Molar Incisor Hypomineralization: A Literature Review

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ABSTRACT

In this paper, the current knowledge about Molar Incisor Hypomineralization (MIH) is presented. MIH is defined as hypomineralization of systemic origin of one to four permanent first molars frequently associated with affected incisors and these molars are related to major clinical problems in severe cases. The prevalence of MIH in the different studies ranges from 3.6–25% and seems to differ in certain regions and birth cohorts. Several aetiological factors (for example, frequent childhood diseases) are mentioned as the cause of the defect. Children at risk should be monitored very carefully during the period of eruption of their first permanent molars. Treatment planning should consider the long-term prognosis of these teeth.

Keywords: *MIH, first permanent molar, hypomineralization.*

INTRODUCTION :

Dental development and mineralization in humans starts before birth and continues to adolescence when the permanent molars complete their mineralization. The first permanent molar is the first tooth in the permanent dentition to mineralize, a process that starts around birth and is completed at approximately three years of age.[1] Enamel and dentin are formed by secretory cells and the enamel forming cells, the ameloblasts, are highly specialized cells of ectodermal origin.[2,3] The ameloblast has a limited reparative capacity; therefore disturbances occurring during the mineralization of enamel will remain as permanent marks. Defects in enamel quality or in other dental hard tissues are important implications for the understanding of evolution, function, origin and relation to etiological factors behind developmental disturbances but also how environmental factors may influence on the mineralization of the dental hard tissues.

There are basically two major developmental defects; enamel hypoplasia and hypomineralized enamel.[4]

Enamel hypoplasia is defined as a quantitative defect of enamel surface that is macroscopically detectable. It may occur as pits or rows and the defect might be shallow or deep, local or generally distributed all over the enamel.[5]

Hypomineralized enamel is defined as a qualitative defect, identified visually. The color of the defect may be white, brown or yellow. The thickness of the enamel is normal at time of eruption. A posteruptive breakdown (PEB) may be seen. If PEB occurs, fractured edges are seen.

The term molar incisor hypomineralization (MIH) was introduced in 2001 to describe the clinical appearance of enamel hypomineralization of systemic origin affecting one or more permanent first molars (PFMs) that are associated frequently with affected incisors.[6] Also referred to as "hypomineralized" PFMs,[7] "idiopathic enamel hypomineralization," [8,9] "dysmineralized" PFMs,[10] "nonfluoride hypomineralization,"[11,12] and "cheese molars,"[13,14] the condition is attributed to disrupted ameloblastic function during the transitional and maturational stages of amelogenesis.[8,15] The limited prevalence data for MIH reflects several diagnostic classifcations. Using the criteria of Weerheijm et al, the prevalence ranges from 4% to 25%.[16]

DIAGNOSIS

Any examination for MIH should be undertaken on clean wet teeth and the age of 8 years is optimum as at this age all permanent first molars and most of the incisors are erupted. Judgements related to individual teeth (all PFM and incisors) should be recorded, helping in the correct diagnosis of the condition. Diagnostic criteria for hypomineralization of PFMs currently available are the modified defect of dental enamel (DDE) index given by Federation Dentaire International (Table 1) and the criteria of Weerheijm et al (Table 2)

Table 1 : Modified DDE Index (FDI 1992)

MILD	<30% of the tooth's enamel surface area visibly disrupted
MODERATE	31 to 49% of the tooth's enamel surface area visibly disrupted
SEVERE	>50% of the tooth's enamel surface area visibly disrupted

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Table 2: Definitions of the criteria used for diagnosing MIH (Weerheijm at al 2001a)

Criteria	Definitions
Opacity	A defect altering the translucency of the enamel, variable in degree. The defective enamel is of normal thickness with a smooth surface and can be white, yellow or brown in color. The border of the lesion is demarcated
PEB	Posteruptive enamel breakdown. A defect that indicates loss of surface enamel after tooth eruption, usually associated with a pre-existing opacity.
Atypical restorations	Size and shape of restoration do not confirm to typical restorative characterstics. They frequently extend to the buccal or palatal smooth surfaces reflecting the distribution of hypoplastic enamel.
Extraction due to MIH	Absence of molar should be related to other teeth in dentition. Absence of first permanent molar in a sound dentition is suspected to have been a MIH molar.

DIFFERENTIAL DIAGNOSIS

Teeth with developmental defects of enamel may present similarly, regardless of etiology, and the development defects of enamel hypoplasia like fluorosis may be confused with MIH. MIH should thus be differentiated with highly prevalent fluorosis and Amelogenesis Imperfecta (Table 3). It can be differentiated from fluorosis as its opacities are demarcated, unlike the diffuse opacities that are typical of fluorosis and by the structure of the enamel (fluorosis is caries resistant and MIH is caries prone). The cause of fluorosis can mostly, be related to the period in which the fluoride intake was too high. Choosing between amelogenesis imperfecta (AI) and MIH as a diagnosis seems a matter of definition; it should be stressed that, only in very severe MIH cases, the molars are equally affected and mimic the appearance of AI. Mostly in MIH, the appearance of the defects will be more asymmetrical in the molars as well as in the incisors.

Table 3: Clinical criteria for differentiation between MIH, Amelogeneis Imperfecta and Fluorosis:

Condition	Findings
Amelogeneis Imperfecta (AI)	Involves all the teeth, family history is present, teeth may appear taurodont on radiograph
Fluorosis	Diffuse opacities which are caries resistant. Number of teeth involved depends on the time of exposure.
Molar incisor hypominer -alization	Involves PFM and incisors, well demarcated opacities will be present which will be caries prone. Only severe cases may resemble AI. No appearance of taurodont on radiograph.

CLINICAL PRESENTATION :

MIH is a hypomineralized defect of the first permanent molars, frequently associated with affected incisors. The number of affected first permanent molars per patient varies from one to four and expression of the defects may vary from molar to molar. Within one patient, intact opacities can be found on one molar, while in another molar large parts of the enamel break down soon after eruption.[16] A clinical classification of the severity of MIH into three categories (levels of severity) has been suggested: mild, moderate and severe.[12] The risk of defects to the upper incisors appears to increase when more first permanent molars have been affected.[17] The defects of incisors are usually without loss of enamel substance.

Clinically, the hypomineralized enamel can be soft, porous and look like discoloured chalk or old Dutch cheese. The enamel defects can vary from white to yellow or brownish but they always show a sharp demarcation between the affected and sound enamel. The porous, brittle enamel can easily chip off under the masticatory forces. Sometimes, the loss of enamel (posteruptive enamel breakdown) can occur so rapidly after eruption that it seems as if the enamel was not formed initially. After occurrence of the post-eruptive enamel breakdown, the clinical pictures can resemble hypoplasia. In hypoplasia, however, the borders to the normal enamel are smooth, whilst in posteruptive enamel breakdown the borders to the normal enamel are irregular.

MIH molars are fragile, and caries can develop very easily. This problem is aggravated because the children tend to avoid the sensitive molars when brushing their teeth, leading to increased stagnation of food and plaque. The fast caries progression can clinically mask the reason behind the susceptibility for caries (hypomineralisation of the enamel) in these molars. Jälevik and Klingberg [2002] found that, compared with normal molars, MIH molars need ten times more treatment time.[18]

The affected teeth can be very sensitive to a current of air, cold or warm. Even with enamel that has not disintegrated, mechanical stimuli, for instance tooth brushing, may instigate toothache in these teeth. It is believed that there is subclinical pulpal inflammation due to porosity of the enamel which could lead to hypersensitivity.

PREDISPOSING FACTORS:

Hypomineralization is thought to be due to disturbed resorptive potential of ameloblasts and proteolytic enzyme inhibition leading to protein retention and interference with crystal growth and enamel maturation. Most common

predisposing factors for disrupted amelogenesis of PFM include systemic and environmental insults influencing natal and early post natal development.

1) **Systemic illness:** Although a number of etiological factors may contribute to MIH, the threshold level needed to cause enamel defects at sensitive stages of amelogenesis is unknown.[20] Conditions common in the first 3 years, such as upper respiratory diseases, asthma, otitis media, tonsillitis, chicken pox, measles, and rubella, appear to be associated with MIH.[7,13,20] Antibiotic usage has also been implicated. Due to the concurrence of disease and antibiotic therapy, however, it is difficult to ascertain whether the MIH was associated with the disease or the antibiotic.[20] Children with poor general health and systemic conditions are more likely to have developmental defects of enamel.[21-22]The systemic conditions implicated to date include nutritional deficiencies, brain injury and neurologic defects, cystic fibrosis, syndromes of epilepsy and dementia (Kohlschutter-Tonz syndrome), nephritic syndrome, atopia, lead poisoning, repaired cleft lip and palate, radiation treatment, rubella embryopathy, epidermolysis bullosa, ophthalmic conditions, coeliac disease, and gastrointestinal disorders.[21,23-25]

2) **Gestational age:** Preterm birth has been associated with increased prevalence of enamel defects, including hypomineralization and hypoplasia in the permanent dentition.[24,26-28] A study of 32 Finnish children 9 to 11 years old found enamel defects in 36% of children born full term and 84% of children born preterm.[29]

3) **Affect of low pH:** Regulation of pH during mineralization is considered necessary for normal apatite deposition and crystallite growth. Sui et al reported that reduced enamel matrix pH disrupted the crystal growth and proteinase function which can result in protein retention and hypomineralization. Speculatively conditions affecting matrix pH during enamel maturation may predispose MIH. A medical condition affecting the pH like cystic fibrosis has been found to be associated with MIH.

4) **Lack of calcium phosphate:** An optimal serum calcium level is important for initial dentin mineralization and proper enamel matrix secretion and mineralization. Impaired calcium metabolism plays a role in development of hypomineralized enamel. Studies using secondary ion mass spectrometry and x – ray microanalysis revealed that increased severity of hypomineralization correlated positively with increasing carbon concentration and decreasing concentration of calcium and phosphorus. Moreover proteins like amelogenin, ameloblastin and enamelin which are essential for enamel matrix formation all belong to the secretory calcium-binding phosphoprotein gene family and are controlled by vitamin D[10] and also certain proteinases processing amelogenins during enamel mineralization at the secretory and early maturation stages like Enamelysin (MMP-20), is also calcium-dependent matrix metalloproteinase. Hence hypocalcemia in any form can predispose a child to develop MIH.

5) **Duration of breast feeding:** Associations have been made between the presence of polychlorinated dibenzo-p-dioxins (PCDDs) in breast milk and enamel hypomineralization in both clinical and laboratory studies.[30-32]The PCDDs belong to a class of environmental pollutants known as polyhalogenated aromatic hydrocarbons.[33] Persistence and accumulation of PCDDs in tissue lipids and in the food chain may result in chronic low-level exposure in humans.[34] The most toxic and widely studied of this general class of compounds is 2,3,7,8 tetrachlorodibenzo-p-dioxin, which is often called simply "dioxin" and which represents the reference compound for this class of compounds. In infancy, children can be exposed to these compounds mainly via breast-feeding. An infant can get even 25% of the mother's dioxin load via lactation and the accumulation of dioxins and dioxin-like compounds in fat may prolong the duration of their action.

RISK IDENTIFICATION, REMINERALIZATION, AND PREVENTIVE MANAGEMENT :

MIH children often experience PFM pain and sensitivity and aesthetic concerns when their incisors are affected. A 6-step management approach is proposed as shown in table 4.[35] Children at risk for MIH should be identified prior to PFM eruption, based upon a relevant history of putative etiological factors in the first 3 years and from careful study under magnification of the unerupted molar crowns on any available radiographs.

The cariogenicity and erosivity of the child's diet should be assessed and appropriate recommendations made for dietary modification. Thorough oral hygiene should be instituted; this could include a desensitizing toothpaste.

Steps	Recommended procedures
Risk identification	Assess medical history for putative etiological factors
Early diagnosis	Examine at-risk molars on radiographs if available
	Monitor these teeth during eruption
Remineralization and desensitization	Apply localized topical fluoride
Prevention of dental caries and post-eruption breakdown (PEB)	Institute thorough oral hygiene home care program
	Reduce cariogenicity and erosivity of diet
	Place pit and fissure sealants
Restorations or extractions	Place intracoronal (resin composite) bonded with a self-etching primer adhesive or extracoronal restorations (stainless steel crowns)
	Consider orthodontic outcomes post-extraction
Maintenance	Monitor margins of restorations for PEB
	Consider full coronal coverage restorations in the long term

Table 4 – A Clinical management approach for the permanent first molars affected by MIH.

TREATMENT MODALITIES:

I. PREVENTIVE:

.The oral hygiene strategies that could be given to parents or patients in cases where tooth-brushing is difficult due to sensitive, poorly mineralized surfaces of affected molars are as follows:

1. Brush affected molars gently with a desensitizing toothpaste (preferably containing fluoride) on a soft toothbrush;
2. Apply a CPP-ACP topical crème daily using a cotton bud; and
3. Apply a low concentration fluoride treatment gel regularly using a cotton bud.

Remineralization therapy should commence as soon as the defective surface is accessible. Remineralization and desensitization may be accomplished with Topical Fluoride treatment and casein phosphopeptide- amorphous calcium phosphate (CPP-ACP) oral care products is recommended

II. RESTORATIVE:

a) Restoration of the permanent first molars : The porous exposed subsurface enamel or dentin may promote chronic inflammation of the pulp, complicating anaesthesia.[37,38]

The choice of materials will depend on the defect severity and the age and cooperation of the child. Adhesive materials are usually chosen due to the atypical cavity outlines following removal of hypomineralized enamel.

With physical properties superior to GIC and RMGIC, the Resin Components are esthetic materials with high wear resistance and adhesion when used with resin-based adhesives.

The RCs are materials of choice in MIH where defective enamel is well demarcated and confined to 1 or 2 surfaces with supragingival margins and without cuspal involvement. Resin composites are not successful in large defects because the etch pattern shows preferential dissolution of rod peripheries, loss of inter rod enamel resulting in enlarged inter rod space and inter crystal space is minimal probably reducing surface area available for bonding. The enamel adhesive interface of hypomineralized enamel is porous with cracks without a uniform hybrid layer. Failures with composite restorations have been thought to be due to these reasons. Hence it is recommended to remove all the hypomineralized enamel prior to placement of resin composite restorations and it is also suggested to pretreat the enamel with 5% sodium hypochlorite to remove the protein encasing the hydroxyapatite prior to etching.

a) **Full coronal coverage restorations :** When PFMs have moderate to severe PEB, preformed SSCs are the treatment of choice.

Properly placed, SSCs can preserve PFMs with MIH until cast restorations are feasible.

Compared to SSCs, cast restorations require minimal tooth reduction, provide high strength for cuspal overlay and maintain periodontal health due to their supragingival margins.

b) **Restoration of the hypomineralized Permanent incisors :** Hypomineralized incisors in MIH may present esthetic concerns to children and their parents. Microabrasion can be an effective treatment in shallow defects, but the defects usually extend through the full enamel thickness.

c) **Extraction of severely hypomineralized first permanent molars:** When PFMs are severely hypomineralized, restorations may be impossible and extraction must be considered. In such cases, early orthodontic assessment is recommended. Since PFMs are rarely an orthodontist's choice for extraction, later orthodontic treatment may be complicated. Factors affecting molar prognosis—such as vitality and restorability, dental age, buccal segment crowding, occlusal relationships, and the condition of other erupted and unerupted teeth—all need to be assessed when considering molar extraction. If restorative

treatment is a major problem, or if it fails, the optimal timing of extractions and follow-up of tooth eruption and development of occlusion can be managed.

CONCLUSION

The prevalence of MIH appears to be increasing, and managing affected children is now a common problem for pediatric dentists. Teeth diagnosed with MIH have significantly lower hardness values (HV) in hypomineralized compared with normal enamel. Although the etiology is unclear and may, in fact, be multifactorial, children born preterm and those with poor general health or systemic conditions in their first 3 years may develop MIH. The early identification of such children will allow monitoring of their PFMs so that remineralization and preventive measures can be instituted as soon as affected surfaces are accessible.

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