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Molar Incisor Hypomineralisation - A Review

Research Article

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Abstract

Molar Incisor Hypomineralisation (MIH) is a type of enamel defect affecting the first molars and incisors in permanent dentition. It usually occurs in children under 10years. It is caused due to lack of mineralisation of Enamel. The enamel appears yellow, brown or white in colour. Children with MIH are more likely to experience tooth decay. Prevention is important at early developmental age to avoid the severity of the MIH. The review concentrates on the diagnosis, features, prevalence, and clinical management of molar incisor hypomineralization.

Keywords: Dentists; Molar; Hypomineralization.

Introduction

Hypomineralisation of systemic origin, presenting as demarcated, qualitative defects of enamel of one to four first permanent molars (FPMs) frequently associated with affected incisors. The occurrence of white to yellow-brown enamel opacities in the first permanent molars was first recognised in Sweden in the late 1970s. This phenomenon was subsequently coined molar incisor hypomineralization (MIH) [7].

Tooth enamel is unique among mineralised tissues because of its high mineral content. Enamel is made up of highly organised, tightly packed crystallites that comprise 87% of its volume and 95% of its weight. Despite its hardness, tooth enamel can be destroyed fairly rapidly by dental caries. Additionally enamel is also afflicted by various structural defects which could be inherited or acquired [10].

These defects, characterized by discoloured opacities or a total absence of enamel, are observed to most commonly affect the first permanent molars (FPMs) with or without involvement of the permanent incisors and have been varyingly referred to in the literature as hypomineralized FPMs, idiopathic enamel hypomineralization, demineralized FPMs, non-fluoride hypomineralization, cheese molars, internal enamel hypoplasia, non-endemic mottling of enamel, opaque spots and enamel opacities [13].

Clinically, MIH can vary from mild demarcated opacities to severe structural loss. The defects can be white, yellow, or brown in colour, but they will show a clear demarcation between the affected and normal enamel (Weerheijm 2004).

MIH's Clinical Management Is Challenging Due To:

(i) The sensitivity and rapid development of dental caries in affected PFMs.
(ii) The limited co-operation of young children.
(iii) Difficulty in achieving anaesthesia; and
(iv) The repeated marginal breakdown of restorations [12].

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The purpose of this paper is to describe the diagnosis, prevalence, and clinical management in molar incisor hypomineralization.

Etiology

MIH was originally described as an idiopathic defect and a clear etiology for the condition is yet to be defined. Animal models suggested preceding events such as hypoxia, high fever, hypocalcaemia, exposure to antibiotics (amoxicillin), and dioxins as possible causes of MIH. When rats were exposed to a daily low dose of bisphenol A (BPA), an endocrine disrupting chemical, the exposed rats developed an enamel hypomineralization condition very similar to the human MIH.

BPA mainly targeted two genes (kallikrein-related peptidase 4, Klk4, and enamelin, Enam), which are responsible for enamel matrix protein secretion and enamel matrix degradation (to allow enamel mineral crystal growth), respectively. It was found that modulation of the expression of these genes led to enamel hypomineralization. BPA stimulates activity on ameloblast proliferation and gene transcription and provides evidence for a hormonal influence on amelogenesis, demonstrating that dental epithelial cells are estrogen targets [6].

Therefore, we propose that MIH is not an idiopathic but a genetic condition related to disturbances in the maturation stages of enamel, which in most instances are localized to first permanent molars and incisors. On occasion, second primary molars and permanent canines and premolars can also be affected. The involvement of additional teeth may be due to the influence of additional gene variants in any of more than 100 genes expressed during late enamel development.

Signs and Symptoms

Large demarcated opacities, whitish-cream or yellow-brown in colour.

May or May not be associated with post eruption enamel breakdown.

Hypersensitivity.

Difficult to anaesthetise.

Rapid caries progression.

The opacities are usually limited to the Incisal or cuspal one third of the crown, rarely involving the cervical one third, the subsurface enamel is soft and porous [5].

Characteristics Of Affected Teeth

MIH is a qualitative defective enamel classified as a hypomineralized type that follows the normal incremental lines of enamel formation, from cuspal to cementoenamel junction.

MIH enamel has substantially higher protein content than normal enamel, but a near normal level of residual amelogenins, this characteristic distinguishes MIH from hypomaturation, defects that contain high residual amelogenins such as Fluorosis [3, 8].

Clinical Implications

Clinically the affected teeth can be very sensitive to stimuli like a current of cold or warm air and mechanical provocations. The children with MIH molars or opacities on the incisors should be monitored carefully until all four permanent first molars have erupted. If molars show signs of opacities and post eruptive breakdown, a child should be seen every three months until the time when the permanent first molars have completely erupted [1].

Management Of The MIH

Ultrastructurally, opaque defects on anterior teeth usually extend through the full thickness of enamel, from the surface or subsurface down to the dentinoenameljunction (DEJ). For this reason, acid or pumice microabrasion techniques tend to produce little improvement when used alone. Direct composite veneering with or without preparation offers the most reliable medium term way of improving aesthetics of these teeth.

Hypomineralized enamel is very susceptible to decay and acid attack. An assessment of your child's diet should be carried out and appropriate recommendations made for dietary changes. In cases where tooth-brushing is difficult due to sensitive teeth, the following oral hygiene strategies may be helpful:-

Brush affected teeth gently with a fluoride containing desensitising toothpaste.

Apply tooth mousse TM Plus daily [9].

Thorough oral hygiene should be instituted; this could include a desensitising toothpaste. Remineralisation therapy should commence as soon as the defective surface is accessible, aiming to produce a hypomineralized surface layer and to desensitise the tooth. Remineralisation and desensitisation may be accomplished with casein phospho - peptide - amorphous calcium phosphate(CPP-ACP) oral care products. CPP-ACP care products enhance remineralisation by creating a state of supersaturation followed by deposition of calcium and phosphate ions at the enamel surface. While clinical protocols for CPP-ACP oral care products await development, anecdotal reports describe surface hardening and reduction in tooth sensitivity from daily home use [4].

Generally, the defects of the incisors are milder than those of the molars. Since masticatory forces on the opacities in incisors are absent, the enamel substance does not disintegrate after eruption. However, treatment is often required for aesthetic reasons. In such cases (and in the rare case of breakdown of the enamel), replacement with composite should be considered as first treatment option [2].

Conclusion

The prevalence of the MIH appears to be increasing and management of MIH affected children is common. As theetiology is multifactorial, children with poor general health should be considered more as they are at risk to develop MIH. Frequent monitoring of these patients is required as preventive measures can be instituted at the earliest.

Hence research on etiology factors and preventive measures are required.

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