



The Official Publication of The Faculty of Dental Medicine For Girls, Al-Azhar University Cairo, Egypt.

ADJ-for Grils, Vol. 4, No. 4, October (2017) — PP. 371:380

Prevalence of Enamel Hypoplasia and Enamel Hypocalcification in a Group of Egyptian Children

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Codex: 01/1710

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ABSTRACT

Purpose. This study was designed to determine the prevalence of developmental enamel defects in a group of Egyptian children and to determine the most common causes of these developmental enamel defects. Subjects and Methods: A total of 1500 children (4-14 years old, 700 boys and 800 girls) were randomly selected from the health insurance public daycare centers. For each child a clinical examination was performed according to the 2013 WHO dental caries criteria and the modified DDE index (FDI, 1982). Children were divided into three groups, according to the type of dentition as follows, group (A) included "692" children and representing the children who had only deciduous dentition, group (B) included "688" children and representing the children who had mixed dentition and group (C) included "120" children and representing the children who had only permanent dentition. Results: Prevalence of enamel defect were observed in 22.5% of cases. Conclusion: Prevalence of enamel defect were observed in 337 cases with the most common defect was demarcated opacity which was found in 197 cases (13.1%). There was a statistically significant association between presence of enamel defects and previous disease. Previous surgery, previous radiation, previous medication and previous hospitalization. Also, high mean dmf, def and DMF indices, high mean GI. There was no statistically significant association between presence of enamel defects and maternal history during pregnancy, present disease, present intake of medication and familial history of disease

KEYWORDS

Enamel Hypoplasia, Enamel Hypocalcification, modified DDE index.

INTRODUCTION

Developmental enamel defects are disturbances during enamel formation and may be manifested as enamel hypoplasia or enamel

- Paper extracted from master thesis entitled;"Prevalence of Enamel Hypoplasia and Enamel Hypocalcification in a Group of Egyptian Children".
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hypocalcification. Hypoplasia is a quantitative defect associated with a reduced or altered amount of enamel. It varies in appearance from pits and grooves to partial or total loss of enamel surface. However hypocalcification is the qualitative defect involving alteration in translucency of enamel. These lesions appear as white, cream, yellow, or brown in color ⁽¹⁾. Developmental defects of enamel (DDE) are daily encountered in clinical practice. The knowledge of etiological factors for DDE is essential for clinicians when explaining and discussing the presence of DDE with patients and their parents ⁽²⁾.

The common causes of enamel defects include: inherited (as in amelogenesis imperfecta); excess fluoride intake; and a systemic upset during amelogenesis. However, in some cases, the aetiology is unknown and the condition is termed 'idiopathic enamel hypoplasia (IEH) (3).

Types of hypoplasia include *Pits* form: tiny areas of enamel loss, which could be single, multiple, shallow or deep, scattered or in rows, Groove (furrow) form: single or multiple, narrow or wide (maximum 2mm) grooves of enamel loss and Area (plane) form: partial or complete absence or enamel over a considerable area of the tooth(4) Types of Hypocalcification (opacity) include Demarcated opacities which are defects involving an alteration in the translucency of the enamel, variable in degree and the defective enamel is of normal thickness with a smooth surface. It has a distinct and clear boundary with the adjacent normal enamel and can be white, cream, yellow or brown in color and Diffuse opacities which are defects involving an alteration in the translucency of the enamel, variable in degree and white in color. The defective enamel is of normal thickness and can have a linear, patchy or confluent distribution, but there is no clear boundary with the adjacent normal enamel (5).

Developmental defects in the enamel present important clinical significance since they are responsible for aesthetic problems, increased wear, dental sensitivity, dentofacial anomalies, as well as for a predisposition to dental caries. In the field of public health, developmental defects in the enamel have taken on a high level of importance for being predictors of dental caries. Populations affected by these changes require as a priority preventive intervention and early treatment ⁽⁶⁾.

SUBJECTS AND METHODS

The study population included 1500 (700 boys and 800 girls) Egyptian children aged 4 to 14 years. Children were randomly selected from a health insurance public daycare center.

Classification of cases

Children were divided into three groups A,B & C.

- Group A: included "692" children and representing the children who had only deciduous dentition.
- 2. Group B: included "688" children and representing the children who had mixed dentition.
- 3. Group C: included "120" children and representing the children who had only permanent dentition.

Every child in this study will be subjected to:

- Extraoral examination: for physical appearance, Skin, extremities, fingers and finger nails, facial symmetry.
- Intraoral examination: the teeth and mouth examined using WHO Basic method of oral health survey ⁽⁷⁾. The defects were assessed by visual and tactile inspection using directed light without the enamel surface being preliminary dried.
- A dental examination chart was filled to assess the soft and hard tissue condition using caries indices, dmf index and def index for primary teeth and DMF for permanent teeth and Gingival index GI.

- Photographs were taken for the child's upper and lower teeth using check retractor and digital Agfa Optima 103 camera.
- A survey questionnaire was designed to collect data from parent of each child.

Statistical analysis

Qualitative data were presented as frequencies and percentages. Chi-square (x^2) test was used for comparisons between boys and girls as well as the three groups. It was also used to study the association between presence of enamel defects and medical history. Quantitative data were presented as mean and standard deviation values. The significance level was set at $P \le 0.05$.

RESULTS

Enamel defect were observed in 337 cases representing 22.5%. The most common defect was demarcated opacity – white/cream <1/3 of the crown which was found in 197 cases (13.1%).

There was no statistically significant difference between prevalence of enamel defects in boys and girls regarding all types of defects except for the following defects:

- Demarcated opacity yellow/brown at least 2/3 of the crown and
- Diffuse opacities patchy At least 1/3, <2/3
 of the crown; boys showed statistically
 significantly higher prevalence than girls.

There was no statistically significant difference between prevalence of enamel defects in the three groups regarding all types of defects except for the following defects: (Table1)

- Demarcated opacity white/cream <1/3 of the crown;
- Demarcated opacity yellow/brown <1/3 of the crown;
- Demarcated opacity yellow/brown, at least 1/3, <2/3 of the crown;
- Diffuse opacities confluent + staining + enamel loss <1/3 of the crown.

Group III showed the statistically significantly highest prevalence of these defects, followed by Group II then Group I.

| Table (1): Comparison | between prevalend | ce of enamel dej | fects in the three ; | groups |
|------------------------------|-------------------|------------------|----------------------|--------|
|------------------------------|-------------------|------------------|----------------------|--------|

| Enamel defects | Group I (692) N (%) | Group II (688) N (%) | Group III (120) N (%) | P-value |
|---|------------------------|-------------------------|--------------------------|---------|
| Demarcated opacity – white/cream <1/3 of the crown | 32 (4.6) | 130 (18.9) | 35 (29.2) | <0.001* |
| Demarcated opacity – white/cream At least 1/3, <2/3 of the crown | 3 (0.4) | 11 (1.6) | 1 (0.8) | 0.092 |
| Demarcated opacity – white/cream At least 2/3 of the crown | 1 (0.1) | 4 (0.6) | 1 (0.8) | 0.322 |
| Demarcated opacity - yellow/brown <1/3 of the crown | 22 (3.2) | 44 (6.4) | 10 (8.3) | 0.006* |
| Demarcated opacity - yellow/brown At least 1/3, <2/3 of the crown | 3 (0.4) | 11 (1.6) | 4 (3.3) | 0.011* |
| Demarcated opacity - yellow/brown At least 2/3 of the crown | 1 (0.1) | 4 (0.6) | 0 (0) | 0.298 |
| Diffuse opacities – lines <1/3 of the crown | 3 (0.4) | 7 (1) | 1 (0.8) | 0.442 |
| Diffuse opacities – lines At least 1/3, <2/3 of the crown | 0 (0) | 2 (0.3) | 0 (0) | 0.307 |
| Diffuse opacities – lines At least 2/3 of the crown | 0 (0) | 3 (0.4) | 1 (0.8) | 0.133 |
| Diffuse opacities – patchy <1/3 of the crown | 5 (0.7) | 8 (1.2) | 2 (1.7) | 0.532 |

| Enamel defects | Group I (692) N (%) | Group II (688) N (%) | Group III (120) N (%) | P-value |
|--|------------------------|-------------------------|--------------------------|---------|
| Diffuse opacities – patchy At least 1/3, <2/3 of the crown | 2 (0.3) | 8 (1.2) | 2 (1.7) | 0.103 |
| Diffuse opacities – patchy At least 2/3 of the crown | 4 (0.6) | 4 (0.6) | 1 (0.8) | 0.942 |
| Diffuse opacities – confluent At least 1/3, <2/3 of the crown | 0 (0) | 3 (0.4) | 0 (0) | 0.170 |
| Diffuse opacities – confluent At least 2/3 of the crown | 1 (0.1) | 1 (0.1) | 0 (0) | 0.917 |
| Diffuse opacities - confluent + staining + enamel loss <1/3 of the crown | 1 (0.1) | 8 (1.2) | 2 (1.7) | 0.039* |
| Diffuse opacities - confluent + staining + enamel loss At least 1/3, <2/3 of the crown | 1 (0.1) | 8 (1.2) | 1 (0.8) | 0.065 |
| Diffuse opacities - confluent + staining + enamel loss At least 2/3 of the crown | 1 (0.1) | 5 (0.7) | 0 (0) | 0.177 |
| Hypoplasia – pits <1/3 of the crown | 7 (1) | 10 (1.5) | 3 (2.5) | 0.394 |
| Hypoplasia – pits At least 1/3, <2/3 of the crown | 1 (0.1) | 2 (0.3) | 1 (0.8) | 0.396 |
| Hypoplasia – missing enamel <1/3 of the crown | 2 (0.3) | 7 (1) | 2 (1.7) | 0.130 |
| Hypoplasia – missing enamel At least 1/3, <2/3 of the crown | 2 (0.3) | 2 (0.3) | 1 (0.8) | 0.612 |
| Hypoplasia – missing enamel At least 2/3 of the crown | 1 (0.1) | 2 (0.3) | 0 (0) | 0.730 |
| Any other defects <1/3 of the crown | 2 (0.3) | 0 (0) | 1 (0.8) | 0.131 |
| Any other defects At least 1/3, <2/3 of the crown | 1 (0.1) | 0 (0) | 0 (0) | 0.558 |
| Diffuse + hypoplasia <1/3 of the crown | 1 (0.1) | 0 (0) | 0 (0) | 0.558 |

^{*:} Significant at $P \le 0.05$

Association between most common diseases and presence of enamel defect

Regarding Anemia patients, cases with no enamel defects showed statistically significantly higher prevalence than cases with enamel defects.

Regarding Bronchial asthma as well as Epilepsy patients, cases with enamel defects showed statistically significantly higher prevalence than cases with no enamel defect.

While for IDDM and Rheumatic fever patients, there was no statistically significant association between presence of disease and enamel defects

Table (2) Results of chi-square test for the association between common diseases and enamel defects

| Disease | No defect (1163) N (%) | Defect (337) N (%) | P-value |
|-------------------------|------------------------------|--------------------------|---------|
| Anemia (n, %) | 41 (3.5) | 4 (1.2) | 0.027* |
| Bronchial asthma (n, %) | 7 (0.6) | 7 (2.1) | 0.013* |
| Epilepsy (n, %) | 3 (0.3) | 5 (1.5) | 0.007* |
| IDDM (n, %) | 4 (0.3) | 4 (1.2) | 0.061 |
| Rheumatic fever (n, %) | 8 (0.7) | 4 (1.2) | 0.365 |

DISCUSSION

Developmental enamel defects (DDE) have been defined as disturbances in hard tissue matrices and their mineralization that arise during odontogenesis. According to their clinical appearances, DDE have been classified as hypocalcification (demarcated opacity, diffuse opacity) or hypoplasia. Enamel hypocalcification is a qualitative defect involving an alteration in the translucency of enamel and may appear white, yellow or brown in color. Enamel hypoplasia is a quantitative defect associated with a reduced enamel thickness⁽⁸⁾.

The present study was carried out to determine the prevalence of developmental enamel defects in a group of Egyptian children aged from 4 to 14 years(to cover all dentition types) and to determine the most common causes of these developmental enamel defects. Several clinical indices have been developed to categorize enamel defects and they can be divided into (a) Specific fluorosis indices which identify and categorize only dental fluorosis, and (b) Descriptive indices with no etiological assumption .The modified DDE Index is a descriptive index. It is more practical and comparable index for epidemiological studies and it allows efficient recording of prevalence and severity of enamel defects(9). Finally, the chi-square test was used to statically analyze the results^(5,10).

The results of this study showed that the prevalence of enamel defects was (22.5%), a finding that was somehow similar to results obtained by a study in Brazilian children who reported (24.4%)⁽⁶⁾, and a study in Chinese children who reported (23.9%) (11). Meanwhile the studies who reported higher results are: (99%) in Australian Aboriginal children⁽¹²⁾, (66%) Norwich (England⁽¹³⁾, (58%) in Australia⁽¹⁴⁾. Observed prevalence differences could be due to specific characteristics of populations with regard to the presence of a particular illness or regional pollutant, as well as methodological aspects, such as index and criteria used. The type of light source used for the exams; if there was tooth brushing, or drying of teeth before the exam; or if only anterior teeth or the whole dentition are examined ⁽¹⁵⁾. Populations with lowest enamel hypoplasia as in Japan (2%) and in Mexico (6%) could be attributed to the racial differences and diversity of the methodological procedures which were used ⁽¹⁶⁾.

The *most common defect* in this study was demarcated opacity (13.1%) this was in agreement with other studies ^(1,10,17). This was may be caused by transient damage to ameloblasts during the maturation phase that leads to formation of demarcated defects, but the cells are able to recover and resume their normal function⁽¹⁾.

Regarding the gender, it was found that no statistically significant difference between prevalence of enamel defects in boys and girls regarding all types of defects except for the demarcated opacities and diffuse opacities, boys showed statistically significantly higher prevalence than girls, these results were in agreement with the results of other studies in Iran⁽¹⁸⁾, Brazil⁽⁶⁾ and in Mexico⁽¹⁵⁾, they showed no statistically significant relationships with regard to gender, while boys showed higher prevalence than girls in other studies; in China (19), in Malaysia (20), in Iowa (17). Definitive reason for this finding is suggested to be because of greater intra uterine nutritional demands in boys than in girls, since boys weigh more, have more muscle mass, and are developmentally delayed both in the uterus and at birth. Greater nutritional requirements of boys due to more rapid growth make them more susceptible than girls to the formation of enamel defects⁽¹⁾·Meanwhile these results were in disagreement with the results of other studies were girls showed higher prevalence than boys^(21,22). This could be related to a cultural factor (the preference for a son within the family), or better nutritional conditions including longer breastfeeding (23), or it could be due to earlier eruption of teeth among girls than boys⁽²⁴⁾.

The prevalence among the three *groups* showed that there was no statistically significant difference between prevalence of enamel defects in the three groups regarding all types of defects except for demarcated and diffuse opacities where Group III showed the statistically significantly highest prevalence of this defect, followed by Group II then Group I. This was assured in other studies^(10,17,14), this may be due to the period between births to 2 years of age is an active phase of amelogenesis for permanent teeth so any common systemic conditions occurring during this critical period makes the teeth particularly susceptible for formation of DDE ⁽¹⁾.

Regarding the association between presence of defects and mean *Caries indices*, in this study cases of enamel defects showed statistically significantly higher mean dmf, def and DMF than cases without enamel defects. These results were in agreement with the results of other studies (25,26,27), this is seems logical that surface irregularities on the smooth surfaces of the teeth from EHP would promote the colonization of mutans streptococci (MS), other cariogenic bacteria (28), also decreased mechanical properties of defected enamel could be a reason since hypocalcifieded enamel is less mature (29) and have reduced thickness (30).

As regards the association between presence of defects and *Medical history* in this study there was a statistically significant association between presence of defects and previous disease (e.g. Anemia, Bronchial asthma, Epilepsy, IDDM, Rheumatic fever). Disruptions of enamel formation were associated with frequent illness⁽³¹⁾. It has been suggested that any health problem prior to 5 years of age can modulate ameloblast activity and therefore impair amelogenesis⁽³²⁾.

For *Anemia* patients, cases with no enamel defects showed statistically significantly higher prevalence than cases with enamel defects this is assured by certain study who found that there is no significant difference between anemic and normal children⁽¹²⁾. While it's opposite to the results

obtained by other studies who found that there is a significant association between dental defects and evidence for anemia^(33,34)

For *Bronchial asthma* cases with enamel defects showed statistically significantly higher prevalence than cases with no enamel defects, this was assured by the findings reported by other studies (32,35,36). This can be hypothesized that pediatric asthma patients with dental enamel defects have probably experienced previous episodes of oxygen deprivation and ameloblasts are highly sensitive to oxygen supply⁽³²⁾, also asthma drugs (either inhaled or oral) have been suspected to increase the prevalence of enamel defects⁽³⁷⁾.

For *Epilepsy*, cases with enamel defects showed statistically significantly higher prevalence than cases with no enamel defects this is confirmed by other studies (38,39) where increased incidence of trauma in epileptic patients during seizures may be a reason of increased incidence of enamel hypoplasia and hypocalcification. Dental tissues being calcified tissues can also be affected by the derangement of mineral metabolism that occurs with antiepileptic drugs, especially during developmental phases (40). Also epilepsy was considered one of the etiologic factors of the defects, this may be caused by antiepileptic drugs as long-term phenytoin therapy is known to cause disturbance in calcium and bone homeostasis (35).

On the other hand the results for *IDDM* patients, showed no statistically significant association between presence of disease and enamel defects, which may be due to antidiabetic drugs taken by all diabetic children since anti-diabetic medication resulted in normal enamel ultrastructure⁽⁴¹⁾, or the onset of disease begin after enamel formation is completed because enamel once formed is not remodeled ⁽⁴²⁾. Meanwhile other results showed the opposite, which reported a decrease in the secretion of enamel matrix forming a thinner enamel layer in diabetes^(43,41)

Regarding *Rheumatic fever* patients, there was no statistically significant association between presence of disease and enamel defects in all groups which is similar to the results obtained by certain studies⁽⁴⁴⁻⁴⁶⁾. On the other hand results from other studies⁽⁴⁷⁻⁴⁹⁾ showed higher prevalence. It is also possible that enamel hypoplasia in cardiac children has resulted from systemic disturbances, such as cardiac failure and surgical complications associated with the cardiac disease during prenatal and neonatal development⁽⁴⁵⁾.

As regards association between presence of defects and *previous surgery*, *radiation*, *medication*, *and hospitalization*. Cases with previous surgery, radiation, medication, and hospitalization showed statistically significantly higher prevalence of enamel defects which is nearly the same obtained by other studies (50,35,42). The reason of this finding is may be due to the fact that dental epithelial cells (ameloblasts) are more sensitive to the drug than dental mesenchymal cells (odontoblasts) the effects depend on the dose of the drug (51).

Concerning cases of previously repaired *Clefts*, cases in the sample having cleft lip history shows different degrees of enamel hypoplasia and hypocalcification this was assured by certain study⁽⁵²⁾ that found that there was a significant relationship was found between the cleft side and enamel defects, the highest prevalence on the cleft side suggests that the cleft does influence the occurrence of enamel defects in permanent teeth, this were in agreement with other studies^(35,53) and more recently⁽⁵⁴⁾ where enamel defects was more common in CL(P) children in both primary and permanent dentition.

As regards association between presence of enamel defects, *maternal history during pregnancy and familial history of disease*, there was no statistically significant association between

presence of enamel defects, maternal history during pregnancy and familial history of disease, this was disagreed by certain studies^(11,55,56), where there was statistically significant association between presence of enamel defects and mothers who had experienced problems during pregnancy while there was no statistically significant association between presence of enamel defects and family history of enamel defects.

CONCLUSIONS

Based on this study results, the following conclusions can be made:

- 1. Prevalence of enamel defect were observed in 337 cases representing 22.5%.
- 2. There was no statistically significant difference between prevalence of enamel defects in boys and girls regarding all types of defects except for Demarcated and Diffuse opacities; boys showed significantly higher prevalence than girls.
- 3. There was no statistically significant difference between prevalence of enamel defects in the three groups regarding all types of defects except Demarcated and Diffuse opacities; Group III showed the statistically significantly highest prevalence of this defect followed by Group II then Group I.
- 4. There was a statistically significant association between presence of enamel defects and previous disease, mean dmf, def, DMF and GI indices, previous surgery, radiation, medication, and hospitalization.
- 5. There was no statistically significant association between presence of enamel defects and maternal history during pregnancy, present disease, present intake of medication and familial history of disease.

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