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ENAMEL HYPOPLASIA: A CONCISE REVIEW OF ITS FACTORS & PATHOGENESIS

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ABSTRACT

Developmental disturbances of the teeth occur with the insult/trauma to the dental lamina. The lesions may differ with the stages of the tooth development and the intensity of the trauma to the dental lamina. A considerable number of environmental factors have been reported to be capable of causing enamel defects and systemic disturbances consist of intoxications, prenatal and postnatal problems, malnutrition, infectious diseases and a range of other medical conditions. This review is an attempt to factors and Pathogenesis associated to enamel hypoplasia, so all clinical features of enamel hypoplasia are not included.

INTRODUCTION

Disorders of development of teeth may be due to abnormalities in the differentiation of the dental lamina and the tooth germs, causing anomalies in the number, size and form of teeth (abnormalities of morpho differentiation) or to abnormalities in the formation of the dental hard tissues resulting in disturbances in tooth structure (abnormalities of his to differentiation). Abnormalities of his to differentiation occur at a later stage in development than abnormalities of morpho differentiation; in some disorders both stages of differentiation are abnormal Developmental disturbances of the teeth occurs with the insult/trauma to the dental lamina. The lesions may differ with the stages of the tooth development and the intensity of the trauma to the dental lamina [1].

Enamel normally develops in two stages

• In the secretory stage: The ameloblasts perform the dual function of matrix production and initial mineralization.

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Matrix production involves the synthesis and secretion of the matrix proteins, amelogenin, enamelin, ameloblastin and tuftelin, of which amelogenin accounts for about 90 per cent. Initial mineralization occurs immediately after secretion.

• The maturation stage: There is withdrawal of protein and water from the enamel accompanied by increase in mineral content before the tooth erupts.

Most classifications of disturbances in enamel formation distinguish between those that affect the secretory stage, resulting in deficient matrix production and thin hypoplastic enamel and those that affect the maturation stage, resulting in deficient mineral deposition and soft hypomineralized enamel [2-4].

Hypoplasia is defined as a quantitative defect of enamel visually and is histomorphologically identified as an external defect involving the surface of the enamel and associated with reduced thickness of enamel [5]. The cervical and the incisal borders of the defect have a rounded appearance due to the prisms in the non-affected enamel being bent, which may be attributed to a change in the prism direction. The macro & microscopical appearances suggest that only some specific ameloblasts

have ceased to form enamel, whereas others are partly or completely able to fulfil their task [6].

Pathogenesis

While the pathogenesis of the dental defects remains unclear, it is probable that both systemic disturbances and local factors contribute to the aetiology [7]. Developmental defects of enamel with a similar appearance are not necessarily caused by similar aetiological agents. Conversely, the same aetiological factors can produce different defects at different stages of tooth development. Enamel defects may also result from a combination of factors. It has been proposed that there are well over 90 different factors that may be responsible for causing developmental defects of enamel [8,9].

In the case of amelogenesis, it is not different cells doing different jobs, but the same cells at different stages of maturation doing the different jobs. First they lay down the organic matrix and then they lay down the hydroxyapatite crystals within this matrix and finally they become quiescent and vestigial once the cells have matured from one phase and moved to the next, they cannot go back and fix any defects. Anything that disrupts the delicate ameloblasts during enamel production will result in defective enamel which may be very porous and weak. This defective enamel is often present at eruption but will soon be lost to abrasive forces. This leaves an area of exposed dentin and rough margins to the surrounding enamel. In some instances the enamel does not form at all and so is missing as soon as the tooth erupts.[10]

A considerable number of environmental factors have been reported to be capable of causing enamel defects. These systemic disturbances consist of intoxications, prenatal and postnatal problems, malnutrition, infectious diseases and a range of other medical conditions [11].

Suckling suggested that the pathogenesis of each type of Developmental defects of enamel is different and therefore should be considered separately. The stage of amelogenesis at which time the dysfunction occurs, the severity of the insult leading to temporary, or permanent inactivity of the cells, the duration of the insult, the phase of ameloblast activity during the relevant period, and the specific agent involved, may affect the final appearance of the defect [12] damage of secretory ameloblasts results in pathologically thin enamel. However, interference during the maturation stage can lead to defects which present as bands, or patches of chalky opaque porous enamel [13] Suga suggested that ameloblasts were very sensitive to disorders at an early stage of maturation. Hence, if a cell is damaged by a systemic or local disorder at this stage, it cannot easily recover from dysfunction during the long period of maturation. Therefore, he hypothesized that demarcated opacities were due to a disturbance in the process of matrix degradation [14]. It's observed that the homologous teeth of children with demarcated opacities were affected to varying extents, and found it difficult to make any assumption about the severity of the insult because the damaging agent seemed to have been rather nonspecific in most of the children. Hence, they assumed that two or more interacting factors were required to produce the defects [15].

Enamel Hypoplasia

Both dentitions could be affected by enamel hypoplasia; however, the incidence is more severe in permanent dentition. The characteristics of clinical enamel hypoplasia include unfavourable esthetics, higher dentin sensitivity, malocclusion and dental caries susceptibility [16]. The treatment of challenge in this type of injury for a complete oral rehabilitation in both esthetics and function

Disturbances during apposition of hard dental tissues during growth of teeth leads to [1]

(a) Enamel hypoplasia
(b) Amelogenesis imperfecta
(c) Dentinogenesis imperfecta
(d) Dentinal dysplasia
(e) Shell teeth

Hypoplasia are categorized into the following types [17]

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.

Other Factors for enamel Hypolasia

Intoxications, the one responsible for affecting most people is probably fluoride.[11] The effect of increasing the intake of fluoride during tooth development upon the appearance of the enamel has been well-documented. Among the many hypotheses that have been proposed for the mechanism by which excess fluoride affects degradation and removal of enamel matrix proteins, three are favoured by most researchers:

(i) fluoride might directly affect ameloblasts [18]
(ii) proteins may be more tightly bound to fluoridated hydroxyapatite and, thus, proteinolysis might be more difficult [19] (iii) fluoride might inhibit enamel proteinases [20]. However, there is no conclusive proof that fluoride alone, either in the form of an excessive intake, or of an abnormal metabolic process in the presence of a low or normal intake, is responsible for all the enamel changes of diffuse opacities.

Other systemic factors operating over a long period of tooth development such as malnutrition, chronic

illness (diabetes insipidus) & hypervitaminosis D [11,21,22].

Children with low birth-weights, i.e. 2000g or below, have also been shown to have a much higher prevalence of enamel opacities in the first permanent molars and lateral incisors than children who had a normal birth-weight [23]. Since anti-neoplastic therapy affects all cells, it is not surprising that developmental defects of enamel have been documented after oncology therapy [11,24,25]. Infectious diseases during early childhood, such as chicken-pox, measles, mumps, scarlet fever [26] tuberculosis [27] except for chicken-pox, Suckling and her co-workers failed to find positive and strong associations between enamel defects and children experiencing one or more of these diseases in spite of extensive statistical testing [11,28]. Prolonged breast-feeding might increase mineralization defects in teeth because of environmental contaminants such as dioxins or dioxin-like compounds in breast milk [29]. Epidermolysis Bullosa Atrophicans Generalisata Graves, epidermolysis bullosa, and Scarring epidermolysis bullosa, osteopetrosis, teeth may be affected by enamel hypoplasia and enamel pits [30]. Epidermolysis Bullosa Atrophicans Generalisata Graves, may be affected by enamel hypoplasia and enamel pits [30]. Turner's hypoplasia, the most likely cause is a traumatic injury to a primary tooth. The traumatized tooth, 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. White or yellow discoloration may accompany Turner's hypoplasia [31].

CONCLUSION

A considerable number of environmental factors have been reported to be capable of causing enamel defects. These systemic disturbances consist of intoxications, perinatal and postnatal problems, malnutrition, infectious diseases and a range of other medical conditions [11]. While the pathogenesis of the dental defects remains unclear, it is probable that both systemic disturbances and local factors contribute to the aetiology all the lesions and or patterns are not discussed as more emphasis is given for pathogenesis and other factors of Enamel hypoplasia, The treatment of challenge in this type of injury for a complete oral rehabilitation in both aesthetics and function.

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CONFLICT OF INTEREST:
The authors declare that they have no conflict of interest.

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