

Developmental defect of email in children and adolescent: a review

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ABSTRACT Developmental defect of enamel (DDE) is a defect on the surface of the enamel layer that occurs during tooth development due to various genetic and environmental factors. Defects that occur result in changes in color and texture on the enamel surface so that it interferes with aesthetics. The defect level can be classified based on the DDE index to determine the option of a case treatment plan consisting of enamel microabrasion, conservative aesthetic restoration, and bleaching. This review aims to analyze and review studies related to DDE in primary and permanent teeth. An electronic search was conducted through PubMed/Medline, Google Scholar, Semantic Scholar, SpringerLink, and Wiley Online Library using the keywords "defect email", "developmental defect email", and "defect on primary teeth" for research published between January 2011 and April 2020, the same article was eliminated, the initial search resulted in a total of 55 articles. The exclusion of articles was carried out based on the title and abstract so that finally, 45 relevant articles were obtained. Three studies were included in this review for qualitative analysis. The results show that Enamel (DDE) Developmental defects can occur in both primary and permanent teeth with the same prevalence rate.

KEYWORDS: Developmental Defect of Email (DDE), DDE Index, defect to primary teeth, defect to permanent teeth

INTRODUCTION

Developmental defect of enamel (DDE) in the form of hypoplastic patches on the tooth surface is one factor that can affect the aesthetics of the patient's smile.¹ Enamel hypoplasia occurs due to imperfect formation of the organic enamel matrix, usually associated with genetic and environmental factors. Environmental factors include systemic disorders, various viral diseases, nutritional deficiencies, trauma, chemical substances such as fluoride or certain drugs, and idiopathic factors.^{2,3}

Hawas et al.⁴ found that the prevalence of children suffering from DDE in Egypt was 38.9%. In another study, Robles et al.⁵ compared DDE prevalence in primary and permanent teeth. The results showed that email defects were higher in

permanent teeth, namely 52%, compared to primary teeth, 40.2%.

Aesthetic treatments are not only sought after by adults but also by pediatric patients and their parents. According to Welbury and Shaw in 1990, aesthetic problems can affect the psychology of patients, especially adolescents, and can affect their social life.⁶ Clinical procedures that can be used for this condition are bleaching, email microabrasion, restorations with adhesive materials, or a combination of these procedures.^{7,8} Involvement of email and dentin can be a determining factor in treatment choice. Dentists must understand and apply artistic and scientific principles when choosing colors and materials to achieve aesthetic results.⁹⁻¹⁴

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MATERIALS AND METHODS

Focused question

Using the Participants, Intervention, Control and Outcomes protocol described in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement,¹⁵ the following

Selection criteria

The following types of studies were included in this review: (1) literature review, (2) systematic review, (3) meta-analysis, (4) case report, (5) clinical management, (6) cohort study, (7) prevalence, (8) epidemiological studies, (9) restorative approach.

Search methodology

An electronic search was conducted in PubMed/Medline databases, using the Medical Subject Headings (MeSH) terms "developmental defect of email", "enamel defect", "defect of enamel in primary teeth", and the studies published between January 2011 to April 2020. A similar search

research question was constructed: "What is the developmental defect of email?", "What is the influence of developmental defect of email?", and "how can DDE affect primary and permanent teeth?"

Identification, quantification and proposal, molar incisor hypomineralisation, amelogenesis imperfecta, animal studies, and committee reports were excluded.

was conducted via Google Scholar, Semantic Scholar, SpringerLink, and Wiley Online. A secondary search was conducted by reading the reference lists of the articles meeting the inclusion criteria for additional studies relevant to this review.

RESULTS

After eliminating the duplicate items, the primary search produced 55 articles. After excluding irrelevant articles based on abstract and title, full texts of 45 articles were read to exclude additional unrelated studies. Four studies were included in this review for qualitative analysis. The Three quantitative studies selected for this review, published between 2011 and 2020, were analyzed by reading the abstract, objective, analytical data to gather information about the prevalence of developmental defect of enamel (DDE) in primary

and permanent teeth. In these studies, 1 study stated that DDE could affect primary and permanent teeth with the exact prevalence. Developmental defects of email can occur due to various causative factors such as hereditary, acquired, systemic or local factors (**Fig.1**) and, enamel defects occur due to disturbances in the enamel organ during the enamel development process (**Fig.2**)

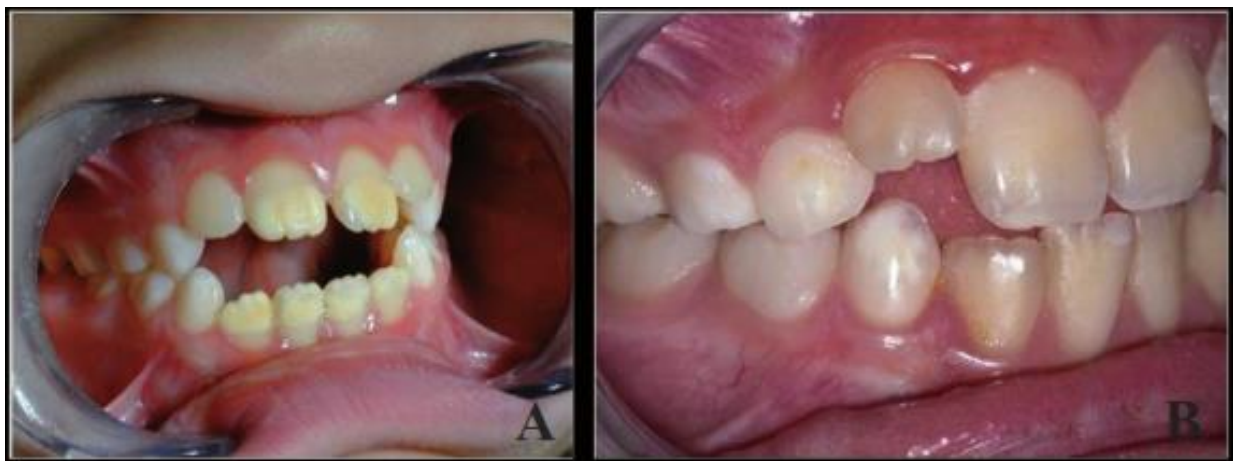


Figure 1. Developmental defect of email; A: well-defined opacity, B: diffuse opacity⁵

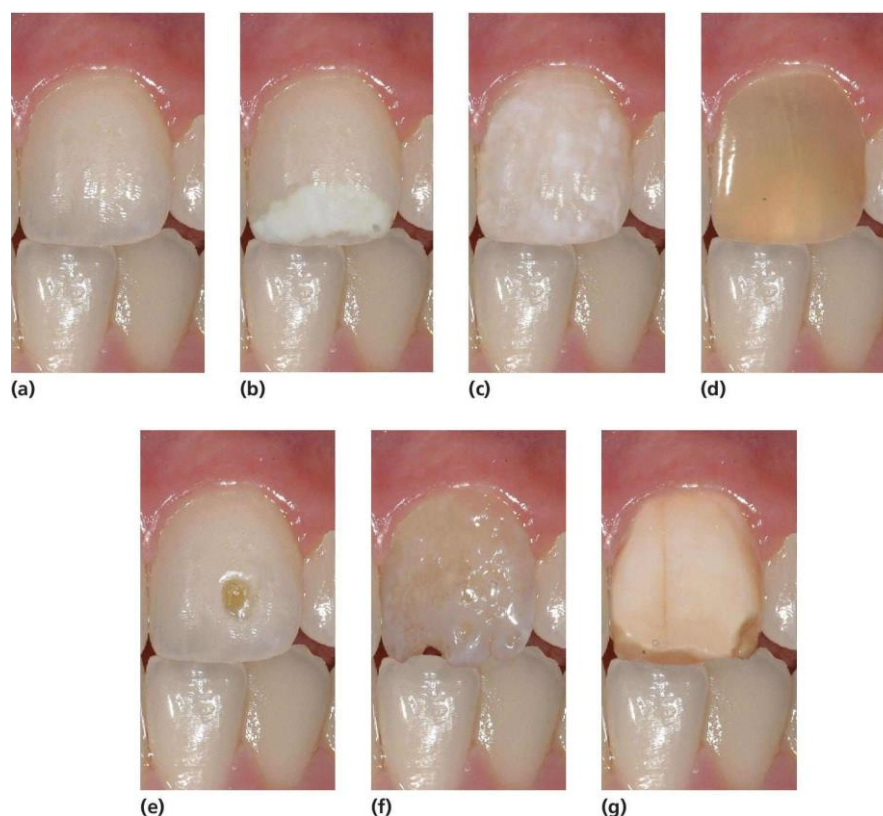


Figure 2. Classification and examples of email defects; (a) normal tooth enamel. (b) well-defined opaque white lesion. (c) diffuse opaque white lesion. (d) email discoloration. (e) single hypoplastic enamel. (f) multiple hypoplastic defects (g) post-eruptive damage due to enamel hypomineralization.¹⁷

One study stated that DDE was more common in primary teeth, while another suggested a higher prevalence in permanent teeth. The general characteristics of the studies are present in **Table 1**. In 1989, Clarkson developed a Modified DDE (MDDE)

Index (**Table 2**), which more efficiently records the prevalence and severity of enamel defects by eliminating and combining several subcategories that existed in the original DDE Index.^{18,20}

Table 1. Research on the prevalence of DDE in primary teeth compared to permanent teeth.

No	Author, year	Sample	Result	Reference
1	Chauhan, D., Chauhan, T 2013	Children aged 9 and 12 Years	DDE in primary and permanent teeth has the exact prevalence	26
2	Casanova et al 2011	Children aged 6 - 12 Years	The prevalence of DDE in permanent teeth was 7.5%, while it was 10.0% in primary teeth.	25
3	Robles et al 2012	Children aged 3 - 12 Years	The prevalence of DDE in primary teeth was 40.2%, while it was 52% in permanent teeth.	5

Table 2. Clarkson's DDE Index Code.¹⁸

A	Defect Type	Code
	Normal	0
	Demarcated opacity	1
	Diffuse opacity	2
	Hypoplasia	3
	mottled hypoplasia	4
	Striped (groove) hypoplasia	5
	Hypoplasia with loss of enamel	6
B	Discoloration	7
	Other types	
	Subtype	
	Demarcated opacity	
	-white/beige	1
	-yellow/brown	2
	Diffuse opacity	
	Diffuse stripes	1
	Diffuse patchy	2
	Diffuse confluent	3
	Coloring by code 2 or 3	4
	mottled <2 mm with code 2 or 3	5
	mottled >2 mm or enamel loss with code 2 or 3	6
C	Widespread defect (a surface area involved)	
	Less than 1/3	1
	At least 1/3 to less than 2/3	2
	At least 2/3	3

DISCUSSION

Developmental defect of enamel (DDE) is a deviation in the quality and quantity of enamel caused by disruption and damage to the enamel organ. The severity of the defect usually depends on the developmental stage and its duration. Enamel hypoplasia is a quantitative defect and appears as an enamel matrix deficiency. In contrast, enamel hypomineralization is a qualitative enamel deficiency that shows a change in the transparency or opacity of the enamel that can occur diffusely or demarcated. The defect can also be white, yellow, or brown.¹⁶

Diffuse opacity in enamel is thought to be associated with groups of teeth undergoing enamel maturation at the time of systemic disturbance, while well-defined opacity is more commonly found in teeth with localized and transient injuries. The DDE index simplified by Clarkson et al.¹⁸ is one of the most popular indexes. However, many other indices, such as Dean and Thylstrup Index and fluorosis Index by Fejeskov.^{16,18} Theoretically, enamel defects occur due to disturbances in the

enamel organ during the enamel development process the enamel structure does not undergo remodeling. Determining the exact time of occurrence of these disorders is difficult due to a lack of knowledge regarding the chronological timing of the different stages of amelogenesis as well as individual variations in enamel formation.¹⁶

Enamel defects can occur due to heredity, both conditions involving only tooth enamel and generalized systemic syndromes. Hereditary diseases that only affect tooth enamel are known as amelogenesis imperfect. Defects in this condition can cause enamel hypoplasia, hypomineralization, or hypomaturization. Abnormalities of genes involved in amelogenesis respond precisely to this defect. Children with imperfect amelogenesis have a DDE characteristic that occurs in both the primary and permanent teeth. Other hereditary diseases that give oral manifestations in enamel hypoplasia are Usher syndrome, Seckel, Ellis van Creveld, Treacher-Collins, Velocardiofacial, and Heimler syndrome.¹⁶

Systemic conditions or locally acquired diseases during antenatal, perinatal, or postnatal development can result in DDE. Prenatal conditions usually cause DDE localized to prenatal enamel formation, while defects resulting from disorders of the postnatal period are found in postnatal enamel development. Systemic disturbances during the birth usually result in excess "neonatal lines" that are clinically seen as enamel hypoplasia of the primary dentition. The condition can be associated with trauma or premature birth. DDE found in infants with low birth weight, and premature can risk various systemic diseases such as respiratory tract diseases, cardiovascular defects, gastrointestinal disorders, hematologic problems, intracranial bleeding, anemia, and renal defects. In addition, DDE can also be caused by metabolic conditions, infection, or environmental factors such as exposure to chemicals.¹⁶

Prenatal conditions associated with enamel hypoplasia in children include maternal vitamin D deficiency during pregnancy and neonatal tetanus. Other antenatal factors are smoking habits during pregnancy and lack of access to health care during pregnancy. Nutritional deficiency in infants, primarily related to the supply and absorption of vitamins A, C, and vitamin D and calcium, is also a risk factor for enamel hypoplasia. Disorders of vitamin D metabolism, inadequate mineral levels, and the gastrointestinal tract's inability to absorb various minerals are essential factors in developing DDE in children due to metabolic immaturity and the mineralization system. Infectious diseases caused by bacteria and viruses such as urinary tract infections, otitis, and upper respiratory tract diseases are DDE. Viral infections such as chickenpox, rubella, measles, mumps, and influenza are also associated with DDE in primary and permanent dentition. Enamel hypoplasia is also found in children with cerebral palsy caused by maternal or fetal infection, fetal anoxia, and hyperbilirubinemia.¹⁶

Local factors such as trauma and infection have also been associated with hypoplasia of the enamel on the teeth surrounding trauma or disease, in contrast to systemic factors that usually involve all developing teeth in the arch, for example, trauma resulting from endotracheal intubation or laryngoscopy of the developing tooth germ. It will cause damage to the ameloblasts and result in opacity or hypoplasia. Another case is a well-defined opacity commonly found on the labial surface of primary canines caused by local trauma caused by the thin buccal cortical bone above the canines.¹⁶

After this, the DDE index, the Developmental Defect of Enamel (DDE) index, has been known since the 1930s, including fluorosis. This Index is divided into two main groups: the specific fluorosis index and the descriptive Index covering all enamel defects. The fluorosis index is designed to measure enamel defects that occur simply due to excessive fluoride consumption, usually described by the presence of patches of enamel or fluorosis. The most widely used Index in this group is the Index of Dean et al.¹⁹. When using Index Dean, the examiner must decide whether the defect is caused by excessive fluoride intake. Fluoride-induced enamel defects are thought to have some characteristics, namely discoloration with an opaque white color and their overall distribution in the dental arch.¹⁸

Another index introduced by Thylstrup and Fejerskov in 1978 to measure fluoride-derived enamel defects such as the Dean index. In this Index, the clinical features of various defects are associated with the histologic features of the affected enamel. The diagnostic criteria for the multiple levels in this Index are such that only defects due to fluoride will be included (Table 2).¹⁸

Since discovering the DDE index, various studies have been carried out regarding the prevalence of DDE in both primary and permanent teeth. Hawas et al.⁴, in 2014, conducted a study on children aged six to fourteen years related to enamel defects. This study showed that out of a thousand children studied, 38.9% of children had enamel hypoplasia or hypo calcification of enamel, while 61.1% of children were free from enamel defects.⁴ In another study, Jalevik et al.²¹ examined enamel defects that occur in permanent teeth caused by trauma to the previous primary teeth and a history of systemic disease in children. Ravindran et al.²² conducted a similar study on DDE. The study results reported that the prevalence of DDE was 32%, with a greater prevalence in preschool children living in cities than preschool children living in villages.^{4,21,22}

Jacobsen et al.²³ conducted a study from a different perspective by looking at the prevalence of DDE in the teeth of children aged 6 to 10 years exposed to anti-epileptic drugs prenatally. This study showed that children exposed to anti-epileptic drugs prenatally had a higher prevalence of enamel hypoplasia, namely 11%, than the control group with a value of 4%. Meanwhile, the defect in diffuse opacity was 18% in the studied group and 7% in the control group.²³ The prevalence of DDE can also be associated with dental caries and nutritional status as in a study conducted by Yadav et al.²⁴ The results of this study indicate that the prevalence of DDE of

39.9% has a positive relationship with dental caries in the group studied with an average DMFT index of 3.5.²⁴

Research on the prevalence of DDE in primary teeth has not been carried out as often as in permanent teeth.¹⁶ Studies related to DDE prevalence in both permanent and primary teeth have been separately studied by many researchers from different countries and perspectives. Robles et al.⁵ tried to compare DDE prevalence in primary and permanent teeth. This study reported that the prevalence of DDE was 40.2% in primary teeth and 52% in permanent teeth, while Casanova et al.²⁵ reported that the majority of DDE in permanent teeth was 7.5%. In comparison, in primary teeth, it was 10.0%. Chauhan²⁶ suggested that both immediate and permanent teeth have the exact prevalence of DDE.

Early diagnosis and preventive treatment are critical to the successful treatment of DDE. Children with a family history of amelogenesis imperfecta or specific medical syndromes associated with enamel defects such as epidermolysis bullosa, cerebral palsy, or premature birth should be examined as soon as possible after the eruption of the first tooth to detect an enamel defect in the permanent dentition. Formation of enamel from permanent molars and incisors occurs at the same time as

primary molars. Hence, the presence of enamel defects in primary molars indicates the risk of defects in permanent teeth.²⁷

Children with severe enamel defects usually require interdisciplinary management involving general, pediatric, and orthodontists. The treatment plan includes complex restoration, orthodontic treatment, exodontia, and prosthodontics. Enamel defects that weaken the tooth structure can cause fracture or damage to the edges of the restorative material under high chewing pressure, resulting in repeated caries and further caries involving the pulp. Such as resin-modified glass-ionomer cement and polyacid-modified composite resin.²⁷

Localized enamel defects that occur in anterior teeth can affect aesthetics, causing different effects in the form of psychological changes in decreased patient confidence when smiling to affect their social life.^{28,29} In some instances, some cosmetic dentistry procedures should be avoided, and invasive treatment in enamel hypoplasia sometimes has to be postponed in advance. Still, the patient's complaints of staining spots must be treated. In less severe cases, bleaching treatments can give good results. The involvement of enamel and dentin determines the treatment to be chosen.^{7,23,27,30,31}

CONCLUSION

Email defects disrupt the apposition and mineralization of tooth enamel and can cause manifestations in the form of enamel hypoplasia or enamel opacity. Disorders of enamel development can occur due to genetic, systemic, local factors, environmental factors, or other factors. Treatment of the enamel defect depends on the lesion's degree of involvement and severity. The treatment approach

consists of enamel microabrasion, aesthetically conservative restoration, and bleaching. The results showed that DDE could occur in primary and permanent teeth with the exact prevalence. Research on the majority of DDE in primary teeth compared to permanent teeth is still very minimal, so other studies are needed from various aspects.

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