary to exposure further radiographic images at a different angulations (65).

Besides periapical radiographs, occlusal (Fig. 7), lateral cephalometric and panoramic radiographs (Fig. 6) may more clearly identify the position and extent of central incisor root dilaceration. As a complementary imaging method, computerized tomography is very useful. It depicts sections at various depths of the region of interest and allows clinicians to assess accurately the exact position of the crown, apex and the degree of dilaceration (46).

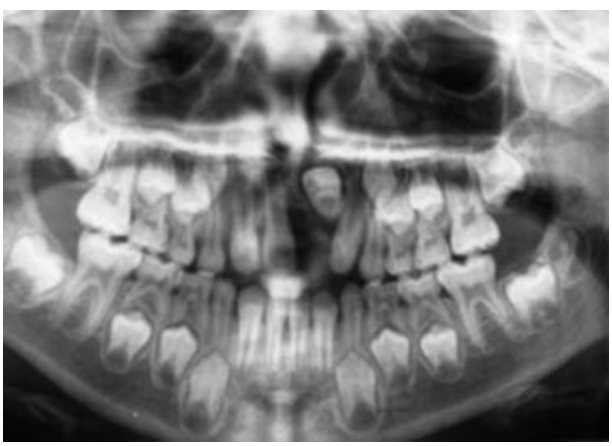

Fig. 6. Panoramic radiographic image of the same child. The characteristic 'Bull's eye' is discernible.

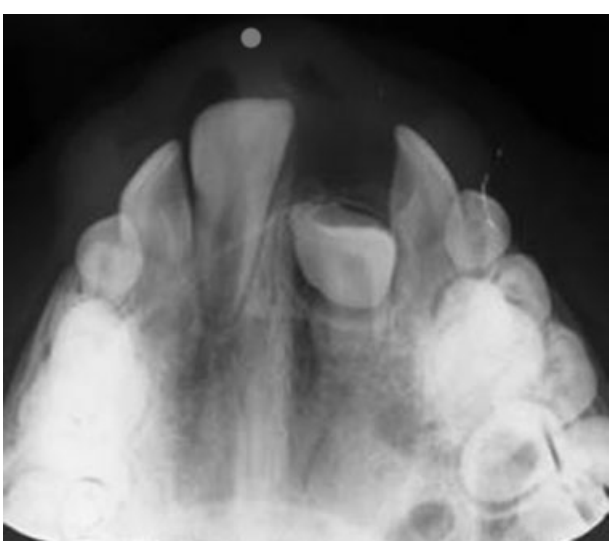

Fig. 7. Occlusal radiographic image of a child with maxillary left permanent central incisor dilaceration.

Prognosis of dilaceration

The success rate of an impacted dilacerated tooth alignment mainly depends on the following factors: (i) the position and direction of the impacted tooth, (ii) the degree of root formation, (iii) the degree of dilaceration and (iv) the availability of space for the impacted tooth (66–69). Machtei et al. (70) also include the condition of the periodontium. McNamara et al. (15) underline the decisive significance of the post-traumatic condition of the Hertwig's epithelial root sheath for a successful therapeutic outcome, as the odontogenic epithelium plays a truly important role in root formation through the effect of its Hertwig's epithelial root sheath. Continuing normal root development depends on the integrity of the Hertwig's epithelial root sheath (15, 51). A dilacerated tooth with an obtuse inclination angle, a lower position in relation to the alveolar crest combined with an incomplete root formation has a better prognosis for orthodontic traction (67, 69).

Treatment of dilaceration

The treatment of dilacerated teeth should start early, to provide the opportunity for the non-calcified root to change direction and develop a proper spatial relationship with the already calcified formed crown (15). Due to its position, the problem is usually recognized by the parents during the child's mixed dentition period (58). Failure to treat in a timely manner may lead to delayed tooth eruption, midline shift, space occupation by adjacent teeth and alveolar crest height differences (58). Treatment should take place following careful planning and requires cooperation of various experts (71).

Two differing treatment approaches to manage an impacted dilacerated permanent teeth, both of which are time consuming, need to be considered – surgical exposure with orthodontic traction vs extraction and prosthetic replacement with fixed bridge or implant placement (12, 13, 15, 68, 69). The most commonly used, but often complex, technique is the surgical exposure of the tooth and its orthodontic traction into the dental arch (69, 72–74). Parents often prefer the option as it maintains a chance for preservation of the impacted tooth. Even after successful orthodontic treatment, aesthetic periodontal surgery might be necessary if the final position of the gingival margin is not acceptable. This is decided when the gingival margin is not acceptable, as in the cases of hyperplasia in the cervical region of the tooth (12), where gingival recession and/or clinical crown lengthening might occur (69, 70, 75–79). On the contrary, in some cases where there is no aesthetic problem, a compromised gingival margin might be

acceptable and further periodontal surgery avoided (69, 70, 75–79). Orthodontists are often reluctant to proceed with moving pronouncedly dilacerated teeth as treatment might fail due to complications such as ankylosis, loss of attachment, external root resorption and/or root exposure following orthodontic traction (59, 69, 80, 81). In cases of root exposure, endodontic treatment or/and apicoectomy would be necessary (59, 68, 69, 73). However, in some cases, dental extraction might be the only option due to pronounced tooth inversion (12, 17, 59).

Should exposure and orthodontic traction either fail or not be initially considered, the treatment must consist of surgical tooth extraction. This would be followed by an implant or space closure with orthodontic traction or fixed bridge or partial denture placement (12, 13, 15, 68, 69).

Of course, if tooth extraction is chosen, there is the consequence of alveolar bone, which will lead to the formation of a horizontal and/or vertical osseous defect (82). Autotransplantation using a premolar has also been described or surgical repositioning of the same tooth in the dental arch (58).

Conclusions

Dilaceration of permanent teeth is a relatively rare phenomenon. However, its significance should not be underestimated, as it is the main reason for their impaction and involves complex treatment. The aetiology of the phenomenon is not completely clear. In many but not all instances of dilaceration, there has been a prior injury to the predecessor primary incisor. The trauma forces the already calcified part of the permanent tooth to follow an upward and labial direction, forming an angle between its calcified and non-calcified parts. Dilacerated teeth are diagnosed clinically, but radiographic imaging plays a decisive role. Treatment should start as early as possible and aim at tooth alignment in the dental arch following surgical exposure and orthodontic traction. To this end, an inter-disciplinary approach is considered necessary to ensure an optimal outcome.

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