Review

Molar incisal hypoplasia - An insight

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Abstract

Hypoplastic molars and incisors are more susceptible to caries. Prevalence of MIH in the literature ranges from 3.6-25%. It has multiple etiological factors which include, systemic factors like birth trauma, infection, nutritional and metabolic disorders and exposure to chemicals such as PCBs (Polychlorinated Biphenyls), Clinical significance of the condition includes poor esthetics, tooth sensitivity and .mal-occlusion, which may provide diagnostic clues as to genetic influences, systemic diseases and trauma. Inherited types form a relatively small component overall, including genetic abnormality of enamel formation. Developmental enamel defects range in the prevalence of 4-60%. Many of these individual factors act through a central mechanism of mineral deficiency. For children with repeated illness in the first year after birth and in children with opacities on erupted molars and incisors it seems useful to increase the frequency of dental check- ups.

Keywords: Hypoplasia, molars, incisors, etiology, incidence, prevalence.

Introduction

Enamel hypoplasia is a defect of enamel matrix formation caused by an insult to the ameloblasts (enamel forming cells). Clinically the defect appears as opacities, coalesced pits, grooves or missing enamel. It is also seen in genetic disorders like Ehlers-Danlos syndrome, Chromosomal anomalies and inborn errors of metabolism along with nutritional deprivation and chronic illness. Other contributing factors include degree of prematurity, mineral deficiency, duration of breast feeding and racial factors. Prevalence of enamel defects were more common in malnourished children within first year of life as compared to well nourished, indicating that malnutrition in children less than three years may be a suggestive etiology.

Enamel defects are scored by modified DDE Index. (Developmental Defects of Enamel)[1].

Normal - Enamel surface is smooth with a pale creamy white colour.

Opacity - A qualitative defect identified visually as an abnormality in the translucency of enamel characterized by white, yellow, brown or a creamy colour where enamel is discoloured.

Enamel Hypoplasia pits - A quantitative defect of enamel visually and morphologically identified as shallow deep row of pits dispersed over parts or entire tooth surface.

Enamel Hypoplasia pits diffuse type - Defective enamel appears as small or large, wide or narrow grooves dispersed vertically or irregularly over the tooth surface.

Enamel Hypoplasia pits linear type - A quantitative defect of enamel morphologically identified with continuous grooves in linear horizontal fashion across tooth surface.

Enamel Hypoplasia pits missing enamel - Defective enamel is widespread over major part of tooth surface and enamel appears partially or completely absent in such a way that the general form of the tooth may be affected.

Pathogenesis

During the initial stages, ameloblasts secrete an enamel matrix, which gets degraded latter and is followed by rapid influx of calcium and phosphate ions.

In malnourished children, the relative protein and mineral imbalance may be responsible for Enamel hypoplasia either at the level of protein synthesis or during rapid influx of calcium and phosphate ions. Synergism between malnutrition and infection may lead to defect in growth and development.

Amelogenesis has been divided into three stages namely, secretary, transitional and maturational stages.[2]

Stage 1

During the secretary stage, ameloblasts secrete large amounts of enamel matrix proteins within which long thin ribbons of enamel mineral, mainly hydroxy -apatite are formed, almost immediately as the matrix is laid down. Enamel formation starts at cusp tips and extends in cervical direction. Through- out the secretary stage enamel crystals grow primarily in length and thickness. The mineral phase is approximately 10-20% by volume, with the remaining portion being occupied by matrix proteins and water.

Stage 2

Once the full thickness of enamel has been deposited, the secretary ameloblasts transform through a short transitional phase into maturation stage ameloblasts responsible for enamel matrix degradation. This is accompanied by massive mineralization of enamel.

Stage 3

The mature ameloblasts regulate the final mineralization of enamel. The enamel layer hardens as the crystallites grow in width and thickness resulting in mineralized tissue that contains more than 95% by weight.

Tooth development is genetically controlled, but sensitive to environmental disturbances. Once teeth are formed, they do not undergo remodeling therefore the effects of an insult to the ameloblasts are detectable as defects in mature enamel. In general, the systemic factors that disturb the ameloblasts during the secretary stage, cause restriction of crystal elongation and result in pathologically thin or hypoplastic enamel.

Disturbances during the transitional and maturational stages of amelogenesis results in pathologically soft enamel of normal thickness. At the early stage of maturation, the ameloblasts are highly sensitive to environmental disturbances[3].

First permanent molars (FPM) start to develop during fourth month of Intra-uterine life. Mineralization starts at cusp tips soon after birth. By six months four cusps unite. By end of first year deposition of enamel matrix is completed in occlusal half of the crown and maturation is ongoing. Enamel formation as a whole takes approximately thousand days and 2/3 of this time is devoted to maturation stage of amelogenesis. Enamel hypomineralization of systemic origin of four first permanent molars and incisors is called MIH. Below the affected enamel, 5% reduction in mineral content of enamel and dentin was reported in MIH teeth.

Discussion

Infectious childhood diseases, high fever, medications (antibiotics), environmental toxins, otitis media[4], pneumonia[5], asthma[6] and chicken pox have been associated with MIH. During the pre-natal period, medical problems like UTI were associated with MIH. Use of amoxicillin during first year of life has been found to increase risk of MIH and fluoride-like defects in permanent incisors and first permanent molars. A significantly high risk for enamel defects was noted in first permanent molars for children with higher intake of macrolides over the first three years of life. The prevalence was also higher in children living in PCB contaminated areas than in a controlled area in Slovenia. In a Finnish study, a significant correlation between MIH and the exposure of children to dioxins via mothers' milk was found[10]. A Turkish study showed a similar prevalence of MIH in children living in an Urban area polluted by dioxin[11].

Several animal studies have shown that teeth are among the most sensitive organs to the effects of dioxin[12]. The most toxic dioxin congener 2,3,7,8 tetrachloro-dibenzoparadioxin arrests degradation and or removal of enamel matrix proteins in developing molars of rat pulps. As a pre-requisite for the completion of enamel mineralization is the removal of enamel matrix, this apparently leads to disturbance in mineralization[13].

Fluoride is thought to affect crystal formation mainly during the maturation stage, inducing defects described as diffuse opacities. A very substantial majority of studies have reported a strong association between the diffuse defects and the level of fluoride in drinking water or Fluoride supplementation. No association between the prevalence of demarcated opacities and fluoride exposure has been found.[15,16,17,18]

A critical review[19] showed evidence that dioxin and Polychlorinated Biphenyl exposure was involved with increased evidence for combination effects.

Conclusion

The notion behind this review is to highlight the clear-cut outlines on suggestive etiological aspects of MIH. Although number of factors has been investigated etiology still remains unclear. It is likely that MIH is not caused by one specific factor but by many different factors. Several harmful agents and conditions may act together and increase the risk of MIH additively or synergistically. Harmful health conditions or agents can affect during pre, perinatal and postnatal period. The problem of most clinical studies relating to MIH or MIH lesions with medical conditions so far is that they are retrospective.

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