Developmental Defects of Enamel : an increasing reality in the everyday practice

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Abstract

Developmental defects of enamel (DDE) are daily encountered in clinical practice. DDE are alteration in quality and quantity of the enamel, caused by disruption and/or damage to the enamel organ during the amelogenesis process. Several clinical indices have been developed to categorize enamel defects based on their nature, appearance, microscopic features or their cause. The aetiology of DDE is not completely clear. Enamel fluorosis is a hypo-mineralization of enamel characterised by subsurface porosity as a result of excess fluoride intake during the period of enamel formation. Several types of treatment have been reported, related to the degree of enamel defect. Correct diagnosis according to lesion depth and prognosis of the technique are fundamental factors in the treatment decision-making process.

Keywords: *Developmental defects of enamel (DDE), enamel hypoplasia, enamel hypomineralization, fluorosis, minimal invasive dentistry.*

Introduction

Developmental defects of enamel (DDE) are daily encountered in clinical practice. DDE are alteration in quality and quantity of the enamel, caused by disruption and/or damage to the enamel organ during the amelogenesis process. The stage of development in which the insult occurs, its duration and extent, determine the clinical appearance of the defect. Enamel hypoplasia (EH) is a quantitative defect and it's a deficient thickness of enamel while enamel hypomineralization (EO) is a qualitative deficiency and is presented as alterations in the enamel translucency or opacity. The opacity defects may be diffuse (DIO) or demarcated (DEO) and coloured white, yellow or brown [1,2]. DDE can have a significant impact on oral health, aesthetics of the smile, tooth sensitivity and altered occlusal functions [3,4]. Enamel defects are also risk indicators for dental caries and erosion in children [5 ,6].

Epidemiology

Epidemiologic data show a high prevalence of DDE both in primary and permanent dentition, reflecting the current increasing trend of this condition, which should be considered as a public health problem .

Over the last 60 years, a large number of surveys have reported on the prevalence of DDE in a variety of populations [7,8,9,10]. These studies used different terminologies and classifications and direct comparison of the results can't be done. To uniform the nomenclature, the Commission on Oral Health Research and Epidemiology of the ''Federation Dentaire Internationale'' has proposed an Epidemiological index of developmental defects of enamel: DDE Index [11,12]. Different indices have been proposed for specific types of DDE, such as the Dean and Thylstrup and Fejeskov (TF) and indices of fluorosis [13, 14], but the DDE Index,

often used in a simplified form, nowadays is one of the most popular [15].

Epidemiological Indices

Several clinical indices have been developed to categorize enamel defects based on appearance, microscopic features or cause. Direct comparisons of the findings of population surveys of enamel defects (including fluorosis) are impossible due to different classifications and indices. The latter can be divided into: a) specific fluorosis indices, which identify and categorize only dental fluorosis; and b) descriptive indices, which make no etiological assumption. The Dean, Thylstrup and Fejerskov, and Tooth Surface Index of Fluorosis (TSIF) indices are the most commonly used fluorosis indices and they require a diagnosis of fluorosis at the clinical examination. Of the descriptive indices, with no etiological assumption, the Al-Alousi and the Developmental Defects of Enamel (DDE) indices are the most commonly used for record the enamel defects.

Table 1. Dean's Index

Normal	**Enamel is smooth and uniform in colour**
Questionable	Enamel may exhibit some white flecks or small white spots. These are cases where there is not definitive fluorosis, but teeth do not qualify as "normal" either.
Very Mild	Less than 25% of the tooth surfaces displays irregular white areas. Often these include cases where there are 1-2 mm of the tooth surface just at the cusp tips are affected
Mild	More than 25% of the tooth surface but less than 50% is affected
Moderate	Generalized areas of hypocalcification on all surfaces of the tooth, may exhibit attrition on susceptible tooth surfaces and brown spots may be present.
Severe	Generalized pitting of the enamel on all surfaces,generalized brown discolorations, tooth shape may be affected as well

Dean's Index

The Dean's Index [13] measures dental fluorosis. H. T. Dean created it in 1934 in an attempt to identify if fluorosis was a health problem that needed to be addressed (National Research Council, 1993). Originally the index had seven categories: normal, questionable, very mild, mild, moderate, moderately severe, and severe. Later, in 1942 he combined the moderately severe and severe categories into one category for severe. This index is commonly used today (NRC, 1993). The criteria for each category are as reported in Table 1.

This index is performed without drying the teeth. Patients are assessed using the scale, and then classified based on the two most severely affected teeth. For example, if someone presents with two teeth moderately affected, but the rest are normal, they would still be classified as "moderate."

Table 2. The TF Index

Score	Criteria
0	Normal translucency of enamel remains after prolonged air-drying.
1	Narrow white lines corresponding to the perikymata. [Dean = Questionable/Very Mild]
2	Smooth surfaces: More pronounced lines of opacity that follow the perikymata. Occasionally confluence of adjacent lines. Occlusal surfaces: Scattered areas of opacity <2 mm in diameter and pronounced opacity of cuspal ridges. [Dean = Questionable/Very Mild]
3	Smooth surfaces: Merging and irregular cloudy areas of opacity. Accentuated drawing of perikymata often visible between opacities. Occlusal surfaces: Confluent areas of marked opacity. Worn areas appear almost normal but usually circumscribed by a rim of opaque enamel. [Dean = Very Mild/Mild]
4	Smooth surfaces: The entire surface exhibits marked opacity or appears chalky white. Parts of surface exposed to attrition appear less affected. Occlusal surfaces: Entire surface exhibits marked opacity. Attrition is often pronounced shortly after eruption. [Dean = Mild/Moderate]
5	Smooth surfaces and occlusal surfaces: Entire surface displays marked opacity wtih focal loss of outermost enamel (pits) <2 mm in diameter. [Dean = Severe]
6	Smooth surfaces: Pits are regularly arranged in horizontal bands <2 mm in vertical extension. Occlusal surfaces: Confluent areas <3 mm in diameter exhibit loss of enamel. Marked attrition. [Dean = Severe]
7	Smooth surfaces: Loss of outermost enamel in irregular areas involving <1/2 of entire surface. Occlusal surfaces: Changes in the morphology caused by merging pits and marked attrition. [Dean = Severe]
8	Smooth and occlusal surfaces: Loss of outermost enamel involving >1/2 of surface. [Dean = Severe]
9	Smooth and occlusal surfaces: Loss of main part of enamel with change in anatomic appearance of surface. Cervical rim of almost unafffected enamel is often noted. [Dean = Severe]

There is a lack of distinction between patients with fewer or more affected teeth. For this reason, this index is preferably used for collecting more generalized data about communities and regions in order to assess prevalence rather than for more specific cases. Dean Index is still widely used in surveys of fluorosis, including the CDC's national surveys of fluorosis in the United States, and its

continued use is important for historical comparisons. For more specific data, the TF (Thylstrup-Fejerskov) Index was developed.

Thylstrup-Fejerskov Index

TF Index (Table 2) is specifically a fluorosis index: it classifies nine types of fluorosis. The Thylstrup–Fejerskov (TF) index grades dental fluorosis in terms of its absence (TF 0), of opaque lesions presence (TF 3), when affecting all the vestibular enamel and producing the appearance of white chalk (TF 4). In more advanced stages, there is a continuing loss of enamel and anatomical dental deformities (TF 5–9) [16].

Tooth Surface Index of Fluorosis" ("TSIF")

Horowitz et al. (1984) developed a fluorosis index based on aesthetic features of affected enamel surface (TSIF). Two values for anterior tooth surface not restored (buccal and lingual) and three values for posterior tooth surfaces (buccal, lingual and occlusal) are assessed (Table 3).

Martinez-Mier EA, Soto-Rojas AE evaluated dental fluorosis prevalence using TSIF index: "Of the 62.5 percent of the White children (from Indianapolis, Indiana) who presented with dental fluorosis upon examination, 41.3 percent had a maximum score of 1 and only 21.2 percent of the children had a maximum score of 2.

Of the 80.1 percent of African American children who had dental fluorosis, a maximum score of 1 was assigned to 50.5 percent of the children, 15.4 percent were assigned a maximum score of 2, 1.5 percent had a maximum score of 3, and 12.7 percent were assigned the highest score of 5. Differences in severity were also statistically significant ($P < 0.001$)" [19].

DDE index

The DDE index allows recording of a wide-ranging variety of defects, with no attributing of etiology. Defects can be: demarcated opacities, diffuse opacities, or hypoplasia (or combinations). This descriptive classification is more appropriate than a fluorosis–specific index: it records both non-fluoride and fluoride- induced defects, and it does not require non-fluoride defects exclusion (which can be a difficult decision) [20]. However, its use is slow and time-consuming, expecially when a large number of defects are present. Diffuse opacities of enamel are the characterising features of the teeth of children in fluoridated areas. Unfortunately, the characteristics of dental fluorosis are not unique: there is also the possibility that some opacities may be idiopathic. This implies that, while fluoride-induced lesions are usually found within the diffuse opacities type, not all diffuse opacities may necessarily be caused by fluoride. No studies have directly compared results of the DDE index and Dean's index, although direct comparison has been made of the TF index and the DDE index, with good agreement reported [21].

Table 3. The TSIF Index

Score	Criteria
0	Enamel shows no evidence of fluorosis.
1	Enamel shows definite evidence of fluorosis, namely areas with parchment-white color that total less than one-third of the visible enamel surface. This category includes fluorosis confined only to incisal edges of anterior teeth and cusp tips of posterior teeth ("snowcapping").
2	Parchment-white fluorosis totals at least one-third of the visible surface, but less than two-thirds.
3	Parchment-white fluorosis totals at least two-thirds of the visible surface.
4	Enamel shows staining in conjunction with any of the preceding levels of fluorosis. Staining is defined as an area of definite discoloration that may range from light to very dark brown.
5	Discrete pitting of the enamel exists, unaccompanied by evidence of staining of intact enamel. A pit is defined as a definite physical defect in the enamel surface with a rough floor that is surrounded by a wall of intact enamel. The pitted area is usually stained or differs in color from the surrounding enamel.
6	Both discrete pitting and staining of the intact enamel exist.
7	Confluent pitting of the enamel surface exists. Large areas of enamel may be missing and the anatomy of the tooth can be altered. Dark brown stain is usually present

DDE modified Index

The Modified DDE Index [22] is a descriptive index derived from the original DDE Index [23], considered more practical and comparable in epidemiological studies [24]. DDE Modified Index allows for efficient recording of prevalence and severity of enamel defects. It divides defects into three types: demarcated, diffuse and hypoplastic. The diffuse opacity category probably contains most of the fluoride-related opacities. However, this group encloses some non-fluoride opacities and no effort is made to differentiate them. The modified version of the DDE index suggested that the defect extent should be recorded in thirds of the tooth surface and that a size of 1 mm in diameter should be used to distinguish between normal and abnormal enamel (Table 4).

Etiology

The aetiology of DDEs is not completely clear. Genetic and hereditary factors such as amelogenesis imperfecta are involved, along with acquired systemic and

environmental factors such as fluoride intake, medications, nutritional deficiencies, prenatal infections, chicken pox or other early childhood diseases [25, 26, 27, 28].

The importance of socioeconomic factors is evident: DDE is less prevalent in developed countries with good nutrition. Comparing the clinical features of the defects can provide insight into the different response of ameloblasts to environmental insults in primary and permanent dentitions, and thereby facilitate the identification of etiological agents.

Table 4. The DDE modified Index

Basic Type of DDE	Subtype of DDE
Demarcated opacities (DEO)	demarcated opacities (white/cream)
	Demarcated opacities (yellow/brown)
Diffuse opacities (DIO)	Diffuse opacities lines/patchy
	Diffuse opacities confluent
	Confluent/patchy stain gloss of enamel
Hypoplasia (EH)	Hypoplasia pits
	Hypoplasia missing enamel
Discolouration	

Hereditary conditions

Enamel defects can be the presenting feature in a hereditary condition or a component of a generalized systemic syndrome. Inherited conditions that involve enamel only, are known as amelogenesis imperfecta and the defects may vary from enamel hypoplasia to hypomineralization or hypomaturation. Abnormalities of the amelogenesis involved genes are primarily responsible for these defects [29,30].

Children with amelogenesis imperfecta present DDE in both dentitions. Many hereditary syndromes present enamel hypoplasia: Usher syndrome [31], Seckel syndrome [32], Ellis Van Creveld [33]. DDE have also been associated with the Treacher-Collins syndrome [34], oto-dental syndrome [35], 22q11 deletion syndrome (also known as velocardiofacial syndrome)[36,37], and Heimler syndrome [38].

Acquired conditions

Systemic disruptions (traumatic or preterm births, as well as metabolic or infectious conditions or environmental exposure to toxic chemicals) around the time of birth often result in amplified neonatal line, clinically visible as EH, in the primary dentition [39]. The neonatal line marks the transition from intrauterine to extrauterine life, separating the prenatally formed enamel from the post-natally formed one [40]. Other prenatal conditions associated with EH in the child are maternal vitamin D deficiency and neonatal tetany [41], also maternal smoking, increased maternal weight gain during pregnancy [42, 43]. Multiple, preterm and low weight births are risk factors for DDE due to the higher rate of neonatal complications [44]. Altered mineralization patterns associated with hypocalcaemia, osteopaenia, rickets and hyperbilirubinaemia are linked with the primary dentition DDE aetiology. Malabsortive disorders such as Coeliac disease is another condition where malabsorption and mineral deficiencies resulting from the gut enteropathy caused by gluten intolerance can cause DDE. Enamel defects encountered in coeliac disease have been proposed as a possible diagnostic sign of 'silent' coeliac disease in children [45, 46]. SM analysis of hypoplastic teeth from children with coeliac disease has evidenced less mineralization and more irregular enamel organization [46].

Table 5. Treatment options for teeth with DDE

1	Do nothing: if the patient or parent is unconcerned about the appearance of the teeth
2	Resin infiltration technique
3	Treatment with CPP-ACP products +/- bleaching
4	Bleaching: usually home bleaching
5	Prolonged bleaching
6	Microabrasion
7	Megabrasion: remove the white area with a handpiece prior to composite bonding
8	Composite bonding to mask quantitative defects
9	Porcelain veneers
10	Crowns if the lesions are severe

Many chemicals and drugs have the potential to damage ameloblasts and cause DDE. Children with levels of fluoride greater than 1 ppm [47], environmental exposure to high lead level, accidental or pica ingestion have been reported to show HE of pitting variety [48]. Tetracycline and more recently also amoxicillin have been connected to HE and dental discolouration [49,50].

Local factors such as trauma and infections have also been associated with enamel hypoplasia of the teeth in the immediate vicinity of the damage, in contrast to systemic factors, which usually affect all developing teeth in the jaws [51].

Fluorosis

Enamel fluorosis is a hypo-mineralization of enamel characterised by greater surface and subsurface porosity than in sound enamel as a result of excess fluoride intake during the period of enamel formation [52].

It has also been defined as 'a dose response effect caused by fluoride ingestion during the pre-eruptive development of teeth'.

The changes in enamel appearance range from fine white lines to pitting or staining of enamel.

Water fluoridation and enamel fluorosis

First use of fluoride in water for caries control was in 1945-1946 in the United States and Canada. Then water fluoridation was introduced in Dublin in 1964 [53]. Today approximately 317 million people in 39 countries benefit from artificially fluoridated water [54]. It was assumed that fluoride needed to be present systemically to be incorporated into enamel during enamel formation. Later work using sophisticated enamel biopsy and fluoride analysis techniques revealed no simple relationship between enamel fluoride levels and caries experience. It became apparent that reduced enamel solubility is not the only factor involved in the cariostatic action of fluoride [55-56].

In recent years the level of enamel fluorosis is increasing. A study assessing the decrease in dental caries in Belgium among 12-year-old children documented an increase in fluorosis from 5% to 30% between 1983 and 1998 [57]. Fomon et al [58] recordered an increase in fluorosis in the US over the previous 30 years both in fluoridated and non-fluoridated communities. Mann et al [59] found that primary tooth fluorosis was closely associated with fluorosis in the permanent dentition. Children with fluorosis of their primary second molars were 1.86 times as likely to develop fluorosis in their permanent incisors than those without primary molar fluorosis [60].

The Forum of Fluoridation 2002 [61] reiterated that the only other risk associated with water fluoridation is enamel fluorosis. The Dean studies [62] observed the maximum caries reduction in a community at 1 ppm fluoride in domestic water supplies. At this concentration 1% mild fluorosis, 19% very mild and 31% with questionable fluorosis were expected: 51% with some degree of fluorosis and 49% with no change in the appearance of the tooth enamel. It was decided that this level of risk (fluorosis) was tolerable taking into account the reduced caries levels. Recently, however, there is evidence throughout the world that the enamel fluorosis prevalence is increasing and in many cases the levels are above those reported by Dean. The recent systematic review of water fluoridation, the 'York Review' [63] concluded that dental fluorosis of aesthetic concern affected 12.5% of residents of fluoridated communities.

Fluoride Metabolism and Enamel Fluorosis

Many hypotheses exist to explain the mechanism of fluorosis. There is some evidence that excessive levels of fluoride can interfere with dental enamel formation and cause fluorosis [64].

Fluoride effects are in apatite crystals size increase, apatite crystallinity improvement and driving force towards apatite nucleation and growth increase [65, 66]. Retention of amelogenins in the early maturation stage characterize fluorosed enamel. Scanning and electron micrographic studies have shown alterations in crystallite morphology and crystal defects [67]. The chronology of teeth calcification in permanent and temporary dentition can indicate when fluoride over-exposure can be dangerous to amelogenesis. The fluoride over-exposure results in enamel hypomineralization. Hypomineralization severity depends on dose, timing and duration of the fluoride intake [68]. Evans et al [69] determined the critical time frame during calcification when enamel is most vulnerable to developing fluorosis. The greatest risk was associated with a 4-month critical period starting at 22 months after birth. The authors concluded that fluoride exposure during the months prior to this period carry less risk than continued exposure for up to 36 months beyond this critical time. Evans et al [70] indicated as the most critical period for developing dental fluorosis of the permanent central incisors between 15 and 24 months for males and 21–30 months for females. In 1993, Evans developed the Chronological Fluorosis Assessment (CFA) Index to examine the chronological development of enamel fluorosis [71]. Fluoride level in water has remained relatively stable and the increase in fluorosis can be correlated with improved consumption of fluoride-containing products by children <6 years [72, 73, 74, 75]. Fluoridated toothpastes have been introduced in Europe in the 70'ies, today they are more than 90% of all. They contain no more than 1500-ppm fluoride, but children swallow toothpaste, increasing fluoride intake. It is dangerous for children to use floridated toothpaste before 2 years old.

Diet supplement

Pendrys and Katz [76] suggested that mild-to-moderate fluorosis was strongly associated with fluoride supplementation during the first 6 years of life. A daily fluoride intake in excess of 0.1-mg/kg/body weights would give rise to enamel fluorosis. These Subjects had a 28-fold increase in the risk of fluorosis as compared to unexposed ones.

Treatment options for teeth with DDE with increasing intervention

Several types of treatment have been reported, related to the severity of enamel defect. Resin infiltration technique [77], CPP-ACP with or without bleaching [78], tooth bleaching [79], microabrasion [80] and remineralization therapy [81] represent a minimally invasive approach [82] in enamel stains removal and masking, and minor enamel surface defects treatment. Enamel defect in quality and quantity can be treated with direct composite resin restorations and produces excellent aesthetic results and stable clinical longevity [83]. In the most severe cases, porcelain veneers appear to be the best option [84].

Bleaching can be one of the first therapeutic options. It will remove orange, brown and yellow pigmentation from the surface of the enamel. Then the background colour of the tooth is lightened and the white lesions start to fade. Sometimes it's necessary to undertake home bleaching treatments for a prolonged period of time. The normal period for home whitening of upper and lower teeth is normally about two weeks for the upper and three weeks for the lower. The treatment time may vary depending on the degree of discolouration.

The resin infiltration technique requires no mechanical enamel removal: only 30 to 40 µm are eroded while enamel microabrasion is around 360 µm. This technique leads to a good, real and fast improvement in labial tooth surface appearance.

CPP-ACP supplementation has been shown to be effective in remineralization of the affected enamel, resulting in an aesthetic improvement.

Microabrasion is a chemical technique to simultaneously erode and abrade the enamel surface of a tooth to remove the brown and white spot enamel. Normally it's associated with a course of bleaching treatment. The materials for microabrasion technique use a compound of hydrochloric acid (10%-18%) and flour of pumice.

Ardu suggested a modification of this technique:
using a combination of the microabrasion paste which hydrochloric acid followed by daily home application of casein phosphopeptide-amorphous calcium phosphate complexes (CCP-ACP)[85].

Conclusion

To satisfy new demands regarding tissue conservation, function, and aesthetics, treatment parameters must be redefined for all kind of smile deficiencies concerning especially young patients with healthy dentitions, particularly if following orthodontic treatment. A more comprehensive case analysis including long-term prognoses should be taken to offer the patient the best available solution with minimal tissue sacrifice. A global and reasonable treatment approach should include preventive issues, bleaching techniques, microabrasion, recontouring, and resin composite bonding. Ideally, non-conservative, additive procedures should be postponed whenever possible. Today, conservative treatment it is often preferable due to minor, foreseen aesthetic limitations that clearly benefit the long-term biomechanical teeth behaviour.

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