

Case Report

Esthetic Management of Turner's Tooth

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Abstract

Disturbances during tooth development can be manifested as enamel hypoplasia, diffuse or demarcated enamel opacities or enamel hypomineralization. These defects can be the result of hereditary factors or environmental factors. Enamel Hypoplasia is an exclusive ectodermal disturbance, related to alterations in the organic enamel matrix which can cause white flecks, narrow horizontal bands, lines of pits, grooves and discoloration of the teeth varying from yellow to dark brown. This case report describes a treatment modality for Turner's hypoplasia done with a conservative approach for the esthetic problem of the defect.

Key Words: Turner's Hypoplasia, Enamel hypoplasia, Fluorosis.

Introduction

Attractive smile is a prime asset to a person's powerful personality and it can be an important factor in the desirable life experiences of a human being. Esthetic dentistry demands attention to the patient's desires and treatment of the patient's individual problems. The objectives of esthetic rehabilitation must be to provide the maximum improvements in esthetics with the minimum trauma to the dentition¹.

Enamel is a unique mineralized tissue in its method of development, structure and chemical nature. It is the only mineralized tissue of ectodermal origin in vertebrates². Developmental defects of enamel are a frequent finding in a primary as well as permanent dentition. Clinically they are of three types – enamel hypoplasia, demarcated enamel opacities and diffuse enamel opacities³. 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 enamel⁴.

Enamel hypoplasia is a serious problem that can result in a compromised oral health that causes physiological and psychological disturbances⁵. The characteristics of clinical enamel hypoplasia include unfavorable esthetics, higher dentin sensitivity, malocclusion and dental caries susceptibility⁴. A large number of causes have been described for enamel defects, both environmental and genetic. Enamel hypoplasia may be inherited as primary defects of enamel or may be acquired as a result of childhood medical problems such as infections, metabolic derangements, premature birth and nutritional disorders².

Turner's hypoplasia, also known as Turner's tooth, is a term used to describe a permanent tooth with a hypoplastic defect to its crown. Localized apical infection or trauma to a deciduous tooth is transmitted to the underlying permanent tooth. If the infection or

trauma occurs while the crown of the permanent tooth is forming, the resulting enamel will be hypoplastic and/or hypomineralized⁶. Frequently, the maxillary permanent central incisors are affected because of trauma to the overlying deciduous incisors⁶.

Enamel Hypoplasia (EH) has been associated with increased prevalence of caries⁷. The primary clinical problems are tooth sensitivity, loss of vertical dimension, dysfunction and aesthetics⁷. The esthetic treatment of EH is limited to the removal of surface stains, elimination of the defective tooth tissue, and masking of the defects. Attempts should be made to achieve treatment objectives while keeping the loss of tooth substance to a minimum⁷. This case report describes treatment modality for Turner's hypoplasia done with a very conservative approach for the esthetic problem of the defect.

Case Report

A 13 year old female reported to the Pedodontics Department, Rungta Dental College, Bhilai (Chattisgarh) with a chief complaint of brownish discoloration in upper front tooth region since 5 years. Patient gave history of fall and trauma to primary anterior teeth around 7-8 years ago. Patient came from high fluoride belt area. On intraoral examination, dentition exhibited generalised diffused opacities in enamel at cervical level with horizontal groove localised at 21. At middle 3rd on the facial aspect of 21 brownish discoloration was noticed (Fig 1,2). Vitality test for 21 showed positive response for thermal and electrical pulp testing indicating the vital status of the pulp. The radiographic status showed no abnormality of the hard tissue at the periapical area (Fig 3). Diagnosis was made as Turner's hypoplasia with 21 along with generalised fluorosis. Treatment planning was done considering conservation of tooth structure and esthetics. During the initial appointment, thorough oral prophylaxis was done. Then anterior tooth was restored using light cure composite (Fig. 4).



Fig 1 - Clinical photograph showing yellowish discoloration on labial aspect of 21 with linear groove running mesiodistally at gingival 3rd level.



Fig 2 - Right and left lateral profile showing cervical opacities.



Fig 3 - Preoperative X-ray i.r.t. 21

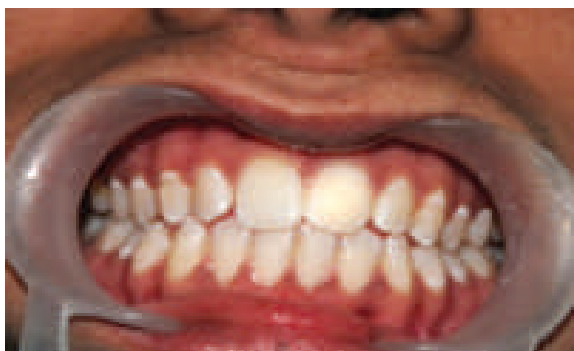


Fig 4 - Postoperative clinical photograph showing composite restoration done with 21.

Discussion

Developmental defects of enamel present a wide range of features. The defects may affect a circumscribed area of one surface of the enamel or, at the other extreme; they may be wide spread, affecting all surfaces of the enamel throughout its full thickness. Similarly the condition may be localized or generalized and may be symmetrical or asymmetrical across the midline of the dentition².

Turner's hypoplasia usually manifests as a portion of missing or diminished enamel, generally affecting one or more permanent teeth in the oral cavity. If it involves anterior teeth, most likely cause is traumatic injuries leading to primary incisors being knocked out or driven into the alveolus affecting the permanent tooth bud.⁸ The traumatized tooth, which is usually a maxillary central incisor, is pushed into the developing tooth underneath it and consequently affects the formation of enamel.

Because of the location of the permanent tooth's developing tooth bud in relation to the primary tooth, the most likely affected area on the permanent tooth is the facial surface. White or yellow discoloration may accompany Turner's hypoplasia⁴. The abnormal discoloration and tooth morphology associated with enamel hypoplasia may compromise esthetics and predispose the affected teeth to dental caries². The enamel hypoplasia causes esthetic problems on anterior teeth resulting to psychologic affect in young patients⁶. Brownish discoloration occurs due to disturbances in ameloblastic layer leading to defective matrix formation caused by traumatic injuries but the stretched inner enamel epithelium continues to induce the differentiation of new odontoblast and hence the dentine formation is not affected⁸. Most defects in enamel present as cosmetic than functional dental problems⁷. In the present case the crown was managed by tooth colored light cure composite which aided in the esthetic management.

Conclusion

The enamel hypoplasia causes esthetic problems on anterior teeth resulting to psychologic effect in young patients⁶. Tooth colored light cured composite is a conservative treatment modality for the affected teeth. Result obtained in this case is very encouraging.

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Prisoners of our genes

Have you ever wondered why there is often a disconnect between an individual's personal fortunes and his emotional status? Why some people, whom the life has treated well and have every reason to be happy, are depressed instead? Why others despite their rotten luck continue to smile and be happy? Until now, we have tried to find answers to these questions in their immediate environment and their interactions with the world around them. But the answers, it now seems, may lie within their genes! In a mega meta-analysis involving 190 researchers, 140 centres and 298,000 subjects, the researchers have identified 3 genetic variants associated with well-being (happiness), two genes associated with depression and 11 genes with neuroticism. The implication is that our emotional status is a genetic predisposition. Our genes determine how happy, depressed or neurotic we are. But other factors like environment and its interactions with genes may also contribute (Nature Genetics, doi:10.1038/ng.3552, published online 18 April 2016)

- Dr. K. Ramesh Rao