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Molar Incisor Hypomineralisation- An Overview

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Abstract

Molar Incisor Hypomineralisation is a deficit in the mineralisation process resulting from a lack of calcium and phosphate fixing on the matrix formed by the ameloblasts commonly found in permanent first molars and, less frequently, incisors. The resulting enamel is not completely mineralised, causing yellowish, hypersensitive enamel, with little resistance. The aetiology of MIH is multifactorial and mainly genetic and systemic factors are involved. This defect must be diagnosed and managed in early stages. This article aims to highlight different aspects related to MIH, from its prevalence to treatment options in young patients.

Keywords: Molar Incisor Hypomineralisation, First permanent molars, Enamel opacities

Introduction

Molar-Incisor hypomineralization is defined as a hypomineralization of systemic origin that affects one to all of the first permanent molars and is often associated with affected permanent incisors (Weerheijm et al., 2001) The condition is also known as nonfluoride enamel opacities, internal enamel hypoplasia, nonendemic mottling of enamel, opaque spots, idiopathic enamel opacities, enamel opacities, and idiopathic enamel hypomineralization.[1] The prevalence of MIH exists with rates varying from 3.6% to 37.5%[2] Several etiological factors are involved in this condition :- Systemic factors such as birth trauma , infection, prematurity, Genetic disorders such as Ehlers – Danlos syndrome, chromosomal anomalies and inborn errors of metabolism along with nutritional deprivation and chronic illness. Other contributing factors include mineral deficiency, Exposure to chemicals such as polychlorinated Bisphenyls (PCB's)., duration of breast feeding. Prevalence of MIH is more common in malnourished children with in first year of life as compared to well nourished child. Clinically MIH usually presents on the buccal or occlusal surfaces of the molars and incisors. The affected molars are sensitive to cold and appear to be more difficult to anaesthetise. The lesions on the incisors are usually not as extensive as those in the molars and present mainly a cosmetic problem. The remaining permanent dentition is usually not affected. The defect appears as white/yellow/brown opacities, coalesced pits, grooves. The hypomineralized enamel can be soft, porous, or resembling discolored chalk or old Dutch cheese. The enamel defects always show a sharp

demarcation between the affected and sound enamels. The porous, brittle enamel can easily chip off under masticatory forces. The defects of incisors are usually without loss of enamel substance and are generally less serious than those seen in molars due to absence of chewing forces.[3]

Mathu-Muju and Wright [4] had classified MIH into three severity levels:

1. Mild MIH: The demarcated opacities located at non-stress bearing areas, no caries associated with the affected enamel, no hypersensitivity and incisor involvement is usually mild if present

2. Moderate MIH: The demarcated opacities present on molars and incisors, the post-eruptive enamel breakdown limited to one or two surfaces without cuspal involvement, atypical restorations can be needed and normal dental sensitivity

3. Severe MIH: Post-eruptive enamel breakdown, crown destruction, caries associated with affected enamel, history of dental sensitivity and aesthetic concerns.

Anatomopathology

Hypomineralisation in MIH begins at the amelodentinal junction (ADJ) and not at the surface of the enamel.[5] In mild MIH the hypomineralisation remains limited to the inner enamel while the outer surface is intact. In severe MIH the whole enamel layer is hypomineralised. During initial stage ameloblasts secrete an enamel matrix, which gets degraded latter and is followed by rapid influx of calcium and phosphate ions. In malnourished children, relative protein and mineral imbalance may be responsible for MIH, either at the level of protein synthesis or during influx of calcium and phosphorus ions. The affected enamel has 20% less mineral concentration according to an investigation by Fearne et al.[6] while the protein content in MIH enamel is three-fold to 15-fold higher than that of sound enamel.

Differential Diagnosis

MIH diagnosis might be difficult to outpoint when the permanent first molars are already very damaged and decayed and have been extensively restored, have unusual occlusal topography or have already been extracted.[7] It is essential to distinguish between MIH and other abnormalities in the dental structures. MIH can sometimes be confused with fluorosis or amelogenesis imperfect. It can be differentiated from fluorosis as its opacities are demarcated, unlike the diffuse opacities that are typical in fluorosis. Fluorosis can be related to a period in which the fluoride intake was too high. Fluorosis is caries resistant and MIH is caries prone. A patient's detailed history is necessary for seeking aetiologies. Only in very severe MIH cases, the molars are equally affected and mimic the appearance of Amelogenesis Imperfecta. In MIH, the appearance of the defects will be more asymmetrical whereas, In AI, the molars may also appear taurodont on radiograph. There is often a family history present. Administration of tetracycline during pregnancy and to children under 6 causes changes to grey and yellowish color of temporary and permanent teeth. If the dose is high, hypoplastic changes occur in the enamel. With calcium, tetracycline forms a chelate complex. This complex becomes irreversibly fixed to the enamel and dentine during formation of tissues in the tooth [Schroeder, 1991].[8]

Treatment Modalities

Restoring Hypomineralized Permanent Incisors And Molars

Yellowish or brownish discoloration in permanent incisors may pose an esthetic issue, which is an important issue to resolve. Minimally invasive techniques are necessary to treat such cases. Cavity design plays a critical role, as defective enamel remnants compromise the end result. It is recommended that the cavity design should involve

removal of all the porous but not necessarily discoloured enamel, until resistance to the bur or to the probe is achieved.[9] Glass ionomer cement (GIC) or resin modified GIC restorations can be considered only as an intermediate approach until definitive restoration is placed.[10] Conservative approach should be used as the first line of treatment before more invasive treatment such as resin restorations/vesneers or crowns that may create problems, resulting from the large pulp size and immature gingival contours in young incisors. Resin composite is the material of choice and recommended for one to three surface restorations[10] and the pre-treatment with 5.25% sodium hypochlorite can improve the bond strength[11]

Bleaching: Bleaching is an another modality especially recommended for adolescents. The possible side-effects of bleaching are: sensitivity, mucosal irritation, and enamel surface alterations. The combined use of CPP-ACP Tooth Mousse and bleaching gel is recommended.[9] as the CPP-ACP Tooth Mousse will protect the tooth structure and remineralise the MIH opacities without interfering with bleaching effect.[12] The combined use of hydrogen peroxide and CPP-ACP, could be done with a ratio range from 1:6 to 3:4, depending on the opacity response to the bleaching agent.[12]

Extraction: In severe cases, where first permanent molars are severely affected, extraction can be done so as to give the second permanent molars (SPM) an opportunity to drift into the FPM position. Full dental assessment before extraction should be carried out to check for the presence, position and normal formation of the developing permanent dentition to ensure favourable orthodontic conditions. Therefore, it is advisable to seek an orthodontic opinion before extraction and consideration should be given to further FPM extractions for balancing and compensation reasons.[13]

Clinical Difficulties During Trearment[9,14]

Anaesthetic Difficulty: Hypersensitivity and rapid damage to the tissues will cause chronic pulpal inflammation, preventing effective local anaesthesia . Loco-regional techniques (Spix), osteo-central techniques or even electronicallyassisted local anaesthesia, such as Quick Sleeper or Wand, may be useful in such cases.[9]

Difficulty In Restoring Teeth as there is difficulty of adhesion of restoration on soft, hypomineralised enamel, so the risk of early loss of restoration and development of secondary decay is higher.

Tooth loss is a major issue in a child having Molar incisor Hypomineralization.

Limited cooperation of child.

Post-eruptive enamel breakdown leading to dentine exposure and this makes the tooth at risk of pulp involvement

Aesthetic problems in anterior teeth

Financial issue for families

Conclusion

Molar-Incisor Hypomineralisation (MIH) is a congenital disease which affects permanent first molars and, often to a lesser degree, permanent incisors with variable severity. The aetiology is unknown, but different hypotheses have been advanced. It is important to diagnose this defect at early stage and must not be confused with other diseases. So, a dentist must know the clinical features of MIH so as to diagnose it at initial stages, which further prevent its progression. Treatment consists in a minimally invasive approach by reinforcing and protecting the existing dental structure. In more severe cases, extraction may be indicated.

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