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Molar Incisor Hypomineralization: An Update

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Abstract : *Molar-Incisor Hypomineralization (MIH) is a congenital disease which is increasing in prevalence. It affects permanent first molars and, often to a lesser degree, permanent incisors with variable severity. The etiology is unknown, but various risk factors have been put forward. Differential diagnosis is mandatory not to confound MIH with other diseases like Amelogesis Imperfecta or Fluorosis. Treatment consists in a minimally invasive approach by reinforcing and protecting the existing dental structure. In more severe cases, restorative treatment or extraction may be indicated.*

Keywords : *Dental Enamel Hypoplasia, Hypoplastic Enamel, Molar Incisor Hypomineralizatio.*

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I. Introduction

Molar Incisor Hypomineralization (MIH) is a form of developmental defect of enamel that affect both function and esthetics of teeth. Weerheijm et al[1] defined "molar incisor hypomineralization" (MIH) as hypomineralization of systemic origin of 1 to 4 permanent first molars frequently associated with affected incisors.Multiple terminologies have been coined in the past to denote this condition including, Hypomineralized permanent molars, Idiopathic Enamel Hypomineralization, Dysmineralized permanent first molar, Non fluoride hypomineralization and Cheese molars. The prevalence of MIH is highly varied and using the criteria laid down by Weerheijm et al[2] it ranged from 4% to 25%.[3] Children affected from MIH are observed to receive dental treatment more frequently than those without MIH.[4] Affected teeth are more prone to dental caries as the enamel is porous and brittle, which also make it sensitive to thermal and mechanical stimulation, making oral hygiene difficult to perform. There is also rapid breakdown upon eruption as the teeth are exposed to masticatory forces. Similar defects have also been observed in primary second molars and are termed as Deciduous Molar Hypomineralization (DMH).[5] Elfrink et al[5] defined DMH as idiopathic hypomineralization of 1-4 second primary molars. DMH is also considered to be a predictive factor for MIH.[6] Prevalence of DMH is reported to be 4.9%- 9%.[5,6]

II. Etiology

Developmental defects of enamel (DDE) can be described either as Hypoplasia or Hypomineralization. Disturbance in ameloblast functioning in Secretory stage of enamel formation results in enamel hypoplasia. While, disturbances in Transitional &/or Maturation stage may lead to hypomineralization defect,[7] of which MIH (& DMH) is a form. It is now widely accepted that when resorptive potential of ameloblasts is disturbed and inhibition of proteolytic enzyme takes place, hypomineralization may result. These alterations result in protein retention, particularly amelogenin, leading to interference with crystal growth and enamel maturation. [8] Though MIH is considered to be idiopathic defect clear etiology is not yet agreed upon. Risk factors associated with MIH can be divided into prenatal, perinatal and postnatal factors.[9] (Table 1)

		Factors
Prenatal		
Lifestyle	Health	Maternal illness or infection Maternal hypocalcemia Nutrition
Perinatal		
	Health	Infant hypoxia Very low birth weight Premature birth Calcium shortage Misc. medical problems
Postnatal		
	Lifestyle	Breastfeeding Nutrition Calcium shortage
	Environment	Dioxins and polychlorinated bisphenols Environmental pollution
	Health	Childhood illnesses (in general) Chicken pox and other viral infections Otitis media Asthma, lung problems, allergy Fevers (irrespective of cause) Medications (in general) Antibiotics Antiasthmatic medication

Table 1: Risk factors associated with MIH

Crombie et al,[10] in a systematic review, observed that acute or chronic childhood illness and its treatments, conditions of birth and neonatal period and nutrition were only weakly associated with MIH; while dioxins had moderate level of association. Further, Allalusua et al,[11] concluded that none of risk factors studied till now can be considered causal factors for MIH. Conversely, Vieira et al[12] proposed that MIH is a genetic, not an idiopathic condition as it is commonly believed. If additional gene variations are also present then it may result in involvement of permanent canine and premolars additional to molars and incisors. As with MIH, etiology of DMH is believed to be multifactorial with prenatal and perinatal factors being more important than postnatal. Ghanim et al[13] observed in a study that 94% of children with DMH had at least one medical problem. Risk of DMH increased with increase in number of medical problems. Observed risk factors for DMH include low birth weight and fever (during child's first year), maternal alcohol consumption during pregnancy and ethnicity. Medication use during pregnancy was not associated with DMH. [14]

1. Characteristics of MIH affected enamel

MIH enamel defects appear in otherwise normal looking enamel as well-demarcated opacities. These defects involve cuspal areas, inclined planes and smooth surfaces with lesion extending from amelodentine junction to the surface of the tooth, while the cervical areas appear normal. It has also been observed that the darker the enamel the higher the severity of the defect i.e. yellow-brown enamel is more porous and has less mineral density than white-chalky enamel, suggesting that risk of fracture under occlusal load increases in darker enamel opacities. [15]

The mechanical properties also vary significantly, being 50-75% less in affected enamel resulting in low resistance to wear and flexure on loading resulting in enamel that is more prone to fracture. Surface of lesion being harder than the body as is the case in normal enamel.[16] Also, there appears to be a transition zone between hypomineralized defect and sound cervical enamel.[17] The hardness and modulus of elasticity of cervical enamel is similar to sound enamel making this region useful for bonding of restorative material to the affected tooth with resin composite as adhesion in hypomineralized region may be compromised. (Table 2)

Table 2: Hardness and modulus of elasticity of hypomineralized vs sound enamel

	Average hardness (GPa)	Average modulus of elasticity (GPa)
Sound enamel (control tooth)	3.66 ± 0.75	75.57 ± 9.98
Sound dentine	0.83 ± 0.13	20–25
Hypomineralized enamel	0.53 ± 0.31	14.49 ± 7.56

Scanning electron microscopy imaging has shown that enamel rods in the affected enamel are partly disorganized and comprised of increased porosities, while maintaining distinct boundary between normal cervical enamel and hypomineralized occlusal part. [7,17]

Transmission electron microscopy images have shown MIH affected enamel as porous and crystals to be loosely packed. The rod sheaths were more porous which could be the reason for easy deformation of enamel rods under occlusal forces.[18] The microshear bond strength of composite restorative material to hypomineralized enamel is upto 54% less than sound enamel leading to higher frequency of cohesive failure, meaning enamel was weaker than the of bond between composite restorative material and affected enamel.[19] As far as mineral density is concerned, it is 20% lower in MIH affected enamel.[20] Cervical enamel has normal mineral density however it drops rapidly as we move to center of the lesion which has the lowest mineral density. In addition, the less dense minerals are associated with 5-25% increase in porosity.[21] This high porosity along with wide dentinal tubules may result in easier chemical and thermal irritation of the pulp.

MIH enamel, despite having higher protein contents, has near normal levels of amelogenins. This feature is suggestive of MIH to be a hypocalcification defect while Amelogenesis imperfecta and Fluorosis to be a hypomaturation defect as they have higher amelogenin level. [22] Pathogenically, MIH is a disturbance of mineralization which could be a result of overabundance of albumin, which promotes KLK4 inactivity resulting in elevated protein and reduced mineral content, thus compromising the mineralization of enamel. Further, calcium and phosphate content is lower in MIH enamel but the ratio of Ca/P remains similar. Carbon content, on the other hand, is higher in amount which reflects an increase in both carbonate and protein content.[21] Though carbonated crystals dissolve more easily under acid attack thus making enamel more prone to caries, the increased protein content renders it more resistance to acid dissolution. Hence the association between MIH and dental caries is still unclear. Trace elements like magnesium are in higher amount suggesting of disruption to amelogenesis. While, chlorine, strontium, sodium, and potassium are in similar quantity as in sound enamel.[17]

III. Diagnosis

Weerheijm et al[23] developed the criteria for the diagnosis of demarcated opacities, post-eruption breakdown (PEB), atypical restorations, and extracted PFMs due to MIH. (Table 3) Teeth are examined in wet condition and age of eight years is considered the most suitable age for diagnostic examination for MIH because by this time all 4 molars and majority of incisor are erupted with signs of MIH still present.

Table 3: Definitions of the judgment criteria to be used in diagnosing MIH

Demarcated opacity	A demarcated defect involving an alteration in 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 colour.
Posteruptive enamel breakdown (PEB)	A defect that indicates deficiency of the surface after eruption of the tooth. Loss of initially formed surface enamel after tooth eruption. The loss is often associated with a pre-existing demarcated opacity.
Atypical restoration	The size and shape of restorations are not conforming to the temporary caries picture. In most cases in molars there will be restorations extended to the buccal or palatal smooth surface. At the border of the restorations frequently an opacity can be noticed. In incisors a buccal restoration can be noticed not related to trauma.
Extracted molar due to MIH	Absence of a first permanent molar should be related to the other teeth of the dentition. Suspected for extraction due to MIH are: opacities or atypical restorations in the other first permanent molars combined with absence of a first permanent molar. Also the absence of first permanent molars in a sound dentition in combination with demarcated opacities on the incisors is suspected for MIH. It is not likely that incisors will be extracted due to MIH.

As the number of affected molars increase the risk of incisor involvement also increases. [23] Though, the incisor involvement is not mandatory for teeth to be classified as MIH. Elfrink et al[24] modified the criteria for the DMH. (Table 4)

Table 4: Definitions of the judgment criteria to be used in diagnosing DMH

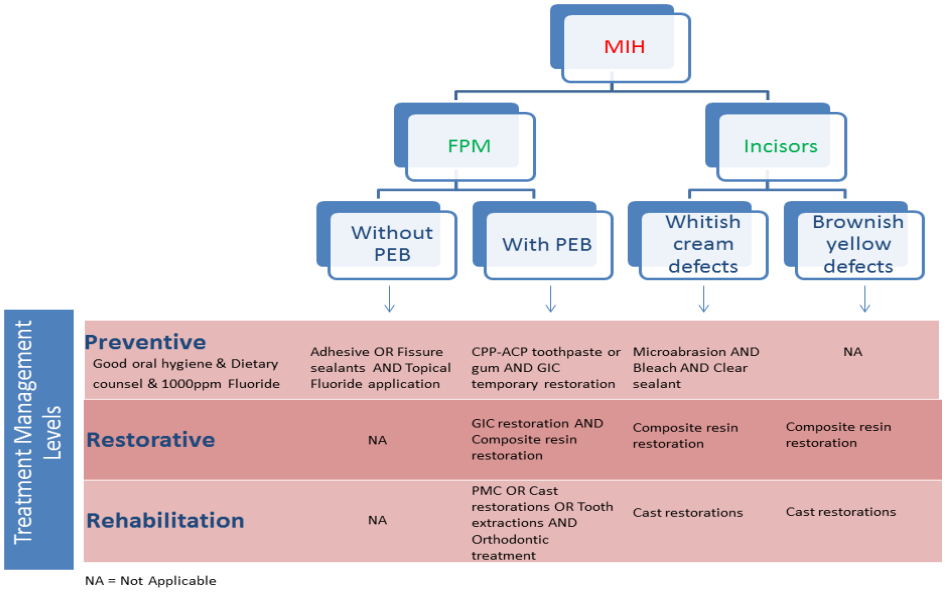
Atypical caries	The size and form of the caries lesion do not match the caries distribution in the child's mouth.
Atypical restoration	The size and form of the restoration do not match the present caries distribution.
Opacity	There is a defect involving an alteration in the translucency of the enamel, variable in degree. The defective enamel was of normal thickness with a smooth surface and can be white, yellow or brown in colour. The demarcated opacity was not caused by caries, fluorosis or amelogenesis imperfecta etc.
Post eruptive enamel loss	A defect indicating a deficiency of the surface after eruption of the tooth, e.g. hypomineralization related attrition. Enamel loss due to erosion was excluded.

Affected enamel can vary from white, cream, yellow to brownish in colour with sharp demarcation between the affected and sound enamel, whereas, if the margins are smooth and rounded it suggests enamel hypoplasia. Fluorosis, on the other hand, presents with typical diffuse opacity unlike that of MIH which has demarcated opacity. If there is generalized opacity present in dentition then Amelogenesis Imperfecta, rather than MIH which involves only molars and incisors, could be the reason. Brown, dull and porous opacities on cuspal tips are generally weaker than white and shiny tooth surfaces and are more prone to chipping leading to post eruptive breakdown. Even if the hypomineralized surface is intact toothbrushing can cause sensitivity.[1,23]

IV. Treatment Planning

MIH is associated with significant increase in treatment needs especially in severe cases, as porous enamel along with PEB encourage bacterial penetration in dentin leading to pulpal inflammation and pain. This is one of the reason children with MIH are more prone to suffer from dental anxiety and fear. Santos et al[25] summarized the treatment options for MIH. (Fig. 1)

Figure 1: Management of Molar Incisor Hypomineralization



Further, Elhennawy et al[26] in a systematic review noted that amalgam restorations show high failure rate in MIH molars as the lack of bonding and extensive cavity preparation compromise the prognosis of restoration. If, as a preventive measure, sealant is applied on MIH enamel it is best to use acetone containing bonding agent as it improves retention. For mild cases remineralization therapies using casein phosphopeptide amorphous calcium phosphate (CPP-ACP) might be an option, especially for anterior teeth.[27] Other treatment modalities include Microabrasion, however, it is associated with enamel loss or a combination of etching, bleaching and sealing MIH lesions which has shown promising results.[28] Resin infiltration treatment for MIH cases has been tested in vitro but is unreliable, as specific protocol for removal of proteins from enamel pores has not been yet established.[29]

When direct restoration is the treatment of choice then composite is the choice of material. It is observed that Self-etch materials has similar or better bonding compared to etch-and-rinse systems.[19] GIC can be used as temporary restoration in these cases because of low wear and fracture resistance. Preformed metal crown (PMC) can be a substitute to temporary direct restoration and can be used in children or adolescent with repeated failure of less invasive treatments. In severe cases of MIH extraction can also be a choice of treatment however, prior orthodontic evaluation of adjacent teeth and occlusion are necessary. When a lower first molar is extracted, second molar can tilt to mesial and distal because of thinner lingual plate resulting in scissor bit or non-working side interferences. It may also lead to poor interproximal contacts making the region more prone to food lodgment and difficulty in cleaning. If upper second molar is unerupted at the time of extraction of MIH affected first molar then it usually erupts in a good position. If the extraction is performed before 8 years of age it may result in misalignment of unerupted second premolars. Age of 9-10 years, when the bifurcation dentin of second molar is mineralizing, is considered ideal for extraction as it increases the odd of optimizing eruption on second molar into first molar position, especially in the lower arch.[30]

V. Conclusion

The prevalence of MIH appears to be increasing and is now a common problem in pediatric dental clinics. Current research is indicating towards genetic etiology. Management of MIH should aim towards early and prompt intervention so as to prevent further tooth destruction and provide durable restorations to the affected teeth.

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