Developmental Enamel Defects: A review Of Their Etiology And Management

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Abstract

Developmental defects of enamel are a frequent finding in deciduous and permanent dentition. Clinically they are seen as hypoplastic defects or as enamel opacities which may be diffuse or demarcated. These defects may be inherited or non-inherited. The non-inherited defects have been recently described under the term Mineral Incisor Hypomineralisation. They represent the disturbance in enamel formation due to systemic disease in early childhood. The clinician should recognize such defects and follow them up frequently as they are susceptible to caries. A review of present concepts in the etiology and methods of management of these defects is presented.

INTRODUCTION:

Developmental defects of enamel are a frequent finding in primary as well as permanent dentition. Clinically, they are of three types - enamel hypoplasia, demarcated enamel opacities and diffuse enamel opacities.

Enamel hypoplasia is associated with reduced localized thickness of enamel and occurs in the form of pits or grooves in the surface of enamel. It is a quantitative defect of enamel, whereas both diffuse and demarcated opacities are qualitative defects. There is alteration in the translucency of enamel and surface of enamel is smooth. Demarcated opacities have a clearly defined margin but diffuse opacities have no clearly defined margins. They may be white, creamish or yellowish brown in colour. These defects actually represent the disturbances occurred during calcification in early childhood, infancy and intra-uterine life.

ETIOPATHOGENESIS:

The etiological factors in the pathogenesis of enamel defects have been broadly classified into two types, hereditary and environmental/ acquired. Clinical manifestations vary based on the causative factors.

Inherited enamel defects form a relatively small component of all developmental enamel defects in the general population. It can occur as inherited disorder where only the tooth enamel is defective, i.e., amelogenesis imperfecta or can be one of the manifestations of a systemic disease or syndromes. (Table 1) In the inherited type, there is multiple teeth involvement and both primary and permanent dentition are affected and family history will be positive.

Non inherited enamel defects can be of two types:

a. Localised where the defect is due to local factor, one or two teeth are involved.

b. Chronological enamel hypoplasia due to environmental causes that affect teeth from each quadrant in a symmetrical manner. Werthein et al suggested the term Molar Incisor Hypomineralisation and defined it as hypomineralisation of systemic origin of one or more first permanent molars, frequently associated with affected incisors.

The various causes are listed in Table 2.

A metabolic/physical damage to the ameloblasts, their supporting cells in the dental follicle and their vascular supply may result in defective enamel formation. Abnormal growth can occur at either nucleation, late secretary or maturation stages of enamel formation. A number of agents may act at these different stages of enamel formation. Similar clinical expression of defect may occur through common pathways. For example, alteration in calcium homeostasis can cause defect in function of ameloblasts.

DISTURBANCE IN CALCIUM METABOLISM AND ENAMEL DEFECTS:

Various metabolic disorders where there is alteration in calcium homeostasis have been known to be associated with enamel defects. They are liver and renal diseases, nutritional disorders such as vitamin D and calcium deficiency, severe infections such as gastroenteritis, pneumonia and rubella, premature birth, birth asphyxia and respiratory distress. Although any one of these conditions can cause enamel hypoplasia individually, a central mechanism has been proposed, Central pathway is osteopenia or metabolic bone disease. The cause of osteopenia can be: (i) Nutritional insufficiency of calcium, (ii) Gastrointestinal malabsorption, (iii) Impaired vitamin D metabolism.

It is hypothesised that the entry of calcium and phosphorous into the developing tooth germs is affected sufficiently to cause enamel defects when there is depletion of bone mineral deposits.

EFFECTS OF PRETERM BIRTH ON DENTAL ENAMEL:

Changes in dental enamel is one of the most noticeable oral defects of preterm birth and can present as enamel hypoplasia or enamel opacities. These defects are located in primary teeth which were undergoing mineralisation at the time of birth. The lower the birth weight of infant and gestational age, greater is the prevalence of enamel defect. Seow et al noted 100% prevalence of defects in primary dentition in preterm infants with neonatal rickets.

In the permanent dentition the first permanent molars and incisors are affected as there is persistence of the metabolic derangement in the first few months after birth.

Liver and renal immaturity results in defective conversion of vitamin D to its active metabolites. Also, it is difficult to supply and ensure absorption of calcium, phosphate and vitamin D to premature infants. Human breast milk/ milk formulae do not supply sufficient calcium and phosphorous to match the estimated intra-uterine supply which otherwise it would have received during preterm