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Molar Incisor Hypomineralisation – A Review

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ABSTRACT
Background: Molar incisor hypomineralization (MIH) is a developmental condition common in childhood. The condition mainly affects one or more permanent first molars (PFM) as well as permanent anteriors. It is noticed as poorly developed enamel.
Objective: To give an overall up to date review of the etiology, diagnosis and management of molar incisor hypomineralization.
Materials and methods: The clinical literature relating to MIH obtained from PubMed and the Cochrane library
Key words:molar incisor hypomineralization, etiology, prevalence, treatment plan

I. INTRODUCTION

The term molar incisor hypomineralization (MIH) was coined in 2001 to describe the clinical appearance of enamel hypomineralization of systemic origin affecting one or more permanent first molars that are associated frequently with affected incisors. (1)The condition is also known as "hypomineralized" Permanent first molars, "idiopathic enamel hypomineralization, "dysmineralized" Permanent first molars, "nonfluoride hypomineralization," and "cheese molars". The condition is due to impaired ameloblastic function during the transitional and maturational stages of amelogenesis.(2)
The clinical management of MIH can be difficult because of the following reasons:

- The sensitivity and rapid development of dental caries
- The limited cooperation of young patient
- Anaesthesia is difficult to be achieved
- Frequent marginal breakdown of the tooth structure

This review paper emphasis on the prevalence, putative etiological factors, diagnosis, and features of molar incisor hypomineralization and the sequential management.

ETIOLOGY AND PREVELANCE OF MIH

MIH has a strong correlation with prenatal, perinatal, and postnatal infection/disorder in the children.(3) Chicken pox, vitamin A deficiency, and ear infections were the most common conditions associated with MIH.(4) Also the use of antibiotics during the first 3 years of birth can cause MIH as it may alter the secretory ability of ameloblast.(4)

Enamel hypoplasia/hypomineralization or complete cellular degeneration is common in case of maternal pyrexia or newborn fever as these have a dangerous effect on amelogenesis.(5)

The world show a wide variation in the prevalence of MIH which can range between 2.8 to 40.2%.(6) In response to this finding Ghanim et al(7) have introduced a standardized scoring system based on the European Academy of Pediatric Dentistry (EAPD) evaluation criteria.
A manual was recently published(6) to facilitate and standardize the use of the scoring system in future epidemiological studies. It is estimated that this condition affects one in six children worldwide.(8)
According to Weerheijm et al, the prevalence of MIH ranges from 4% to 25%.(9)The affected number of teeth can vary from 1-4 number of hypomineralized PFMs in an individual can vary from 1 to 4, and the teeth are moderately or severely affected.(9) When more PFMs are affected the risk of involvement appears to increase.
The difference in prevalence may be due to variation in ethnicity, sample size, and age of population.(10).Indian pediatric population has a prevalence of 13.9%.(3).

In a study, involvement of both incisors and molars was significantly high in female.(3) It

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was observed that prevalence of affected single molar was high. Also the study shows mean age of the affected children was 10.25 years and observed that more severe lesion was present with increased age(3). This is due to the fact that a hypomineralized tooth after eruption is subjected to masticatory stress and caries attack.

MIH DIAGNOSIS

Weerheijm et al. introduced the criteria for the diagnosis of opacities over dentition, post eruptive break down, atypical restoration, and permanent molars with MIH.(9) Generalized white opacities as seen in amelogenesis imperfecta was not included under MIH, as MIH is limited to permanent molars and permanent incisors.(9) The demarcated opacities post eruptive break down (PEB) and atypical restorations are examined after thorough cleaning of teeth and wetting the permanent molars and permanent incisors.(11)

Demarcated opacities alter the enamel translucency and appear white-cream or yellow-brown in colour with a distinct boundary from normal enamel. The enamel is of normal thickness and has a smooth surface. The opacities rarely involve the cervical one third but seen in incisal or cuspal one third.(11)The intact enamel surface is typically hard, smooth, but the subsurface enamel is soft and porous.(11) Opacities can also be seen adjacent to restoration margins.

Diagnosis of MIH should be done as soon as it is clinically apparent. It is ideal to do the examination on wet teeth. In order to be recorded as MIH the lesions should be larger than 1 mm.(12) The dentist should ask the parents about any illness that occurred in prenatal, perinatal, postnatal or the first three years of life to support the diagnosis of MIH.

Mathu-Muju and Wright(13) had classified MIH into three severity levels:

1. Mild MIH: The demarcated opacities located at non-stress bearing areas, no caries associated, no hypersensitivity and incisor involvement is usually mild
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 concern.

DIFFERENTIAL DIAGNOSIS

Conditions present with hypomineralized enamel lesions include (6,14)

1. Fluorosis: Fluorosis presents as diffuse, linear, patchy or confluent white opacities without a clear boundary and associated with fluoride ingestion during enamel development. The extent of lesion can range from barely striations to gross disfiguration of affected teeth with almost complete loss of the external part of the enamel. Symmetrical bilateral pattern is seen unlike MIH which is asymmetrical. The affected teeth are caries-resistant while in MIH they are caries-prone.
2. Enamel hypoplasia: This is a quantitative defect of enamel with reduced thickness. Regular and smooth enamel lesions are shown by developmental and pre-eruptive lack of enamel. The margins in MIH with post-eruptive enamel breakdown are sharp and irregular.
3. Amelogenesis imperfecta: The enamel is hypoplastic, hypomature, or hypomineralized. This is a genetic condition, both primary and permanent dentitions are affected and usually have familial history.
4. White spot lesion can indicate the earliest clinical sign of caries. Chalkier, matt or more opaque lesions can be seen compared to sound enamel.

They are commonly seen at the cervical margin of tooth where the plaque accumulation is more.

5. Traumatic hypomineralisation: This is associated with a history of dental trauma to the primary tooth. Periapical infection of the predecessor tooth disturb mineralization of the underlying tooth germ. It has a wide variety of clinical presentations differing in shape, outline, localization and colour. Usually limited to single tooth and asymmetrical pattern.

ENAMEL CHARACTERISTICS IN MIH

Enamel is an ectodermal origin mineralized tissue formed by the ameloblasts that differentiate from inner enamel epithelium. Hypomineralization occurs due to protein retention which interference with crystal growth and enamel maturation. This occurs due to disturbed resorptive potential of ameloblasts and proteolytic enzyme inhibition.(15)

Reduced enamel matrix pH can cause disruption of crystal growth and proteinase

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function which leads to protein retention and hypomineralization.(16)

Formation of hypomineralized enamel can also be due to lack of calcium phosphate.(17) With increase in severity of hypomineralization carbon concentration increases and calcium and phosphorus concentration decreases. This results in lowering of calcium/phosphorous ratios in the enamel.(18) This impairment in calcium metabolism may have a role in the development of hypomineralized enamel.(18)

The differences in hardness, porosity, and mineral content can attribute to the discoloration of hypomineralized enamel.(11) White defects and normal enamel have higher Knoop hardness number and lower porosity than yellow brown defects.(11) Scanning electron microscopy, shows increased porosity and disorganized rod structure in the defective enamel region.(19)

Severity of PFM can differ, in some cases all four molar germs appear to have been affected. In some cases single or several molars shows randomly distributed opacities(2) The mineral content decreases from dentinoenamel junction to subsurface enamel. Zones of normal enamel in hypomineralized molars show reduced mineral concentration (of about 5%) and lower calcium/phosphorus ratios.(2)

TREATMENT PLAN

The treatment protocol should be the early detection and prevention rather than extensive restorative treatment. Currently there are no guidelines available for the management of MIH, but the European Academy of Pediatric Dentistry (EAPD) published a consensus paper in 2010 as 'Best clinical practice guidance for clinicians dealing with MIH.'(12)

More recently, the Würzburg MIH work group introduced a treatment need index for MIH (MIH TNI).(20) It is a unique index as it is not only based on the extent of the destruction of tooth structure but also the possibility of hypersensitivity. MIH TNI can be used for epidemiological studies as well as for assessment and treatment planning.(20)

There are six measurements for index reference. These measurements are as follows: maxillary right; maxillary front; maxillary left; mandibular right; mandibular front; mandibular left. (Table)

INDEX	DEFINITION
0	No MIH, clinically free of MIH
1	MIH without hypersensitivity, without defect
2	MIH without hypersensitivity, with defect
2a	<1/3 defect extension
2b	>1/3<2/3 defect extension
2c	>2/3 defect extension or/and defect close to the pulp or extraction or atypical restoration
3	MIH with hypersensitivity, without defect
4	MIH with hypersensitivity, with defect
4a	<1/3 defect extension
4b	>1/3<2/3 defect extension
4c	>2/3 defect extension or/and defect close to the pulp or extraction or atypical restoration

Dr M Hubbard(8) stated that MIH is a common dental problem (affects one in six children on average worldwide) he also stated that affected molars may have a ten-fold higher risk of developing caries if its severely hypomineralized.

PREVENTIVE MEASURES: Appropriate dietary and preventive advice should be provided to affected children and their parents. Fluoridated toothpaste with at least 1450 ppm F should be given to reduce caries risk and tooth sensitivity. According to some in-vivo and in-vitro studies improving the mineralization of MIH teeth after eruption is possible but, due to the depth and/or the thicknesses of these lesions a complete resolution seems to be difficult. (21)

The long-term use of products containing casein phosphopeptide amorphous calcium phosphate (CPP-ACP) to remineralise the lesion is recommended especially at early stages where the surface enamel of newly erupted teeth is not completely matured.(21) It helps to increase the bio-availability of calcium and phosphate within saliva.(12) To get an enhanced benefit the combined use of fluoride and CPP-ACP can be used.(22)

Novamin is another product that has been found to enhance remineralisation and reduce

sensitivity. This material mineralizes tiny holes in the dentine leading to decreased sensitivity. According to multiple studies novamin containing toothpastes have better remineralisation capability than CPP-ACP.(23)

Fissure seal can be used as a preventive measure in MIH molars. Resin-based fissure sealants can be used with adhesive application, if the enamel is intact as this will increase fissure sealant retention.(6)

According to some researchers enamel pre-treatment with deproteinising agents such as 5% sodium hypochlorite or papain-based papacarie gel for 60 seconds after etching increases bond strengths.(24) In case of partially erupted MIH molars, hypersensitivity or post-eruptive enamel breakdown, it is recommended to fissure seal them with glass ionomer cement, this will serve as a temporary management.(6) The occlusal surface coverage, with glass ionomer cement, flowable or filled composite should be desired up to cusp level. Generally, the applied fissure sealants should be regularly monitored to avoid getting dislodged or lost.(25) Also, regular professional applications of fluoride varnishes/gels should be done as a part of the enhanced prevention protocol and to reduce tooth sensitivity.(6)

It is important for the dentist to achieve adequate local anaesthesia for restorative treatment of MIH teeth, in order to perform good quality restorative treatment, this also will avoid behaviour management problems. Hypersensitive MIH tooth is difficult to anaesthetize even with increasing the local anaesthetic dose. Use of inhalation sedation to increase the pain threshold is suggested by some researchers(13). Anaesthetic adjuncts such as intraligamental, intraosseous and palatal anaesthesia is also an effective option. (26)Apart from this, buccal infiltration with articaine as a LA adjunct to inferior alveolar nerve block was found to be effective in achieving more profound anaesthesia.

Rubber dam isolation while doing the procedure can prevent sensitivity from other teeth during which are not anaesthetized, also use of saliva ejector instead of high-volume suction could be a more gentle option for hypersensitive tooth.

RESTORATIVE MEASURES: Use of desensitizing toothpaste before the restorative appointment is a preoperative management technique. Application of fluoride varnish at a pre-restorative appointment(27) and using sedative interim restorations such as glass ionomer cements can be used in case the pain is uncontrollable.

Clinical consideration is whether to restore or extract and this depends on factors such as: child's age; severity of MIH; pulp involvement; presence of third molar germ(s); restorability of the tooth/teeth; expected long-term prognosis; and long-term treatment cost.(25)

Resin infiltration/ erosion-infiltration, uses a very low viscosity resin which is capable of penetrating demineralized enamel. Icon by DMG (Hamburg, Germany) is the only material available for this procedure. It would improve enamel micromechanical properties and decrease post-eruptive enamel breakdown and may improve the bonding and restorative outcomes.(28) Due to its greater porosity and reduced mineral density, teeth with severe MIH can be infiltrated more than mild cases.(29).

When restoring hypomineralized teeth, cavity design plays a critical role, as defective enamel remnants compromise the end result. Cavity design should involve removal of all the porous but not necessarily discoloured enamel. Resin composite with pre-treatment with 5.25% sodium hypochlorite is used as a definitive restorative material and this pre-treatment can improve the bond strength.(30)

In case of severely damaged MIH molars with high long-term survival rates full or partial coverage Preformed metal crowns (PMCs) can be used successfully.(6) It can prevent further post-eruptive enamel breakdown and manage sensitivity.

AESTHETIC MANAGEMENT: If the discoloration is limited to the outer surface of enamel micro abrasion can be done and it is more effective at eliminating brown mottling. (27) This technique followed by home application of CPP-ACP products was found to improve remineralisation outcomes.(31)

In order to camouflage white opacities tooth bleaching can be done which increase the overall brightness of the teeth. This treatment is advised for adolescents.(6) The possible side-effects of bleaching are: sensitivity, mucosal irritation, and enamel surface alterations.(25) To avoid the side effects such as sensitivity or mucosal irritation, combined use of CPP-ACP Tooth Mousse and bleaching gel is recommended.(32).

Etch-bleach-seal technique suggested by Wright(33) can be used to remove yellow-brown stains 37% phosphoric acid is applied for 60 seconds to etch, followed by continuous application of 5% sodium hypochlorite for five to ten minutes. Then after re-etching, the tooth is covered with a

protective layer such as clear fissure sealant or composite bonding agent. This technique leave a white mottled appearance to the yellow brown stain.(33)

Composite restorations involve removal of defective enamel and composite resin build-up using opaque resins and is indicated for large enamel defects that require treatment due to exposed dentine or chipped enamel(6). Pre-treatment with 5.25% sodium hypochlorite for one minute after etching can improve the bond strength to hypomineralized enamel, long-term maintenance is required as the restoration is prone for discoloration.(30) Porcelain veneers can be given for patients aged 18 years and above when the gingival margin has matured(25).

EXTRACTION: This is an option for severely affected PFMs with poor prognosis, at the dental age of eight to ten years. Second permanent molars may have an opportunity to drift into the PFM position. (34)

II. CONCLUSION

There is an increase in prevalence of MIH. The etiology may be multifactorial and commonly associated with preterm babies and children with poor general or systemic health in the initial three years of life. The early identification can help in initiating remineralisation protocols and preventive measures. Resin composite restoration can be given in case of areas with limited lesions, cavity design should involve removal of all the porous but not necessarily discoloured enamel. Self-etching primer adhesive shows more favourable bond strength.(35) Extra coronal restorations can be given in case of extensive lesions or extraction can be considered as a last resort treatment(34).

REFERENCES

[1]. Weerheijm KL, Jälevik B, Alaluusua S. Molar-incisor hypomineralisation. Caries Res. 2001 Sep-Oct; 35(5):390-1. Doi: 10.1159/000047479. PMID: 11641576.

[2]. Fearne J, Anderson P, Davis GR. 3D X-ray microscopic study of the extent of variations in enamel density in first permanent molars with idiopathic enamel hypomineralisation. Br Dent J. 2004 May 22; 196(10):634-8; discussion 625. Doi: 10.1038/sj.bdj.4811282. PMID: 15153976.

[3]. Weerheijm KL, Duggal M, Mejàre I, Papagiannoulis L, Koch G, Martens LC, Hallonsten AL. Judgement criteria for molar incisor hypomineralisation (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. Eur J Paediatr Dent. 2003 Sep; 4(3):110-3. PMID: 14529329.

[4]. Jälevik B, Norén JG. Enamel hypomineralization of permanent first molars: a morphological study and survey of possible aetiological factors. Int J Paediatr Dent. 2000 Dec; 10(4):278-89. Doi: 10.1046/j.1365-263x.2000.00210.x. PMID: 11310241.

[5]. Lygidakis NA, Wong F, Jälevik B, Vierrou AM, Alaluusua S, Espelid I. Best Clinical Practice Guidance for clinicians dealing with children presenting with Molar-Incisor-Hypomineralisation (MIH): An EAPD Policy Document. Eur Arch Paediatr Dent. 2010 Apr; 11(2):75-81. Doi: 10.1007/BF03262716. PMID: 20403301.

[6]. Mathu-Muju K, Wright JT. Diagnosis and treatment of molar incisor hypomineralization. Compend Contin Educ Dent. 2006 Nov; 27(11):604-10; quiz 611. PMID: 17133930.

[7]. Ghanim A, Silva M J, Elfrink M E C et al. Molar incisor hypomineralisation (MIH) training manual for clinical field surveys and practice. Eur Arch Paediatr Dent 2017; 18: 225–242

[8]. Denis M, Atlan A, Vennat E, and Tirlet G, Attal J P. White defects on enamel: diagnosis and anatomopathology: two essential factors for proper treatment (part 1). Int Orthod 2013; 11: 139–165

[9]. Wright JT, Hall K, Yamauchi M. The protein composition of normal and developmentally defective enamel. Ciba Found Symp 1997; 205:85-99, 106 (discussion).

[10]. Sui W, Boyd C, Wright JT. Altered ph regulation during enamel development in the cystic fibrosis mouse incisor. J Dent Res. 2003 May; 82(5):388-92. Doi: 10.1177/154405910308200512. PMID: 12709507.

[11]. Van Amerongen WE, Kreulen CM. Cheese molars: A pilot study of the etiology of hypocalcifications in first permanent molars. J Dent Child 1995; 62:266-269.

[12]. Jalevik B, Odelius H, Dietz W, Noren JG. Secondary ion mass spectrometry and X-ray microanalysis of hypomineralized enamel in human permanent first molars. Arch Oral Biol 2001;46:239-247

[13]. Mahoney EK. Mechanical properties and microstructure of hypomineralized enamel of permanent teeth. Biomaterials 2004; 25:5091-5100.
[14]. Ghanim A, Elfrink M, Weerheijm K, Marino R, Manton D. A practical method for use in epidemiological studies on enamel hypomineralisation. Eur Arch Paediatr Dent 2015; 16: 235–246
[15]. Hubbard M J. Molar hypomineralization: What is the US experience? J Am Dent Assoc 2018; 149: 329–330
[16]. Weerhejim KL, Mejàre I. Molar-incisor hypomineralisation: a questionnaire inventory of its occurrence in member countries of European Academy of Paediatric Dentistry (EAPD). Int J Paediatr Dent 2003 Nov; 13(6):411-416.
[17]. Mishra A, Pandey RK. Molar Incisor Hypomineralization: An Epidemiological Study with Prevalence and Etiological Factors in Indian Pediatric Population. Int J Clin Pediatr Dent 2016; 9(2):167-171.
[18]. Beentjes VEVM, Weerheijm KL, Groen HJ. Factors involved in the aetiology of molar-incisor hypomineralisation. Eur J Paediatr Dent 2002 Mar; 3(1):9-13.
[19]. Fragelli CM, Souza JF, Jeremias F, Cordeiro Rde C, Santos-Pinto L. Molar incisor hypomineralization (MIH): conservative treatment management to restore affected teeth. Braz Oral Res. 2015; 29:S1806-83242015000100271. Doi: 10.1590/1807-3107BOR-2015.vol29.0076. Epub 2015 Jun 16. PMID: 26083091.
[20]. Teffen R, Krämer N, Bekes K. The Würzburg MIH concept: Part 1. The MIH Treatment Need Index (MIH TNI). Eur Arch Paediatr Dent 2017; 18: 355–361
[21]. Crombie F A, Cochrane N J, Manton D J, Palamara J E, Reynolds E C. Mineralization of developmentally hypomineralized human enamel in vitro. Caries Res 2013; 47: 259–263.
[22]. Shen P, Manton D J, Cochrane N J et al. Effect of added calcium phosphate on enamel remineralization by fluoride in a randomized controlled in situ trial. J Dent 2011; 39: 518–525.
[23]. Mehta A B, Kumari V, Jose R, Izadikhah V. Remineralization potential of bioactive glass and casein phosphopeptide-amorphous calcium phosphate on initial carious lesion: An in-vitro ph-cycling study. J Conserv Dent 2014; 17: 3–7
[24]. Ekambaram M, Anthonappa R P, Govindool S R, Yiu C K Y. Comparison of deproteinization agents on bonding to developmentally hypomineralized enamel. J Dent 2017; 67: 94–101.
[25]. Lygidakis NA. Treatment modalities in children with teeth affected by molar-incisor enamel hypomineralisation (MIH): A systematic review. Eur Arch Paediatr Dent. 2010 Apr; 11(2):65-74. Doi: 10.1007/BF03262715. PMID: 20403300.
[26]. Da Silva-Junior GP, de Almeida Souza L M, Groppo F C. Comparison of Articaine and Lidocaine for Buccal Infiltration After Inferior Alveolar Nerve Block For Intraoperative Pain Control During Impacted Mandibular Third Molar Surgery. Anesth Prog 2017; 64: 80–84
[27]. Fayle S A. Molar incisor hypomineralisation: restorative management. Eur J Paediatr Dent 2003; 4: 121–126
[28]. Crombie F, Manton D, Palamara J, Reynolds E. Resin infiltration of developmentally hypomineralized enamel. Int J Paediatr Dent 2014; 24: 51–55.
[29]. Kumar H, Palamara J E A, Burrow M F, Manton D J. An investigation into the effect of a resin infiltrant on the micromechanical properties of hypomineralized enamel. Int J Paediatr Dent 2017; 27: 399–411.
[30]. Chay PL, Manton DJ, Palamara JE. The effect of resin infiltration and oxidative pre-treatment on microshear bond strength of resin composite to hypomineralized enamel. Int J Paediatr Dent. 2014 Jul; 24(4):252-67. Doi: 10.1111/ipd.12069. Epub 2013 Oct 17. PMID: 24134408.
[31]. Cobourne M, Williams A, Harrison M. A guideline for the extraction of first permanent molars in children. 2014.
[32]. Pliska B T, Warner G A, Tantbirojn D, Larson B E. Treatment of white spot lesions with ACP paste and microabrasion. Angle Orthod 2012; 82: 765–769
[33]. Mastroberardino S, Campus G, Strohmenger L, Villa A, Cagetti M G. An Innovative Approach to Treat Incisors Hypomineralization (MIH): A Combined Use of Casein Phosphopeptide-Amorphous Calcium Phosphate and Hydrogen peroxidea Case Report. Case Rep Dent 2012; 2012: 379, 593

[34]. Wright JT. The etchbleachseal technique for managing stained enamel defects in young permanent incisors. Paediatr Dent 2002; 24: 249–252.
[35]. William V, Messer LB, Burrow MF. Molar incisor hypomineralization: review and recommendations for clinical management. Pediatr Dent. 2006 May-Jun;28(3):224-32. PMID: 16805354.