



NONVITALITY AND TURNERS HYPOPLASIA - A REVIEW

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ABSTRACT

The topographic relationship of the primary tooth to the developing permanent tooth germ explains the possible developmental disturbances which may mild alteration in enamel mineralisation, dilacerations of the crown or root, crown or root duplication, complete or partial arrest of root formation to severe sequestration of tooth germ.⁽²⁾ Aberrations affecting the internal and external morphology can at times be the cause of complex pathological conditions involving the pulpal and periodontal tissues and can pose a challenge to the clinician for the diagnosis and clinical management. Detecting and treating the anomalies at an early phase is essential as it poses a threat for the loss of vitality of the concerned teeth. In Turner's hypoplastic types, enamel may be pitted, rough, or glossy. Assessment of enamel thinness or absence, through use of dental radiographs, is not only used for diagnosis but also provides clinical information necessary for the development of an optimal treatment approach. Endodontic treatment with minimum surgical intervention is used for management of turner's hypoplasia.

KEY WORDS

primary tooth, turner's hypoplasia.

INTRODUCTION

The main aim of this article is to determine the sequelae of permanent teeth due to traumatic injuries of their predecessors, causing hypomineralization and defective enamel matrix formation. The alterations may either occur at the time of accident or be caused by post-traumatic consequences. Hypoplasia is defined as a quantitative defect of enamel visually and is histomorphologically identified as an external defect involving the surface of the enamel and associated with reduced thickness of enamel.⁽¹⁾

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an early phase is essential as it poses a threat for the loss of vitality of the concerned teeth.⁽³⁾ Turners hypoplasia usually is manifested as a portion of missing or diminished enamel on permanent teeth. Unlike other abnormalities which affect a vast number of teeth, Turner's hypoplasia usually affects only one tooth referred as Turners tooth

Cause for hypoplasia

The growing dental structures, particularly the enamel and dentin, yield accurate, prompt, and permanent records of both normal fluctuations and pathologic accentuations of mineral and general metabolism. Fortunately, these records are easily read, by the orderly and rhythmic growth of these tissues in their daily ring like succession. In enamel hypoplasia, the history of systemic disturbances is indelibly recorded by a cessation of ameloblastic activity (Sarnat and Schour, 1941:1989).

Thus, the enamel and dentin in the formative and calcifying stages of their growth serve as kymographs on which are permanently recorded the physiologic or pathologic changes in metabolism that occur within the organism. This concept has been verified many times

by histologic studies of the teeth of experimental animals and of man (Massler et al., 1941:36)

Human deciduous teeth are far less hypoplastic and have far fewer Wilson bands than their permanent successors. While the low frequency of defects on deciduous teeth has most often been attributed to their developing during a protected period, they may also have a differential susceptibility. Interestingly, the same pattern of susceptibility seems to hold true for opacities.⁽¹⁶⁾

Ameloblasts that function for a longer period may become increasingly susceptible to disruption. This may explain the general trend to find a high proportion of defects on bigger teeth.

Additionally, several potentially significant phenomena tend to be coincident, including 1) angle of prisms, 2) curvatures of the striae of Retzius, 3) "age" of the secretory ameloblasts, 4) rates of enamel secretion, and 5) **lengths** of prism. Of all these factors, we suggest that the age of the ameloblast might be most significant. When enamel secretion is disrupted the more recently activated ameloblasts, those that are most cervical, produce the least disrupted prisms. A defect near the CEJ often displays a number of defective striae, while having only a shallow surface disruption.⁽¹⁷⁾

Physiologically, it is plausible to suggest that older ameloblasts might somehow become fatigued.

Turners hypoplasia

Turners hypoplasia is a developmental abnormality found of tooth, which is manifested as a portion of missing or diminished enamel on permanent teeth. If Turner's hypoplasia is found on a canine or a premolar, the most likely cause is an infection of the primary predecessors. The periapical inflammation around the decayed primary tooth affects the development of the permanent tooth. The tooth most likely affected is the canine.⁽⁴⁾ The appearance of the abnormality will depend on the severity and longevity of the infection. Turner's hypoplasia usually affects the tooth enamel if, the trauma occurs prior to the third year of life. Injuries occurring after this time are less likely to cause enamel defects since the enamel is already calcified.

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Hypoplasia was categorized into the following types 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.

Both enamel hypoplasia could affect dentitions. However, the incidence is more severe in permanent dentition. The characteristic features of enamel hypoplasia include poor esthetics, dentin hypersensitivity, and more susceptible for caries formation, which can lead to nonvitality of the tooth.⁽⁶⁾

Sequelae of turners hypoplasia in permanent dentition.

Sequelae in the permanent tooth stemming from trauma to the primary dentition can affect the crown, root, or the entire permanent successor. Sequelae that affect the crown are structural alterations associated with hypoplasia of the enamel, dilaceration of the crown and yellow or brown coloration. Sequelae that affect the roots include duplication and partial or total dilaceration. When the entire bud of the permanent successor is affected, there may be alterations in the eruption process, retention, or malformation of the permanent tooth.⁽⁷⁾

The prevalence of caries in hypoplastic teeth is more. The association between dental caries and enamel opacities was statistically significant which was also observed by Seow, Golpaygani *et al.*, Hong Levy. This may be because in addition to the lack of maturation the presence of developmental structural defects in

enamel may increase the caries risk. Gillepsie in a study on enamel hypoplasia and dental caries found that, caries superimposed on hypoplastic teeth becomes more extensive as the age advances.⁽⁸⁾ The hypoplastic teeth have an increased susceptibility to dental caries than a normal tooth, and in type IV hypoplasia the susceptibility rate is significantly increased. The hypoplastic permanent teeth are seven times more sensitive to carious attack compared to those without hypoplasia. Hypoplastic primary teeth are two times more prone to caries than normal teeth.⁽⁵⁾

The brown discoloration seen in the hypoplastic tooth occurs due to disturbances in ameloblastic layer, leading to defective enamel matrix formation caused by traumatic injuries, but the stretched inner enamel epithelium continues to induce the differentiation of new odontoblasts and hence the dentine formation is not affected.⁽⁹⁾ Consequently, a horizontal band of dentine without enamel on the facial aspect is evident whereas the displaced inner enamel epithelium and ameloblast form a cone of hard tissue on the lingual aspect usually projecting into the pulp canal. The intact lingual cervical loop forms an enamel covered cusp.⁽¹⁰⁾ These structurally defective teeth are not only weak but also provide favorable area for colonization of bacteria. Long standing untreated carious tooth can lead to turners hypoplasia, pulpal infection and pulpal necrosis o the primary dentition can cause increased osteolysis of the inter radicular bone and causes premature exposure of the successor tooth before the root formation.⁽⁵⁾

Non-vitality in hypoplastic teeth

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.⁽¹⁸⁾ Pulpal reaction to dental trauma varies. The most common complications are calcification and obliteration of the pulp.⁽¹⁹⁾ Calcification can vary from a small denticle to total obstruction of the pulpal canal.

Because most 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.^(20,21) 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.⁽²²⁾ 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.⁽²⁶⁾

The hypoplastic tooth can also turn non-vital without any history of trauma or any carious insult. The loss of vitality is because of the defective enamel and open dentinal tubules which act as a nidus for bacterial entry into the pulp space, thereby leading to pulp necrosis.⁽¹¹⁾

Pulp tissue is apt to form calcified structures because of traumatic force.⁽²³⁾ Although calcified structures and obliterations frequently occur in both primary and permanent teeth that have been directly traumatized. Katz-Sagi and others 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.⁽²⁴⁾ Bassiouny and others also reported a case of total and partial pulp obliteration of the maxillary central incisors after trauma to their associated primary teeth.⁽²⁵⁾

Management

Treatment planning for patients with hypoplasia is related to many factors:

The age and socioeconomic status of the patients, the type and severity of disorder and intraoral situation. Interdisciplinary approach is used to resolve esthetic problems using a combination of orthodontic, prosthodontic, and restorative treatment.⁽¹²⁾ Esthetic restoration of anterior teeth has been achieved with complete crowns, porcelain laminate veneers, and acid-etched composite resin restorations.⁽¹³⁾

A new therapeutic method named the edge-up technique developed by Fischer *et al.*, has long-term experience with the facet technique, which allows treatment of defects in the anterior area with a maximum preservation of enamel. The name edge-up describes the principles and components of the

restoration, edge focusing on the location, and set up aiming at the rebuilding of the incisal part of a tooth.

(14)

In Turner's hypoplastic types, enamel may be pitted, rough, or glossy. Assessment of enamel thinness or absence, through use of dental radiographs, is not only used for diagnosis but also provides clinical information necessary for the development of an optimal treatment approach. Endodontic treatment with minimum surgical intervention is used for management of turner's hypoplasia. (15)

CONCLUSION

Early detection of developmental defects and periodic dental visits is essential in maintaining a good dentition and sustaining the vitality of the tooth. Parents should be educated in the early detection and intervention of minor injuries which may have occurred in the early childhood and not caused any symptoms but can influence the developing permanent tooth.

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