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Review Article

Molar-Incisor Hypomineralization: Review of its Prevalence, Etiology, Clinical Appearance and Management

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Abstract

Molar-incisor Hypomineralization (MIH) is defined as hypomineralization of systemic origin of one to four permanent first molars frequently associated with affected incisors. Affected molars may present major clinical problems in terms of extensive tooth structure loss, hypersensitivity and difficulty in restoration and retention. The prevalence of MIH ranges from 2.5–40% and seems to differ in certain regions and birth cohorts. Several factors are associated with etiology of MIH. The general dental practitioners should be aware that MIH is common and should be able to diagnose and manage at the early and appropriately.

Keywords: Developmental Enamel Defects; MIH; Molar-Incisor Hypomineralization; Post-Eruptive Enamel Breakdown.

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Introduction

The Permanent first molars (PFMs) are considered the most caries-susceptible teeth in the permanent dentition because of their early eruption.[1] A very common factor predisposing these teeth to caries which is also the most frequently unrecognized or overlooked is a developmental defect in the enamel referred to as Molar-Incisor Hypomineralization (MIH).[2] First recognized in the late 1970s by Swedish dentists working within the public dental services, it may have existed long before it was identified.[3] However, when caries prevalence is high, the developmental defect responsible for initiation of the cavity is probably masked.[4]

The dental literature contains an abundance of terms that have been used to refer to hypomineralized molars including 'non-fluoride enamel opacities', 'internal enamel hypoplasia', 'non-endemic mottling of enamel', 'opaque spots', 'idiopathic enamel opacities', 'cheese molars', 'hypomineralized PFMs', 'idiopathic enamel hypomineralization' and 'dysmineralized PFMs'.[4,5] It was in 2001 that Weerheijm et al[6] coined the term Molar-Incisor Hypomineralization and later defined MIH as 'a hypomineralization of systemic origin of 1–4 permanent first molars, frequently associated with affected incisors'. For a patient to be diagnosed as suffering from MIH, at least one permanent first molar must be affected with or without involvement of the incisors.[7] The term 'Molar Hypomineralization (MH)' has been used, sometimes, to distinguish children in whom the incisors are not affected.[8]

Prevalence of MIH

After initial presentation and establishment of diagnostic criteria[7] for MIH many prevalence studies for MIH were carried out in various countries and large variations found in the prevalence rates, ranging from 2.5-40.2%.[8-30] This wide range could be because of difference in recording methods, indices used and different age or population investigated.[4,9,10] In some countries, caries levels may mask the true prevalence of MIH.[4,9,10] No much difference in prevalence has been reported so far between the male and female genders.[16,18,25]

Characteristic features of MIH-affected teeth

a. Clinical presentations

Molars and incisors affected by MIH exhibit demarcated opacities, which are whitish-yellow or yellowish-brown in color.[3,7,31] The affected PFM may undergo post-eruptive enamel breakdown because of occlusal loading, whereas incisors rarely exhibit post-eruptive enamel breakdown.[2,10,32] MIH should not be mistaken for enamel hypoplasia, which is a quantitative developmental defect resulting from deficient enamel matrix formation.[4,8] Clinically, in cases of hypoplasia, the margins are smooth, while in MIH the borders are

irregular.[2] Affected molars may at times be hypersensitive and difficult to anesthetize.[2,33,34] The structural defect of enamel of the molars affected by MIH may lead to early caries involvement and rapid progression which may be hastened by the difficulty in brushing those acutely sensitive teeth.[2,8,32] (Figure 1 & 2)

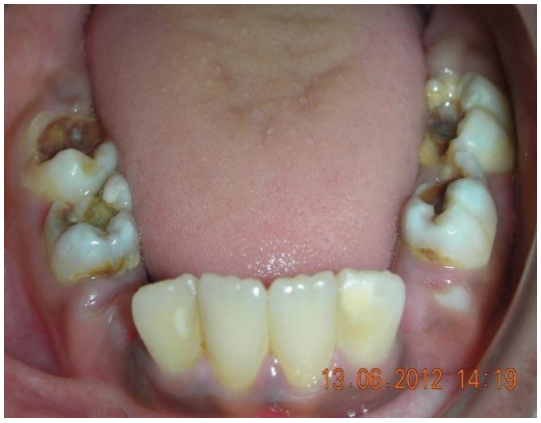

Figure 1: Extensive enamel destruction of hypomineralized PFMs and demarcated opacities on lateral incisors

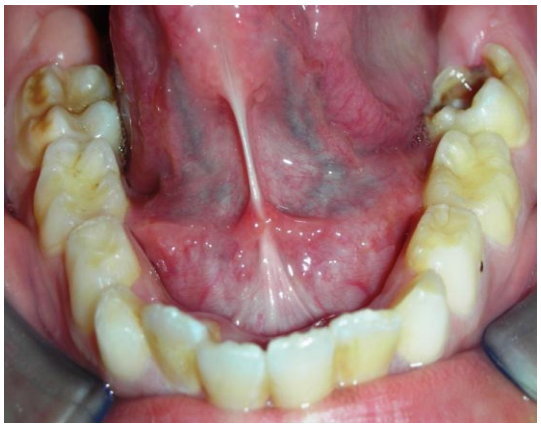
Figure 2: Different presentations of MIH in the same arch

Older children seem to have more severe lesions than younger ones which may be because the affected teeth have undergone post-eruptive enamel breakdown under masticatory load. Severity of MIH can vary between different individuals but it can also vary within the mouth of a single individual, such that not all FPM will be affected to the same degree, indeed some molars may apparently be unaffected in some cases.[2,4,5,9]

b. Microstructure of hypomineralized enamel

Severity of hypomineralization correlated positively with increasing carbon and decreasing calcium and phosphorus concentrations using secondary ion mass spectrometry and X-ray microanalysis.[5,32,35] Yellow-brown defects have lower Knoop hardness values and greater porosity than white defects and normal enamel.[5] Nano-indentation studies have shown significantly lower values for hardness and modulus of elasticity than seen in unaffected enamel[36]. Under scanning electron microscopic analysis, these defects revealed increased porosity and disorganized rod structure of fractured surfaces.[36]

c. Surface protein content of hypomineralized enamel

Hypomineralized enamel was found to have from 3- to 15-fold higher protein content than normal, but a near-normal level of residual amelogenins. Hypomineralized enamel was found to have accumulated various proteins from oral fluid and blood, with differential incorporation depending on integrity of the enamel surface. Pathogenically, these results point to a pre-eruptive disturbance of mineralization involving albumin and, in cases with post-eruptive breakdown, subsequent protein adsorption on the exposed hydroxyapatite matrix.[37]

d. Dentin characteristics of MIH-affected teeth

Fagrell et al[38] have found that oral bacteria may penetrate through hypomineralized enamel into the dentinal tubules and create inflammatory reactions in the pulp, thus possibly contribute to hypersensitivity of teeth with MIH. In sections where bacteria were found in the cuspal areas or deeper in the dentin, a zone of reparative dentin was found, and in sections from one tooth, the coronal pulp showed an inflammatory reaction with inflammatory cells. The dentinal tubules with odontoblastic processes were filled with bacteria.

e. Pulpal status of hypomineralized first permanent molars

It was found that the pulps of non-carious hypomineralized PFMs present the changes indicative of inflammation. Innervation density was significantly greater in the pulp horn and subodontoblastic region of hypomineralized teeth than in sound teeth. Immune cells were most abundant within pulps of hypomineralized teeth exhibiting enamel loss. Vascularity was found to be similar for both hypomineralized and sound teeth, but was significantly greater in hypersensitive hypomineralized samples.[34]

Clinical importance of diagnosing MIH

Clinically, hypomineralized molars can create serious problems for the dentist in terms of management as well as for the child due to severity of affected teeth.[5,33] For dentists, the problems are related to unexpectedly rapid caries development in the erupting first permanent molar[2,4,31-33], difficulty in anaesthetizing the MIH molar when treatment is indicated[2-5,33,34] and unpredictable behavior of apparently intact opacities in terms of esthetics and retention of restorations.[5] The child, on the other hand, will experience pain and sensitivity during brushing and eating.[5,34] General dental professionals should be able to recognize it early and manage appropriately for the long-term outcomes of affected children.

Diagnostic Criteria for MIH (Table 1)

Subsequent to European Academy of Paediatric Dentistry seminar held at Athens in 2003, the judgment criteria for MIH in epidemiological studies have been established.[7] For a patient to be diagnosed as suffering from MIH, they have to have at least one PFM affected with or without the involvement of incisors. However, if a patient has opacities affecting the incisors only, the condition is not MIH.[7,31] The recommended age for examination of children is 8 years.[3,7,9,31] Index teeth (4 PFMs and 8 erupted permanent incisors) should be cleaned thoroughly and kept wet for examination to distinguish from incipient carious lesions.[7,9]

Criteria	Definitions
Permanent first molars and incisors	One to all four permanent first molars shows hypomineralization of the enamel. Simultaneously, the permanent incisors can be affected. To diagnose MIH, at least one PFM has to be affected. The defects can also be seen in second primary molars, incisors and the tips of canines. More the molars and incisors affected, the more severe is the defect.
Demarcated opacities	The affected teeth show clearly demarcated opacities at the occlusal and buccal parts of the crown. The defects vary in colour and size. The colour can be white, creamy or yellow to brownish. The defect can be negligible or comprise the major part of the crown. It is recommended that defects less than 1 mm not be reported.
Enamel disintegration	The degree of porosity of the hypomineralized opaque areas varies. Severely affected enamel subjected to masticatory forces soon breaks down, leading to unprotected dentin and rapid caries development.
Atypical restorations	PFMs and incisors with restorations revealing similar extensions as MIH are recommended to be judged as affected.
Tooth sensitivity	The affected teeth may be reported frequently as sensitive, ranging from a mild response to external stimuli to spontaneous hypersensitivity; these teeth are usually difficult to anaesthetize.
Extracted teeth	Extracted teeth can be defined as having MIH only in cases where there are notes in the records or demarcated opacities on the other PFM. Otherwise it is not possible to diagnose MIH.

Table 1: European Academy of Paediatric Dentistry recommended diagnostic criteria for MIH

In cases of large caries lesion with demarcated opacities at the border of the cavity or on the non-carious surfaces, the teeth should be judged as having MIH. Other changes in the dental enamel such as amelogenesis imperfecta, hypoplasia, diffuse opacities, white spot lesions, tetracycline staining, erosion, fluorosis, white cuspal and marginal ridges should be excluded from the types of enamel defects out lined as above.[7]

Categorization of MIH

Initially hypomineralized areas were categorized into three grades of severity: severe (loss of enamel in association with affected dentin), moderate (loss of enamel only), and mild (colour change: white, yellow

or brown).[39] After encountering certain difficulties in differentiating between moderate and severe cases, Lygidakis et al[31] proposed a classification with mild and moderate-severe categories. The mild category includes intact enamel surfaces with occasional sensitivity and mild aesthetic concerns. Post-eruptive breakdown, persistent or spontaneous hypersensitivity and strong concerns regarding aesthetics form the moderate-severe category. Severity can vary between individuals and can also vary within the mouth of a single individual.

Possible etiology

Many retrospective, cohort and case-control studies have analyzed a wide variety of putative causes for MIH.[5,10,15] Factors associated with disrupted amelogenesis including systemic conditions and environmental insults influencing natal and early development of PFMs have been studied.[11,22,29,40-42]

1. Prenatal period: The last trimester of pregnancy is a critical period during which the amelogenesis of PFMs and incisors teeth starts. Multiple episodes of maternal high fever, viral infections like rubella and chickenpox, prolonged medications during the last month of pregnancy, prolonged vomiting up to last month, urinary infections, maternal hypertension, maternal diabetes, renal deficiency, malnutrition during the last trimester of pregnancy are some of the presumed causative factors listed.[9,10,41,42]

2. Peri-natal period: In the peri-natal period different medical conditions alone or in combination may affect the welfare of a child.[5,10] In a Greek study, the most common peri-natal problems/conditions associated with MIH were caesarian section, prolonged/complicated delivery, premature birth and twinning. Hypoxia, low birth weight, hemorrhage and detachment during delivery are some more supposed peri-natal causes for defective ameloblast function.[42]

3. Post-natal period: Special attention has been paid to prolonged childhood illnesses, prolonged high fever due to infections, repeated/prolonged medications (antibiotics like amoxicillin)[40-42] and exposure to environmental contaminants such as polychlorinated biphenyls and polychlorinated dibenzop-dioxins / dibenzofurans (dioxins). Infections such as otitis media, pneumonia, asthma, bronchitis, upper respiratory tract infections, urinary tract infections and exanthamatuos diseases like chickenpox, rubella, measles have been positively associated with MIH. Hypocalcaemia, nutritional deficiencies, brain injury and neurologic defects, cystic fibrosis, syndromes of epilepsy and dementia, nephritic syndrome, atopia, lead poisoning, repaired cleft lip and palate, radiation treatment, rubella embryopathy, epidermolysis bullosa, ophthalmic conditions, celiac disease, and gastrointestinal disorders have also been suggested as possible causes. Coeliac disease is one of the most commonly reported conditions associated with enamel defects.[10,15,40-43]

On the other hand, a few other researchers have contemplated that there could be a genetic component responsible for MIH and suggested that family studies may provide further information.[41,44] Due to the developmental timing of the permanent first molars and upper and lower anterior teeth, the search for a cause has centered on the time of birth and early childhood.[5,43] However, Crombie et al[10], in a critical review on etiology of MIH, have concluded that current evidence is insufficient to establish any particular aetiological factor(s). Alaluusua[43] has opined that MIH is not caused by one factor alone but by many different ones, and that several factors may act together at sensitive stages of amelogenesis, increasing the risk.

Pathogenesis

Enamel is a highly mineralized tissue of ectodermal origin, secreted from ameloblasts that differentiate from the internal dental epithelium. Hypomineralization is thought to follow deposition of the full thickness of enamel matrix. The transitional ameloblast is considered most vulnerable and when these cells do not undergo complete maturation, full-thickness hypomineralization occurs.[5]

Enamel maturation involves: a) the removal of acid-labile mineral; b) replacement with more acid-resistant apatite; and c) an influx of calcium and phosphate ions, increasing the crystal width and thickness.[45] Disturbed resorptive potential of ameloblasts and inhibition of proteolytic enzyme leading to protein retention and interference with crystal growth and enamel maturation may result in enamel hypomineralization.[5]

Management

Treatment of MIH affected molars and incisors depends upon a number of factors like age of the child, severity of the condition, the social background and expectation of the child and parents. Clinical management of teeth affected by MIH is challenging due to the sensitivity and rapid development of dental caries in affected PFMs, the limited cooperation of a young child, difficulty in achieving anesthesia and the repeated marginal breakdown of restorations.[5] Neglect of such teeth may result in early and fast pulpal infection which may be life threatening in some cases.[46]

A very useful six-step management approach for a child with MIH has been proposed by William et al.[5]

1. Risk identification: Assess medical history for putative etiological factors.
2. Early diagnosis: Examine at-risk molars on radiographs if available and monitor these teeth during eruption.
3. Remineralization and desensitization: Apply localized topical fluoride.
4. Prevention of dental caries and post-eruptive enamel breakdown (PEB): Institute thorough oral hygiene home care program, reduce cariogenicity and erosivity of diet, and place pit and fissure sealants.
5. Restorations or extractions: Place intra-coronal (resin composite) restorations bonded with a self-etching primer adhesive or extra-coronal restorations (stainless steel crowns). Consider orthodontic outcomes post-extraction.
6. Maintenance: Monitor margins of restorations for PEB. Consider full coronal coverage restorations in the long term.

Research has found that, by the age of nine years, MIH-affected Swedish children have undergone dental treatment on their permanent first molars nearly ten times more often than healthy controls and that each defective tooth has been treated, on average, twice.[33] Therefore behavior management problems and dental fear and anxiety are more common in affected individuals than in controls.[2,4,33] It has also been reported that additional treatment or retreatment was required in almost half of 18-year-old Swedish children with MIH who had had their molars restored.[47] Once the MIH children are identified, dietary counseling should be given to the parents and children. Children should be encouraged to maintain good oral health and use fluoridated toothpaste. Remineralizing agents like topical fluoride varnishes, casein phosphopeptide-amorphous calcium phosphate may help in reducing sensitivity and aid mineralization of the hypocalcified areas.[4,5,31] Fissure sealants may be useful for PFMs that are mildly affected and where the enamel is intact. Long-term frequent follow up is mandatory since chances of failure and replacement requirements are high.[4,5,31] Composite resins in small 1- or 2-surface cavities and full coverage restorations like stainless steel crowns are the better option for teeth with extensive tooth structure loss.[4,5,31]

Extraction may be an option for PFMs with non unrestorable crown or poor long-term prognosis. However extraction must be followed by occlusal guidance in very young children (to guide the second molar into the position of the first molar) or orthodontic correction of resultant malocclusion.[4,5,31,48] Partially erupted PFMs affected by severe MIH are a challenge to clinicians since it is difficult to define the margins when a portion of tooth is covered by peri-coronal flap. A desensitizing paste or fluoride varnish could be applied to minimize sensitivity and to increase post-eruptive maturation of enamel. Once these teeth erupt completely full coverage crowns can be placed.[5]

Hypomineralized areas on incisors of MIH affected children are frequently a cause for esthetic concern for the patient and their parents. Micro-abrasion and bleaching may work for the full thickness yellow or brownish-yellow defects but not for creamy yellow or whitish-cream defects located in the inner part of the enamel.[4,5,31,32] Mild hypomineralized areas do not require any treatment unless in case of hypersensitivity, at least in a very young child. Direct and indirect composite veneers can improve aesthetics, but they might have to be modified or replaced as the child ages.[4,5,31,32]

Conclusions

The first permanent molar has a significant role in development of occlusion and its early loss can have a considerable effect on dental health of the child in future. Hence for a child with a positive medical history during the mother's pregnancy or during the first

three years of life, eruption of first permanent molars should be monitored with a greater care so that the early tooth structure loss and early carious involvement can be prevented. The general dental practitioner should be aware that MIH is common and should be able to diagnose and manage at the early and appropriately.

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