

Dental Dysplasia as Structural Anomalies in Child Oral Pathogeny

MADALINA MATEI¹, ODETTE-ELENA LUCA^{2*}, MIHAELA BLAJ^{2*}, ANAMARIA ZAHARESCU¹,
CATALIN PLESEA CONDRATOVICI¹, DANIELA ANISTOROAEI², CRISTIAN ROMANEC²

Dunarea de Jos University of Galati, Faculty of Medicine and Pharmacy, 47 Domneasca Str., 800008, Galati, Romania
Grigore T. Popa, University of Medicine and Pharmacy, Faculty of Dental Medicine, 16 Universitatii Str., 700115, Iasi, Romania

Dental dysplasia such as structural anomalies, complex pathogenic foundations, although of a relatively low incidence in the child oral pathology is of a wide interest, attested to a considerable wealth of scientific material reserved in the specialized literature. The prevalence of defective enamel on permanent teeth in the maxilla is smaller in the jaw. The permanent superior incisors are in the first place from the point of view of frequency in the enamel defects, usually symmetric, either at the 2 central, or at all the 4. The first permanent molars and the premolars are permanently affected to a lesser extent, and on canines injuries occur very rarely, only in very serious cases (genetic). We have examined a number of 300 children school communities, with the chronological age between 6 and 11 years 195 girls (65%) and 105 boys (35%). On distribution of children in age groups, we found a concentration of distribution at the age of 8-9 years. Changes to the DDE index in order to be efficiently and flawlessly divided by enamel defects, being suitable for screenings, but also for more detailed and larger studies, when they can be adapted to the research requirements. The most frequent affectation of the upper permanent incisors correlated with permanent primary molars, implies an intensification of the dental education activity oriented to the young schoolchildren, then the awareness of the dental factor in the facial aesthetics appears late, during puberty-adolescence when the lesions remain aggravate and the oral rehabilitation is complex and difficult, in these cases.

Keywords: dental dysplasia, progress defects, enamel development.

The interest for the development of the possibilities and methods of clinical and paraclinical evaluation of dental dysplasia appears in the literary version from 1916, when MC Key describes a discromy of the enamel called the "mottled" enamel (spotted) that favored in establishing that the fluoride in the water is one of the causes of fluorosis and that it is an association regarding the creation of the fluor concentration in water and the discharge of caries in the studied population [1-3].

Embryogenesis as an oral cavity including through the dental system is closely related to extreme cephalic ontogenesis.

The ontogenetic dynamics of the cells, in the formation of the human embryo is variable as the growth rate in the embryos is not the same, with large differences in terms of growth rate. Growth is a process of exchange which changed the zygote into a multicellular organism.

The cell has put the codes of care of the genetics, by expressing them, it contributes to the formation of the maxillary bones and teeth [4-6].

In the 13 mm embryo, the epithelium as a primitive oral cavity is bistratified. It has a layer of high, prismatic basal cells and a superficial layer in superficial format up to a single row of cells is applied with its help in much glycogen cytoplasm. At the end of the first embryonic month, the cells in their care will be dental organs: ectodermal, ectomezodermal, ectodermal.

Enamel formation (amelogenesis) is the result of cell secretion processes and there are three phases: the precursors, the cells differentiated by chronological age, are aligned so that the cells that are older and the youngest cervical; secretory, when there are strings of ameloblasts withdraw from the dentin in full, but no extension is left inside the matrix, which can be degraded, being replaced by water; maturation starts at this time, when the full thickness of an enamel format is increased, protein and water are eliminated and protruded the ions.

Temporary teeth undergo a much more accelerated maturation process. When determining the enamel, the differences regarding the orientation of the enamel prisms, which, at the level of the cracks and the occlusal holes are almost parallel with the longitudinal axis of the temporal teeth [7-9].

*email: lucaodette@yahoo.com; mibblaj@yahoo.com

In the specialty literature the term of dysplasia designates a process of a disturbance of anatomical tissues, organs or parts, taken before or after training to form malformations or deformations.

Dental structural abnormalities are disturbances of the dental organs, a special quantity of teeth and tissues and therefore can be used in terms of dental dysplasia in general terms; there are metabolic disorders of the hard dental tissues that occur during dentistry. Macro and microscopically, it may present a defect, more or less, until the temporary and the definitive period. Structural defects affect enamel, dentine or cement, or all the hard dental tissues.

The pathogenesis of dental dysplasia are abnormal: an organic matrix for a hard tissue; mineralization or maturation for this matrix; formation and mineralization of the matrix.

Hereditary dental dysplasia causes disruption of amelogenesis, dentinogenesis, or both (dentinogenesis); it is most often manifested in isolation, but can be accompanied and managed in the administration systems; data are modified exclusively to lesions on isolated genes, there are mutations and general hereditary syndromes, care induce changes of enamel, dentine, or both [10-12].

The imperfect amelogenesis includes a dysplasia of hereditary origin of the enamel. Three main mechanisms are responsible for structural changes of the enamel: a genetic mutation of the enamel gene, with Mendelian transmission, a genetic or chromosomal abnormality during malformative syndromes; or altered phosphocalcial metabolic metabolism, all phenomena involved in the postmeloblastic phase of amelogenesis [13- 15].

Other structural changes in dentin may be encountered in the form of imperfect dentinogenesis or dental display.

Congenital dental dysplasia depend on the individual organization while they may occur during births; or refers to the diseases or injuries present from birth, manifest or with potential manifestation that will be revealed later.

Pregnancy is a period needed to determine physiological changes in the body of women, changes in care take on an appearance of disorder and may be pathological. These changes are: endocrine, blood, respiratory, metabolic.

Three risk factors are presumed to influence a foetus: diseases affecting pregnancy; that coexists with pregnancy; drug risks [16-18].

Dyschromic dysplasia, intrinsic dyschromias or pigments are associated with lesions of enamel, dentine or pulp.

Congenital dyschromias are: fluorosis, coloration of tetracycline modifications, porphyria, due for enzyme conversion abnormalities, with recessive transmission[19-21].

Acquired dysplasia are the consequence of disruption of organic formation or disruption of mineralization and / or maturation of hard dental tissues [22-24].

Turner's dysplasia is represented by isolated lesions, affecting a permanent tooth. It is of local origin, in relation to a periapical infection or a trauma of the temporary teeth.

30% of children are victims of trauma caused by a direct or indirect shock, as a result of the famous fall multiple times [25-27].

Fluorosis is an endemic disturbance of a formation in question due to excessive intake of fluorine. In the dischromic dysplasia due to tetracycline, tetracycline has the ability to deposit an organic matrix of a bone or the whole, before triggering calcification, as well. It is not necessary for the caries to affect the child immediately after the temporary eruption, leading to their coronary destruction [28-30].

Experimental part

Methods and materials

There were examined 300 children from school communities, with a chronological age between 6 and 11 years, 195 girls (65%) and 105 boys (35%). Regarding the distribution of sample of children by age groups, we found a concentration of the distribution at the age of 8-9 years.

Results and discussions

With the help of the indicated DDE (classification of enamel defects) – modified, we appreciated the development enamel defects, marked and diffuses opacities, hypoplasias, dischromas and their severity for the studied school population.

The codes of the modified DDE index proposed by Clarkson O Mallone-11: the normal-enamel look is smooth, glossy, pale, white-yellowish; opacity - a qualitative defect of the enamel identified with the naked eye as abnormal in its translucency. It is characterized by a white or a discolored one (yellowish, brown), but in all cases it remains very small and its thickness is normal; enamel-cavity hypoplasia is a quantitative defect of the enamel, the morphological visualization and identification as superficial or deep or cavities are partially or totally dispersed on the surface as a whole; diffuse hypoplasia, the enamel appears thin or thick, with narrow or broad grooves, cast vertically or irregularly on

the surface of the tooth; the linear type - a quantitative defect of enamel, visualized and morphologically identified with continuous grooves, so as or dysplasia a horizontal line on a large surface.

The missing enamel, deficient, is responsible for a large part of the tooth surface, with no area and general shape to be affected.

In order to establish the indication of DDE, modified: temporary examinations, the vestibular and linguistic surfaces for each anterior part and the vestibular and occlusal surfaces of each posterior tooth; of these the previous examination shall be examined without being washed and dried.

The use of DDE index has changed our lot in the conditions of distribution of enamel defects.

The prevalence of enamel defects extends over an area of 15%, naturally equal to that appreciated by the DDE index, but there are some changes in the distribution of defects on codes.

An important change was introduced in this study to be able to use the original code to clearly distinguish between the diffuse and demarcate opacities. This change gave authorization to separately register the diffuse opacities.

Using the DDE-modified index the opacities can be classified according to the different aspect or well demarcated and this point of view, in our lot can respond to opacity can represent 3.6%, delimited 2.3%, and the hypoplasia with dents is the most encountered - 3.3% then hypoplasia with the absence of enamel 2% and hypoplasia with cavities 1.6%. In the DDE modified index we studied the defect extension and it is observed that, for the batch of schoolchildren, only 23% of the cases have spread a defect.

In most cases there is still 48%, the lesion is extended by more than 2/3 of the second vestibular part inside, and 34% is less to 1/3 of the tooth.

It is important to emphasize that the DDE index can be used in a flexible manner, depending on the demands of the study. The codes are not closed within strict limits, they can be added or used on demand, depending on the research objective.

For general screening it is advisable to record only three tips to make defects; any other defect does not fit in these categories, it should be registered as "Other defects".

If epidemiological studies are extensive, an analytical and descriptive form is recommended to obtain the most important results. A criterion standard is needed to examine when using a DDE index.

The focus group examined needs to be standardized. They must provide a sufficiently wide area for a prevalent set of defects to envisage an enamel in early and late eruption.

The distribution of glaze attachment defects (DDE) on the maxilla and jaw highlights or spreads a glaze defect on the teeth that is maintained up to a maximum of 48% on the maxilla and 27% on the lower jaw.

It was observed that there may be a significant difference regarding the dental dysplasia in the incisors on the maxilla 49% compared to the mandibular ones 21%, which may indicate an argument in favor of using the indicated SCOTS index in the epidemiological population survey, when they can defect according to the enamel.

The SCOTS index was created and applied as a public health index to monitor the glitches of choice in enamel in Scotland, and this is only at the small upper incisors.

Analyzing the details of the location of the dysplasia on the incisors after they are affected, we noticed more frequency in symmetrical cases, marked by an even number of teeth 2 and 4. These results are consistent with the celebrity achieved by Li Y and Navia JM, China, who are careful to report both make-ups with a relatively low degree of symmetry and the lesions examining the two hemiarchades.

It is also observed that the temporary maxillary incisors may be affected during pregnancy, it may also be sent to study, when they are physiologically exfoliated from the arch.

Dysplasia devices are symmetrical, and permanent mandibular incisors are very rarely affected and, usually, all 4 (16 cases).

The same analyze that once is made for the canines to notice their affectation is very small, and the distribution of cases is very close for one or both canines of each jaw.

More impairments, but statistically insignificant, are temporary ($p > 0.05$). Some authors show that the lesion more encountered is opacity (13, 9%), diffuse hypoplasias affect the central and lateral incisors.

Affecting the molars and premolars, it is noted that the presence of jaw dysplasia is wider than that on the mandibula, without needing any guarantee to be able to signify the statistics (less cases).

Statistical analysis shows a maximum value testing remaining enamel defects evolving to maintain permanence that is dependent on the presence of defects over time, but the risk factor may present only the defects on the permanent molars for permanent canines.

For the jaw the values of the test reveal statistical significance only for defects of the temporal molars and the permanent incisors and respectively for the appearance of the defects while they are the temporary incisors as compared to the

permanent incisors, but at the same time, a change of the enamel defects to the temporary dentition is not a risk factor for the enamel defects of permanent dentition.

By making or simple regression according to higher incisor number of enamel defects to molars receive permanence that refers to a correlation coefficient $r = 0.54$, with confidence intervals (0.46; 0.62), the significance threshold 95%, which shows that it could have a fairly high correlation with regard to the two types of tooth. The explanation for this correlation could be the close chronological record for importance and mineralization of the two dental types, which starts for the first permanent molar at about 4 months intrauterine, and for incisors at 5 months intrauterine, continuing with the onset of mineralization, at birth for the primary molar and at 3-4 months postnatal for central incisors.

The study has been continued with a method more detailed between the association of the enamel in different types of teeth, which refers to different types of dental instruments. For this we took into account the correlation indexes for temporary and permanent dentition. The most powerful correlation is calculated for affecting the permanent molars and canines, 0.82% maximum and teeth, for a period of time and for a permanent one. It was found that the strongest correlation can affect canines and molars permanently, of 0.82, this information is the most serious for canine damage is very rare, only serious, genetic cases, when other information on architecture and structure is presented. Next, the wines and the correlation coefficient between canines and incisors of only 0.4 are explained, which may indicate the incisors can be affected exclusively in acquired cases.

They are analyzed and given to each part, it must pay more attention and be noticed, which remains maxillary and which allows a stronger correlation to remain permanent, in order to make a greater contribution to examine, considering the age group of the subjects. Thus, the canine-molar correlation coefficient is maintained on the maxilla a 0.67, and the temporary at 0.63; also, on the jaw, the correlation between the appearance of enamel defects in the permanent canines and molars is 0.72, while in the temporary is only 0.52.

In our study we have shown localizations on the incisors, after the affected teeth, have a higher frequency and a very close correlation with an effect on the permanent molars.

The explanation of the phenomenon is possible for each dental group that there are certain forms of formation and maturation, when the etiological factor may be involved. If a disruptive factor occurs in the 13th intrauterine month, the dysplasia lesion will affect the maxillary and mandibular central incisors. When the etiological factor appears at birth, it is important that the teeth be affected in a different degree.

With these nursing children, they presented diffuse enamel defects at the molars (RR = 1.45; 95% CI, 1.05-2.0) which may create a significant risk of an enamel defect, diffuse in permanent incisors.

Up to 7 years old, it disperses almost 0 and can be created when it can be seen for 9 years, reaching the maximum point of 11 years; this shows that there may be a variety of functions depending on age.

EDS score (enamel defect score) is an average value ranging from 0.06 to 7 years in 0.3 to 11 years. This score is low compared to the low prevalence in the studied child population.

The score limits are very wide, from 0 to 10.4. For boys, more than 0.3 (2.6%) and 2.2 (1.9%) for girls are obtained. 91.6% of boys and scores 0 only when 85.1% of girls are low.

The temperature indicators of gravity are between 10% and 20%, respectively 13, between 50-59% , 7 cases. There are 2 gravity indicators between 80% and 100%. a much more dispersed distribution of children with enamel defects, almost every class is represented as follows: between 0.1-9% -12cases 10% -19% 16 cases, 20-29% 7 cases, between 30 and 37% - 11 cases, between 40 and 49% there are 3 cases, while 50 and 59% 3 cases and more than 60% up to 4 cases.

Having in mind the age of the studied group, the severity index for permanence is higher than the time required, then there are fewer temporary than permanent teeth, and on the other hand the high severity index of those permanently is closely related to a level high in injuries.

Conclusions

The EDS index can offer a global imagine on the gravity of the development defects of the enamel in terms of community care, it's relatively straightforward to calculate and relevant to the common gravity.

The prevalence of defects extends over a considerable area or with a public health problem, but it is indicated by the severity of very large ones, which refers to a new orientation regarding their prevention, interception and treatment.

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