Molar Incisor Hypomineralization: A Review

Vikram Jhamb¹, Yoginder Yadav²

ABSTRACT
Molar incisor hypomineralization (MIH) is a common condition resulting in defect in permanent first molars and incisors. Etiological associations with systemic conditions or environmental insults during the child’s first 3 years have been implicated. The high prevalence of molar incisor hypomineralization indicates the need for research to clarify etiological factors and improve the durability of restorations in affected teeth. The purpose of this paper is to review the diagnosis, prevalence, etiological factors, features and clinical management of molar incisor hypomineralization.

Keywords: Enamel Hypomineralization

INTRODUCTION
“Molar Incisor Hypomineralization” is a condition in which permanent incisors and first molars show demarcated enamel opacities ranging from white to brown.¹ This condition usually seen in permanent molars and incisors. In this condition enamel becomes soft, porous and sensitive. Sometimes it undergoes posteruptive breakdown resulting in cavities. Patient’s affected by molar incisor hypomineralization shows enamel loss and become susceptible to caries.²

DEFINITION
Molar Incisor Hypomineralization is defined as the clinical appearance of morphological enamel defects involving the occlusal and/or incisal third of one or more permanent molars or incisors as result as “hypomineralisation of systemic origin.”(Weerheijm et al. 2003)

PREVALENCE
In many countries, researchers have established the prevalence of MIH in healthy children. The reported prevalence varies between 2.4% and 40.2%. Studies showed there is equal gender distribution. The risk of involvement of the permanent maxillary incisors appears to increase when more PFMs are affected.³,¹⁷

ENAMEL HYPOMINERALIZATION
Enamel is the hardest tissue in the human body, but its formation can be disturbed rather easily. Disturbances in enamel formation leave a permanent mark in the tooth. These disturbances can be inherited (e.g., amelogenesis imperfecta), acquired (e.g., induced by chemicals such as in fluorosis) or idiopathic (e.g., Molar Incisor Hypomineralization). MIH are probably caused by a disturbance in the initial calcification and/or during the maturation phase of the enamel, causing demarcated opacities.⁴ In MIH these opacities contain more carbon and less calcium and phosphate.⁵ The mineral content of the enamel is reflected in the mechanical properties of the enamel.⁶ In MIH molars, the enamel density in the hypomineralised areas is lower than in sound areas.⁷

DIAGNOSTIC CRITERIA
Modified DDE(developmental defects of enamel) index (FDI) 1992
• Mild- less than 30% of tooth enamel surface area visibly disrupted.
• Moderate- 31-49% of enamel surface area visibly disrupted.
• Severe – more than 50% of enamel surface area visibly disrupted.

Criteria for scoring molar incisor hypomineralisation (MIH) according to European Academy of Paediatric Dentistry (2003)

<table>
<thead>
<tr>
<th>Code</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Enamel defect free</td>
</tr>
<tr>
<td>1</td>
<td>White / creamy demarcated opacities, no PEB</td>
</tr>
<tr>
<td>1a</td>
<td>White / creamy demarcated opacities, with PEB</td>
</tr>
<tr>
<td>2</td>
<td>Yellow/ brown demarcated opacities, no PEB</td>
</tr>
<tr>
<td>2a</td>
<td>Yellow/ brown demarcated opacities, with PEB</td>
</tr>
<tr>
<td>3</td>
<td>Atypical restoration</td>
</tr>
<tr>
<td>4</td>
<td>Missing because of MIH</td>
</tr>
<tr>
<td>5</td>
<td>Partially erupted (i.e., less than one-third of crown height) with evidence of MIH</td>
</tr>
<tr>
<td>6</td>
<td>Unerupted/partially erupted with no evidence of MIH</td>
</tr>
<tr>
<td>7</td>
<td>Diffuse opacities (not MIH)</td>
</tr>
<tr>
<td>8</td>
<td>Hypoplasia (not MIH)</td>
</tr>
<tr>
<td>9</td>
<td>Combined lesion (diffuse opacities/hypoplasia with MIH)</td>
</tr>
<tr>
<td>10</td>
<td>Demarcated opacities in incisors only</td>
</tr>
</tbody>
</table>

In 2009 these criteria were updated to simplify the use of MIH score.

MILD
Opacity: A defect that changes 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. The demarcated opacity is not caused by

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How to cite this article: Vikram Jhamb, Yoginder Yadav. molar incisor hypomineralization: a review. International Journal of Contemporary Medical Research 2016;3(2):479-482.
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MODERATE/SEVERE

Post-eruptive enamel loss: A defect that indicates surface enamel loss after the eruption of the tooth e.g. hypomineralization related attrition. Enamel loss due to erosion was excluded.

Atypical caries: The size and form of the caries lesion do not match the present caries distribution in the child’s mouth.

Atypical restoration: The size and form of the restoration do not match the present caries distribution in the child’s mouth.

Atypical extraction: The absence of a molar that does not fit with the dental development and caries pattern of the child.

HYPOMINERALIZED ENAMEL CHARACTERISTICS

Molar incisor hypomineralization is a qualitative type of enamel defect that follows the natural incremental lines of enamel. In this type of enamel changes can be seen in hardness, porosity and mineral content of enamel. (Farah et al., 2010) Some studies have shown this type of enamel defect increases the porosity and lowers the hardness.

CLINICAL FEATURE

Enamel is soft and porous. Demarcated opacities can be seen. Enamel colour change into white cream or yellowish brown.

- 1-4 First Permanent Molar’s may be affected.
- In severe cases, defective enamel is lost soon after eruption to expose underlying dentine.
- Increased risk of hypomineralised incisors where molars are more severely affected.
- Teeth affected from hypo-mineralization are hypersensitive.

In this defect enamel shows opacities ranging from white to yellow brown. (Baroni and Marchionni, 2011). Yellow brown opacities occurs more frequent than the white patches. (Chawla et al., 2008)

ETIOLOGY

The causes of hypomineralised teeth are unclear although several factors that occur in the first four years after birth may be responsible. A recent study in Greece of 151 MIH children reported that 78% had experienced medical problems:

1. Prenatally (19%) 2. Perinatally (44%) 3. Neonatally (22%). Only 15% of the children did not appear to have a putative etiological factor in their history.

MIH may have a multifactor aetiology acting additionally or even synergistically (Alalussu, 2010, Crombie et al., 2009, Fagrell et al., 2011), with a genetic predisposition associated with one or more of a range of systemic insults occurring at a susceptible stage in the development of specific teeth. These factors may be environmental like antibiotics, vaccines, socioeconomic factors, nutrition etc. medical like chickenpox, infectious disease, respiratory disease, vitamin D deficiency, genetic factors like DLx gene, Enamelysin protein. Systemic factors like severe malnutrition, bilirubinemia, thyroid and parathyroid disturbances, maternal diabetes.

TREATMENT MODALITIES

A 6-step management approach is proposed to treat the molar incisor hypomineralization. William et al. [2006]

<table>
<thead>
<tr>
<th>Steps</th>
<th>Recommended procedures</th>
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</thead>
<tbody>
<tr>
<td>Risk identification</td>
<td>Assess medical history for putative etiological factors</td>
</tr>
<tr>
<td>Early diagnosis</td>
<td>Examine at-risk molars on radiographs if available</td>
</tr>
<tr>
<td>Remineralization and desensitization</td>
<td>Apply localized topical fluoride</td>
</tr>
<tr>
<td>Prevention of dental caries and post-eruption breakdown (PEB)</td>
<td>Institute thorough oral hygiene home care program</td>
</tr>
<tr>
<td>Restorations or extractions</td>
<td>Reduce cariogenicity and erosivity of diet</td>
</tr>
<tr>
<td>Place pit and fissure sealants</td>
<td></td>
</tr>
<tr>
<td>Maintenance</td>
<td>Maintenance</td>
</tr>
<tr>
<td>Consider orthodontic outcomes post-extraction</td>
<td></td>
</tr>
<tr>
<td>Consider full coronal coverage restorations in the long term</td>
<td></td>
</tr>
</tbody>
</table>

Mathu-Muju and Wright [2006] who proposed a treatment approach according to the level of defect severity (mild, moderate, severe) and to the length of treatment time needed (short and long term).

PREVENTIVE APPROACH

In children’s with this type of defect parents should encouraged to use their children’s fluoride toothpaste. Willmott et al, 2008. Another product that might be also useful for MIH patients and requires further research [William et al., 2006a; Willmott et al., 2008] is Casein (Phosphopetide-Amorphous Calcium Phosphate, CPP-ACP). This product has been shown to create and stabilise a super saturated solution of calcium and phosphate followed by deposition at the enamel surface. CPP-ACP has been incorporated into sugar-free chewing gum and encourages remineralisation of the sub-surface carious lesions [Shen et al., 2001]. The use of 0.4% stannous fluoride gels on a daily basis have also been proposed to be helpful for reducing sensitivity in defective teeth [Fayle, 2003].

Fissure sealants (FS) may also be useful for FPM with mild defects, not sensitive and without breakdown, particularly when they are regularly monitored and replaced when lost [Fayle, 2003; Mathu-Muju and Wright 2006; William et al., 2006a].
RESTORATIVE APPROACH
Defective enamel should be removed and should be stored with GIC, Resin Modified Glass Ionomer Cements (RMGIC), Polyacid modified composite resins (PMCR), and Composite resins (CR), William et al., 2006, 2008. GIC has been additionally proposed as an intermediate layer restoring the dentinal contours, prior to composite placement, in cases that the cavity involves large areas of dentine [Mathu- Maju and Wright, 2006]. The only material that appears to be usable for one or more surfaces restorations in MIH molars is Composite Resin. There are clinical studies dealing with the outcome of such restorations in MIH molars. Lygidakis et al. [2003] evaluating the success rate of CR restorations placed on two or more surfaces including cusps of affected molars, reported good acceptable results after 4 years.

RESTORING HYPOMINERALISED PERMANENT MOLARS WITH FULL CORONAL COVERAGE
Preformed metal crowns (PMC) for use on FPM have been used for many years to cover molars with defective enamel and they are still recommended as a treatment option for MIH posterior teeth [Fayle, 2003; William et al., 2006a; AAPD, 2008]. They prevent further tooth loss, control sensitivity, establish correct interproximal and proper occlusal contacts, are not costly and require little time to prepare and insert. Kotsanos et al. [2005] reported that no replacement was needed for PMC placed on 24 molars with MIH, for a period of 3-5 years. Zagdwn et al. [2003] reported good success rate with only one failure of 19 PMC placed over a 2-year period, they also found that there were no significant differences between the longevity and success rates for PMC and cast adhesive copings (nickel chrome alloy). Acceptable results were reported for laboratory-fabricated crowns on defective molars, either gold in 29 teeth or tooth-coloured in 12 teeth [Koch and Garcia-Godoy, 2000].

RESTORING HYPOMINERALISED PERMANENT INCISORS
Microabrasion using abrasive paste and 18% hydrochloric acid might be effective only in shallow patchy whitish defects [Fayle, 2003; William et al., 2006a; Mathu-Maju and Wright, 2006; Joiner, 2006]. Restorations with CR and veneers are an alternative choice for anterior MIH defective teeth in children and adolescents with larger enamel defects that require treatment [Wray and Welbury, 2001]. Veneers using CR in long term may suffer from susceptibility to discoulouration, wear and marginal fractures, reducing thereby the aesthetic long-term result [Peumans et al., 1997]. In such cases and in older children and adolescents porcelain veneers are indicated [Wray and Welbury 2001; AAPD, 2008] The etch-bleach-seal technique [Wright, 2002] should be clinically evaluated further in large samples of MIH incisors, as it appears promising for interceptive early approach in aesthetic problems. Chair-side bleaching with 10% carbamide peroxide, for brownish-yellow defects should be investigated but only in older children. Note should be taken of the side effects of sensitivity, mucosal irritation and enamel surface alterations [Wray and Welbury, 2001; Dahl and Pallesen, 2003; Joiner, 2006].

EXTRACTION AND ORTHODONTICS
Study by Jälevik and Møller [2007] stated that the extraction of severely affected FPM in MIH patients was an adequate treatment alternative to restorative care. They examined the orthodontic status of 20 patients 3.8-8.3 years after extractions and concluded that 15 of them had an acceptable occlusion. Space reduction and favourable development could be expected if the extractions were undertaken prior to the eruption of the permanent second molar teeth.

CONCLUSION
Although the etiology is multifactorial early identification of children with this type of defect is important so that preventive and restorative measures can be applied as soon as possible to avoid the complications

REFERENCE
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Source of Support: Nil; Conflict of Interest: None
Submitted: 26-12-2015; Published online: 16-01-2016