

Clinical Management of an Epigenetic Enamel Hypoplasia- A Case Report

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Abstract

Enamel hypoplasia is quantitative enamel defect, having reduced thickness of enamel. Dental enamel defects have been associated with a broad spectrum of etiologies including genetic and epigenetic factors such as systemic (bronchial asthma), local and environmental factors. Partial anodontia can be caused due to environmental and genetic factors which can cause failure of tooth development. The purpose of the case report is to emphasize the possible correlation between bronchial asthma and enamel hypoplasia and the clinical management of the same.

KEYWORDS: Epigenetic; Enamel hypoplasia; Enamel Defects; Bronchial asthma; Partial Anodontia.

Introduction

Enamel hypoplasia is quantitative enamel defect, having reduced thickness of enamel.^{1,2} Enamel formation is a complex and highly regulated process. Permanent human anterior teeth begin calcification at 3-12 months and complete enamel formation by 4-7 years of age. The presence of enamel defects is highly dependent on conditions occurring during critical time of enamel formation and mineralization.

Dental enamel defects have been associated with a broad spectrum of etiologies including genetic and epigenetic factors such as systemic, local and environmental factors. Systemic conditions such as perinatal or prenatal illnesses, low birth weight, regular antibiotic consumptions, malnutrition, celiac disease and respiratory disorders like asthma are associated with enamel defects.³⁻⁷ A rare case of enamel hypoplasia of epigenetic origin with partial anodontia (maxillary right and mandibular left lateral incisors) and clinical management for the same is presented in this case report.

Case Report

A ten year old boy reported to the Department of Pedodontics and Preventive Dentistry with a chief complaint of discolored and stained anterior teeth. Patient's past medical history revealed that he was asthmatic when he was two years old for which he was under medication till the age of four years. He was given asthalin 0.1mg/kg of body weight was given three times a day. Currently he is not under any treatment for bronchial asthma. Intra oral examination disclosed all the erupted permanent teeth were hypoplastic (pitted type) involving incisal and occlusal third and multiple carious deciduous teeth (Fig. a, b, c).



Fig. a. Anterior view showing pitted and stained maxillary and mandibular hypoplastic permanent teeth

Fig. b. Pre-operative maxillary view showing pitted hypoplastic permanent first molars

Fig. c. Pre-operative mandibular view showing pitted hypoplastic permanent first molars

Fig. d. Anterior view showing full composite resin veneer coverage of anterior teeth

Fig. e. Maxillary occlusal view showing Nance palatal arch

Fig. f. Mandibular occlusal view showing lingual arch

Digital orthopantomogram showed the maxillary right lateral incisor and mandibular left lateral incisors were congenitally missing. The patient has no siblings and clinical examination of the parents revealed normal healthy teeth, which rules out any hereditary association. Mixed dentition analysis showed excess space in both arches due to congenitally missing lateral incisors. After complete diagnosis a multistage treatment protocol was formulated and executed.

Since esthetics was the concern, anterior teeth were restored with direct composite veneer restorations (Fig. d). Carious permanent first molars were restored with posterior composites followed by stainless steel crowns. Caries excavation was done for primary canines and restored with composites. The primary posterior teeth were non-restorable and mobile and hence extracted under local anesthesia. Lingual arch space maintainers were given for both arches from implant placement and prosthetic replacement point of view (Fig. e, f).

The patient was recalled after 7-days, 1-month 3-months and 6-months intervals.

Discussion

Children with bronchial asthma demonstrate a higher prevalence of enamel hypoplasia than do nonafflicted children.⁸ The prevalence of dental enamel defects was found to be 11 times higher in permanent dentition amongst Brazilian pediatric patients with asthma.⁹ Some authors observed positive correlation between respiratory disorders, such as asthma, and the presence of demarcated opacities in first permanent molars¹⁰, while other authors have also suggested that asthma is associated with the occurrence of developmental defects of enamel in permanent teeth.^{11,12}

Enamel hypoplasia is a developmental defect of enamel produced by a disturbance in the formation of the organic enamel matrix, clinically visible as pitting, grooves or agenesis. Pitted type of enamel hypoplasia appears as a result of reduction in the amount of enamel matrix formation. This may be because of a primary defect in odontoblast or in ameloblast or may be a result of defective interaction between odontoblasts and ameloblast. Dental enamel pits are associated with tuberous sclerosis, Pitted amelogenesis imperfecta, vitamin D dependent rickets, pseudo hypothyroidism, tricho-dento-osseus syndrome, water fluoridation, respiratory disorders such as asthma. Ameloblast are highly sensitive to oxygen supply and he hypothesized that pediatric asthma with dental enamel defects have probably experienced previous episodes of oxygen deprivation. These defects may be related to the disease itself rather than its treatment⁹ Use of tooth paste containing peppermint can also cause shortness of breath and hence bronchospasm and further oxygen deprivation for the ameloblast. Tooth enamel dust can also be a stimulant for bronchospasm.^{13,14}

In this case it is difficult to pin point the exact etiology of enamel hypoplasia. The fact that the patient's grandparents could have had amelogenesis imperfecta cannot be ruled out. The genes causing enamel hypoplasia could have been recessive in the parent. Hence, the parent's teeth could have been without any enamel hypoplasia.

Hypodontia without any systemic disorders is the mildest and most common phenotype. Second premolars and upper lateral incisors are the teeth most frequently affected. In the present case, congenital absence of maxillary right and mandibular left lateral incisors could be attributed to environmental factor as both parents had normal dentition. Ahmed et al (1998) reported a recessively inherited hypodontia in a large family mapped to chromosome 16q12.1 affected individuals had associated dental anomalies such as enamel hypoplasia, hypocalcification and dentinogenesis imperfecta. Baccetti (1998) included enamel hypoplasia in seven types of dental anomalies including missing teeth.^{15,16}

Conclusion

The presence of bronchial asthma can have deleterious effect on the enamel of the child in an early developmental stage of teeth. Hence it is imperative to take various precautionary measures in a patient with bronchial asthma during this stage. The medications taken for bronchial asthma should be sugar free. Toothpaste containing peppermint should be avoided since it causes further bronchospasm. Routine preventive measures like establishment of dental home at the earliest, periodic topical fluoride applications, meticulous oral hygiene maintenance & sealants placements for posterior teeth should be carried out. More studies should be carried out to correlate the effect of bronchial asthma and enamel hypoplasia.

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