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Enamel Hypoplasia

A Project Submitted To

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By:

Marwa Marwan Mudhafar

Supervised by:

Lect. Meena Osama

B.D.S., M.Sc. (Pediatric dentistry)

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DECLARATION

This is to certify that the organization and preparation of this project has made by the student **Marwa Marwan Mudhafar** under my supervision in the College of Dentistry, University of Baghdad, in the Partial Fulfillment of the Requirement for The Degree of Bachelor of Dental Surgery.

Signature

Lect. Meena Osama

B.D.S., M.Sc.

DECLARATION

I declare that this dissertation was prepared, written, and entirely the result of my own work and I have faithfully and properly cited all sources used in the dissertation.

Signature

Marwa Marwan Mudhafar

2022

DEDICATION

This thesis is dedicated to:

The sake of **Allah**, my Creator and my Master.

My great teacher and messenger, **Mohammed** (May Allah bless and grant him), who taught us the purpose of life.

My great **parents**, who never stop giving of themselves in countless ways, especially my **MOM**, the kindest heart for her continuous support and love.

My **friends** who encourage and support me.

(To all the people in my life who touch my heart, I dedicate this research).

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LIST OF ABBREVIASION

CLP	**Cleft Lip and Palate**
DDE	**Developmental Defects of Enamel**
MIH	**Molar-Incisor Hypoplasia**

INTRODUCTION

Enamel is highly calcified/mineralized tissue in the body, enamel formation requires interplay of several factors which can be genetic or environmental, and any disturbance in this synchronization leads to hypoplasia (Molla *et al*., 2010).

Enamel hypoplasia, thus, is a surface defect of the tooth crown that is caused by a disturbance of enamel matrix secretion, defective calcification or defective maturation (Beijing, 2009).

Enamel hypoplasia or hypo mineralization may be caused by hereditary factors and environmental factors that include systemic factors such as nutritional factors, exanthematous diseases like measles and chicken pox, congenital syphilis, hypocalcemia, birth injury or premature birth, fluoride ingestion or idiopathic causes, and local factors such as infection or trauma from a deciduous tooth (Rajendran *et al*., 2012).

Although it can occur in any permanent tooth, the most commonly involved sites of hypoplasia are the permanent first molars and incisors with specific areas of defect and well-demarcated areas of hypomineralization (Fagrell *et al*., 2011).

Treatment of enamel hypoplasia depends on the severity of the condition and may range from dentist monitor to severe condition affect the esthetic that need filling or crown (Martos *et al*., 2012).

Tooth regenerative agents like fluoride, calcium phosphate agents can prevent further breakdown and halt the carious process which was done for the present patient, diet counseling and establishment of good oral hygiene procedure is done to caries activity, pit and fissure sealants and preventive resin restorations for teeth can arrest caries (Lygidakis *et al*., 2010).

Restorations with glass ionomer cement, composite, stainless steel crowns, full veneer metal-ceramic crowns, fixed-removable partial dentures and or implants are the different treatment options (Arkutu *et al*., 2012).

AIM OF STUDY

The goal of this study is to know the types, causes and treatment options that is suggested for enamel hypoplasia in order to recognize its effect and eliminate its complications especially in early pediatric patient.

REVIEW OF LITERATURE

1. Enamel

Enamel is translucent, and varies in color from light yellow to gray-white, it also varies in thickness, from a maximum of approximately 2.5 mm over working surfaces to a feather edge at the cervical line, this variation influences the color of enamel because the underlying yellow dentin is seen through the thinner regions, enamel is the most mineralized substance in the body, it is the thin outer covering of the tooth, this tough shell is the hardest tissue in the human body, enamel covers the crown which is the part of the tooth that's visible outside of the gums. (Alfred et al., 2020).

Dental enamel serves as the wear-resistant outer layer of the dental crown, it forms an insulating barrier that protects the tooth from physical, thermal, and chemical forces that would otherwise be injurious to the vital tissue in the underlying dental pulp, because enamel has no living cells, the body cannot repair chipped or cracked enamel (Hariri *et al*., 2012).

1.1 Enamel formation

The process of enamel formation is referred to as amelogenesis, enamel development involves two major functional stages, secretory and maturation ,the enamel organ is formed by a mixed population of cells, among these are ameloblasts, which are primarily responsible for enamel formation and mineralization, and form a monolayer that is in direct contact with the forming enamel surface, the formed enamel has a characteristic prismatic appearance composed of rods, each formed by a single ameloblast and extending from the Dentin Enamel Junction (DEJ) to the enamel surface, and the interred enamel located around the enamel rods, by weight mature enamel is ~95% mineral, ~1–2% organic material, and ~2–4% water (Rodrigo *et al*., 2017).

Ameloblasts makes a variety of specialized proteins that guide different steps in enamel production, enamel buzzes with cellular and biochemical activity as it is being made, but within the finished product, cells, proteins and other signs of life have all but vanished (Jeffrey Norris *et al*., 2010).

1.2 Enamel composition

Enamel consists of over 95% (carbonated) apatite, a calcium phosphate mineral that can be found in all mineralized tissues in vertebrates, apatite crystals thereby displaying elongated shapes, in mammalian enamel, these elongated crystals align parallel to one another, effectively forming an enamel rod that can reach tens of micrometers in length, between these rods, space is filled by apatite crystals (Beniash *et al*., 2019).

Table (1) Enamel composition (Sikri, 2017)

Inorganic matter (95-97%)	Calcium 34.5-37.1% Phosphorus 17.1-18% Carbonate 2-2.8% Magnesium 0.2-0.6% Sodium 0.2% Potassium0.05% Iron0.2% Chlorine0.3% Fluoride0.02%
Organic matter (0.2-0.8%)	Glycoproteins Carbohydrate matrix
Water (1.2-4%)	Collagen type 1

1.3 Enamel hypoplasia

Hypoplasia it’s the word from Ancient Greek (hypo-, "under" plasia, "formation"; it is under development or incomplete development of a tissue organ (Peter *et al*., 2004).Enamel hypoplasia is generally defined as a defect in the enamel characterized by a lack of tooth contact, a rapid breakdown of the occlusal surfaces, and a yellowish-brown stain where the dentin is exposed, in hypoplasia the enamel is hard, but it is also thin and deficient in quantity, the condition is a result of defective enamel matrix formation (Louis, *et al*., 2009). Enamel hypoplasia occurs when the growth of the organic matrix, which is subsequently mineralized to form enamel, is disturbed, the resulting defect may take the form of lines, furrows, pits, or large areas of missing enamel (Hillson *et al*., 1997).

Some of the signs of enamel hypoplasia are obvious, but others are more difficult to detect and may not be noticeable until they cause major dental problems, having thin tooth enamel can lead to (Christine Frank *et al*., 2018). :

- pits, tiny groves, depressions, and fissures
- white spots
- yellowish-brown stains (where the underlying layer of dentin is exposed)
- sensitivity to heat and cold
- lack of tooth contact, irregular wearing of teeth
- susceptibility to acids in food and drink
- retention of harmful bacteria
- increased vulnerability to tooth decay and cavities

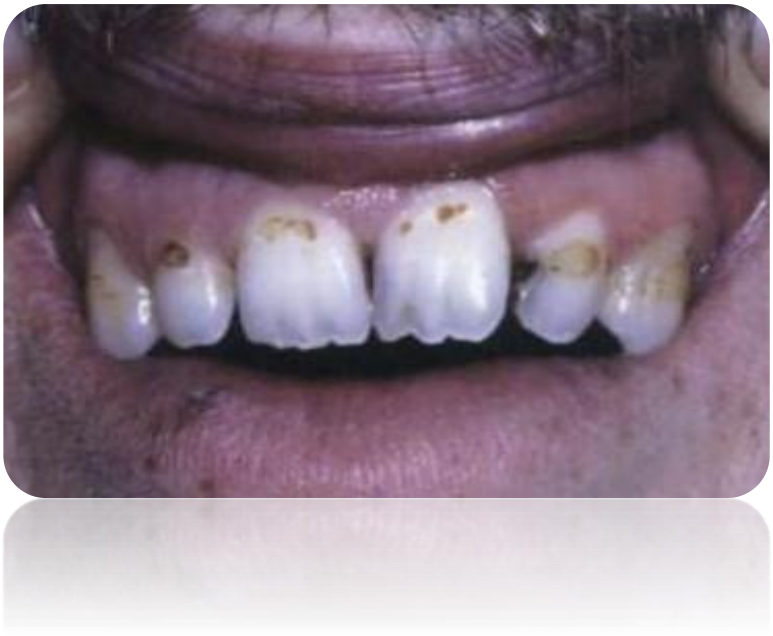

Figure 1: enamel hypoplasia (Leslie *et al*., 2007)

1.4 Enamel hypoplasia prevalence

An observational study in Al-Anbar Province, Fallujah city, was conducted to estimate the enamel hypoplasia in primary and secondary schoolchildren, the enamel hypoplasia was more prevalent in both genders, i.e., boys 122 (9.2%) and girls 117 (8.82%), and the statistical analysis showed that there were no statistically significant differences between boys and girls in the present study (Abdullah , 2018). Aaloosi et al (2020)., in Badra/Iraq studied the prevalence of enamel hypoplasia in primary school children aged from 5-12 years, the results showed that the prevalence of enamel hypoplasia was 0.86% among the observed sample with slightly more than the boys.

Enamel hypoplasia represent 2.04% in examination done in secondary and intermediate schools in Missan Governorate done for ages range from (14-17) years with an equal male: female ratio in each age group (Najm *et al*., 2009).

1.5 Types of enamel hypoplasia

1.5.1 Hereditary type

Only enamel is affected , enamel malformations may come from defects in the genes that encode the proteins related to the mineralization process (simmer and Hu *et al*., 2001) or as a part of generalized familial conditions , such tissue like skin and share the same embryologic origin of neuroectodermal mesenchyme with teeth (Freiman *et al*. , 2009).

1.5.2 Environmental type

Either dentition or single tooth is affected, both enamel and dentine are affected (Wright *et al*., 2015).Enamel hypoplasia is important clinically because it can result in increased caries susceptibility, increased wear, tooth sensitivity and poor aesthetic, this defect considered as an indicator for the child early environmental conditions and may be the child will have a similar effect in the future in the permanent teeth (Slayton *et al*., 2001).

1.6 Causes of enamel hypoplasia

1.6.1 Hypoplasia resulting from nutrition Deficiency

Enamel hypoplasia is associated with malnutrition due to the disturbance of ameloblastic activity during the secretory phase of amelogenesis (Sarnat *et al*., 2000).The severity of the insult determines the extent of defect and the translucency of partially formed enamel (Wong *et al*., 2014).A case of enamel hypoplasia resulting from severe calcium deficiency was reported, the structure of the teeth is influenced by nutrition during tooth development, and such defects occur due to the ameloblasts sensitivity to environmental changes, leading to distinctive bands of malformed enamel (Nanci *et al*., 2008).

1.6.2 Hypoplasia related to brain injury and neurologic defects

Martinez *et al*., (2002) examined 170 children between 4 and 17 years of age (mean age, 12.03 years) with mental retardation and no history of dental trauma, they found that 37% of these children had dental enamel defects.

1.6.3 Hypoplasia associated with allergies

The occurrence of dental enamel defects presented a statistically significant correlation with asthma severity, as well as with symptom onset, the risk being greater in pediatric patients with moderate/ severe asthma, especially in those presenting symptoms before 3 years of age, one group of authors reported that the risk of dental enamel defects is higher in children with poor health during the first three years of life (Wright *et al*., 2006).

1.6.4 Hypoplasia caused by local infection and trauma

Hypoplasia usually affects only one tooth in the mouth and it is referred to as a Turner's tooth, if Turner's hypoplasia is found in the anterior area of the mouth, the most likely cause is a traumatic injury to a primary tooth, 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 (Norén *et al*., 2010).

If Turner's hypoplasia is found on a canine or a premolar, the most likely cause is an infection that was present when the primary tooth was still in the mouth, most likely, the primary tooth was heavily decayed and an area of inflamed tissues around the root of the tooth affected the development of the permanent tooth, the appearance of the abnormality will depend on the

severity and longevity of the infection, white or yellow discoloration may accompany Turner's hypoplasia (Thomson *et al*., 2005).

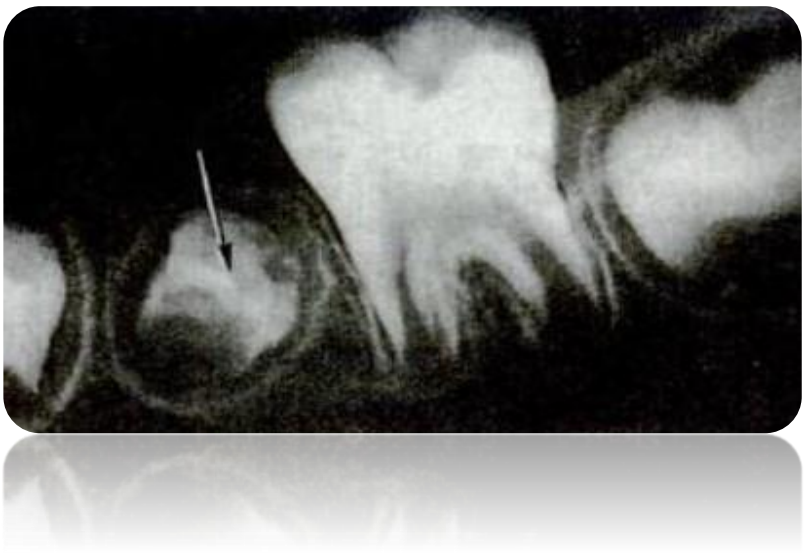

Figure 2: Turner’s tooth (Rajendran, 2009)

Hypoplasia was categorized into the following types by (Silberman *et al*., 2009):

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.

1.6.5 Enamel hypoplasia with cleft lip and palate

Hypoplasia and opacities occurred more frequently in individuals with clefts, especially with alveolar involvement (Patricia Saldias *et al*., 2015)

In the Cleft Lip and Palate (CLP) group, the highest prevalence rate of hypoplasia was observed in the cervical one-third of the tooth (34.8%) (Ajami *et al*., 2017)

Increased incidence of dental enamel hypoplasia in patient with cleft lip and palate is difficult to understand, it is may be as a result of environmental, genetic, nutrition or surgical factors that affect enamel formation (Eslami *et al*., 2013).

1.6.6 Enamel hypoplasia caused by fluoride (dental fluorosis)

Fluoride is one of the most successful measures for prevention of dental caries in public health (Petersen *et al*., 2004). The use of fluorides over the last decades has led to a decreased incidence of dental caries, however, it also resulted in a higher incidence of fluorosis due to greater exposure of individuals to this microelement associated to an increased intake of fluorinated compounds (Ramos *et al*., 2004).

Dental fluorosis occurs as a result of excess fluoride ingestion during tooth formation, the white opaque appearance of fluorosed enamel is caused by a hypomineralized enamel subsurface, with more severe dental fluorosis, pitting and a loss of the enamel surface occurs, leading to secondary staining appearing as a brown color (Monogr *et al*., 2011).Genetic factors have been shown to dictate the severity of enamel fluorosis (Everett *et al*., 2002). The effects of chronic fluoride exposure have also been linked to effects on other tissues and systems (Washington *et al*., 2006).

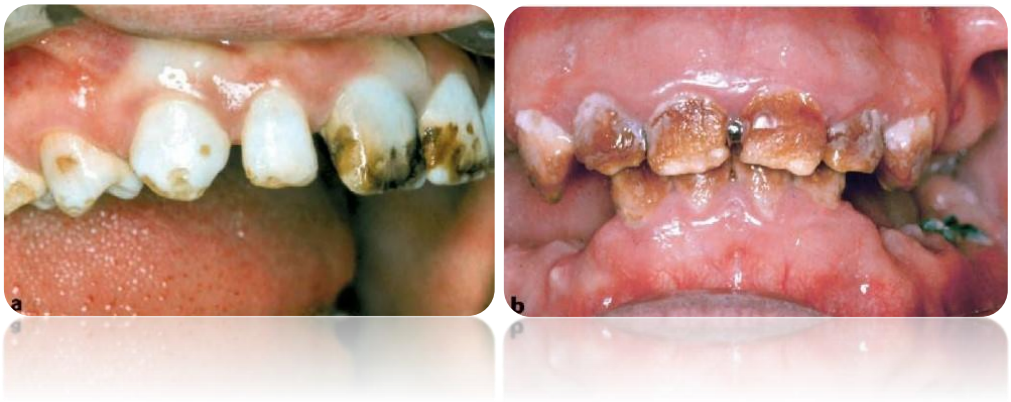

Figure 3: dental fluorosis (a-moderate b-severe) (Pieter, 2007)

1.6.7 Enamel hypoplasia during pregnancy and infants

Prenatal conditions which may be associated with enamel hypoplasia in the child include maternal vitamin D deficiency during pregnancy and neonatal tetany, other antenatal factors which have been shown to contribute to enamel hypoplasia include maternal smoking during pregnancy, increased maternal weight gain during pregnancy and failure to access antenatal care (Ford *et al*., 2009).

Although not commonly encountered in developed countries, nutritional deficiencies in the infant, particularly those associated with insufficient supply and absorption of vitamins A, C and D and calcium are well known to be risk factors for enamel hypoplasia in preterm and Indigenous communities (Roberts-Thomson.*et al*., 2006).

Children born prematurely and those with low or very low birth weight have a higher prevalence of enamel hypoplasia compared to children born at full term with normal birth weights (Thong *et al*., 2007).

1.6.8 Enamel hypoplasia due to certain diseases

Although uncommon in developed countries, congenital syphilis acquired from maternal Treponema pallidum infections can cause enamel hypoplasia or ‘notching’ of the incisor teeth (Stanton, *et al*., 2011)

1.7 Enamel hypoplasia complications

1.7.1 Erosion of enamel

Erosion of enamel by acid from food and drink, as the enamel solubility increases and decreases its minerals so increase the susceptibility to acid attack (Sedano *et al*., 2013).

1.7.2 Dental caries

Teeth with enamel hypoplasia had a significantly higher risk for caries at age 5 and 9 after controlling for other risk factors, enamel hypoplasia appears to be a significant risk factor for caries and should be considered in caries risk assessment (Molars *et al*., 2009).The best way to analyze the incipient caries and hypoplasia is to dry the tooth surface and to examine them under magnification with good lighting, incipient caries are visible when the enamel is dry, but will virtually disappear when the enamel is wet, and hypocalcification remains visible wet or dry (Roberson *et al*., 2002).

Even on relatively intact, non-carious surfaces, penetration of bacteria into porous enamel to sites close to the dentino-enamel junction has been documented (Fagrell *et al*., 2010). Cariogenic bacteria can colonize the porous subclinical Developmental Defects of Enamel (DDE) lesions with relative ease to initiate caries (Seow *et al.,* 2005).

1.7.3 Psychological problems

Psychological problems due to an aesthetic appearance, as the enamel hypoplasia causes tooth discoloration while posterior teeth discoloration may have no great impact on the patient's psychological aspect while anterior teeth discoloration expected to have negative impact on the patient’s quality of life (Folayan *et al*., 2018).As incisor teeth affected by DDE can result in compromised aesthetics due to staining and morphological alterations, children with DDE teeth may experience feelings of anxiety and social embarrassment regarding their dental Appearance (Rodd *et al*., 2011).Furthermore, in many affected children there is increased dental sensitivity due to enamel hypomineralization and exposed dentine (Hong *et al*., 2009).

1.7.4 Tooth loss

Extraction of tooth as a sequence of dental caries, untreated tooth decay can lead to severe pain, root canals or tooth extraction (Sedano *et al*., 2013),

frequent loss of filling and repeated treatment leads to poor prognosis of the tooth (Allazzam *et al*., 2014).

1.8 Management of enamel hypoplasia

In dentistry, the concept of aesthetics is extremely subjective and is related to beauty, harmony and the needs of the patient, the interactions between new restorative materials and techniques allow the reproduction of dental structures, restoring form and function in such a way that restorative procedures become imperceptible (Baratieri *et al*., 2007).

1.8.1 Early detection and prevention of enamel hypoplasia

It should focus on early diagnosis and preventive care, Screening and early detection of DDE and caries is one of the benefits of having a child's first oral examination by 12 months of age (American Academy of Pediatrics, 2008).

Also, as enamel formation of the permanent molars and incisors occur at the same time as the primary molars, the presence of DDE in the primary molars should also indicate a risk for Molar-Incisor Hypoplasia (MIH) in the permanent dentition (Aghareed *et al* 2013).

As DDE predisposes the teeth to increased caries risk and tooth wear, parents need to be informed that teeth with enamel defects are highly susceptible to decay and erosion from acids in foods and drinks (Kazoullis *et al*., 2007).

1.8.2 Crowns as restoration

Stainless steel crowns are the restorations of choice for both primary and permanent molar teeth affected by enamel hypoplasia, as they offer the highest durability and best protection against further breakdown, this method reduces the sensitivity that is frequently encountered in hypoplastic teeth, and helps

preserve tooth structure and maintain space and crown height (Kindelan *et al*., 2008).

1.8.3 Management of enamel hypoplasia in mixed dentition

Patient with mixed dentition, it was found that safer treatment selected for the current patients is: 10% carbamide peroxide, reducing the possibility of sensitivity, this treatment was performed in an individualized way for this patient: using direct applications on the more pigmented areas followed by application with personal trays for 14 days, and only with the affected teeth (Heymann *et al*., 2005).The mock-up was performed to ensure predictability of the result, by using the stratification technique, the opaque shades restored the affected dentin and reconstructed anatomical structures, such as dentin mamelons (Ardu *et al*., 2006).

As to begin the treatment planning the enamel and dentin involvement was decisive for choosing the restorative treatment, the teeth had already completed eruption, an essential factor for the procedure (Ribas *et al*., 2004).

1.8.4 Composite restoration

The composite restoration was chosen to be the more conservative option because it is associated with excellent mechanical, esthetic, and functional properties, however, the imminent difficulty in achieving perfect esthetics because of the amber pigmentation at the stain margin indicated the need for bleaching (Carvalho *et al*., 2010).

1.8.5 Enamel micro abrasion

For superficial enamel stains or defects, enamel micro abrasion is preferred, as it is considered an esthetic and conservative treatment (Sundfeld *et al*., 2014). The main indication for enamel micro abrasion is intrinsic

discoloration or texture alteration due to enamel hypoplasia, amelogenesis imperfecta, or fluorosis (Sundfeld *et al*., 2007). The enamel stain or defect is removed by a combination of the erosive and abrasive effects of the recommended mixture containing low acid concentrations and an abrasive agent, applied mechanically using a low-rotation micromotor (Benbachir *et al*., 2007). As the technique is considered safe and minimally invasive, it can also be combined with tooth bleaching when necessary (Sundfeld *et al*., 2014). Croll *et al* (1986) recommended the use of a mixture of 18% hydrochloric acid, hydrogen peroxide and ether and pumice resulting in a slurry that was applied using a rubber cup coupled to a micromotor and use silicon carbide as an abrasive with different granulations.

Micro abrasion may be utilized in cases of localized or idiopathic enamel hypoplasia that is limited to the outer enamel layer (Benbachir *et al*., 2007; Celik *et al*., 2013). In addition to improving esthetics, it may reduce the need for enamel wear for a restorative approach, which is mainly important in young patients (Reston *et al*., 2011).

Enamel micro abrasion is not indicated if the patient presents deficient lip sealing, as the teeth are always exposed to air and dehydrate more easily, thus a moistened film is not formed under the enamel (Sundfeld *et al*., 2014). The most important factors contributing the success of enamel micro abrasion are the location and depth of the enamel stain or defect (Paic, *et al* 2008). The alteration must be restricted to enamel tissue, without involvement of the dentin (Sundfeld *et al*., 2007). An ideal micro abrasion technique should produce insignificant enamel loss, no damage to pulp or periodontal tissues, and satisfactory and permanent results in a short clinical time without discomfort to the patient (Pavesi *et al*., 2015).

Recommendation:

Preventive advice given to parents should include replacing cariogenic snacks with healthy foods, twice daily tooth brushing, and topical fluoride application, to reduce sensitivity from tooth brushing, a very soft toothbrush and lukewarm water for mouth rinsing may be suggested, topical fluoride is one of the most effective anti-caries agent for teeth affected by DDE, and may be given as neutral sodium fluoride gels or fluoride varnishes applied professionally 3 or 6 monthly or used as daily or weekly rinses in children who are able to expectorate after rinsing (Adair et al., 2006).

CONCLUSION

Enamel hypoplasia is a defect that affects the tooth function and aesthetic and makes it more prone to dental caries, it presents in either dentition due to different causes hereditary or environmental origin

Variety of treatment options are available to treat the defect and restore the tooth structure, with special attention to aesthetic appearance of the teeth as the defect have great impact on patient social and psychological aspects, the severity of the defect with the patient age and economy are largely affected the treatment options.

Goals of treatment is to prevent tooth decay, maintain a good bite, preserve tooth structure and keep teeth looking their best.

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