Molar Incisor Hypomineralisation: A Review
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ABSTRACT
Molar Incisor Hypomineralisation is the term used to describe a specific pattern of enamel defects. It causes hypomineralisation of one to four First Permanent Molar, frequently associated with affected incisors. Investigators have put forward a number of possible causes; asthma, pneumonia upper respiratory tract infections, otitis media, antibiotics, dioxins in mothers milk, tonsillitis and tonsillectomy exanthematous fevers of childhood. At present times the etiology remains unclear. MIH requires a multidisciplinary approach hence a general physician/dentist should consult a pediatric dentist for management of such teeth.

KEY WORDS
First permanent molar, molar incisor hypomineralisation

INTRODUCTION
Molar Incisor Hypomineralisation (MIH) is the term used to describe a specific pattern of enamel defects.1 Children with the condition have higher levels of treatment needs and as a result are more anxious regarding future dental treatments.2-4 Weerheijm et al defined MIH as hypomineralisation of systemic origin of One to four First Permanent Molar (FPM), frequently associated with affected incisors.” The condition seems to have been recognised first in the 1970s and prevalence varies between 2.4% and 40.2%, dependent upon the study. This wide range of prevalence is likely to be secondary to environmental and genetic factors of the population under study.1

Weerheijm et al [2003] explained that any examination for MIH should be undertaken on clean wet teeth and that the age of 8 years was optimum, as at this age all permanent first molars and most of the incisors will have erupted. He also suggested that the second primary molars, second permanent molars and the tips of the permanent canines can also show enamel defects occasionally.

Synonyms5
Cheese Molars [weerheijm et al.2000]
Idiopathic Hypomineralisation [Jalevik et al.2000]
Conditioned Hypomineralisation [Beentjes et al.2000]
Non Fluoride Hypomineralisation [Leppaniemi et al.2000]
Molar incisor Hypomineralisation [Weerheijm et al.2001]

ULTRASTRUCTURE AND BIOCHEMICAL MAKE UP
Clinically MIH appears as disturbance to the translucency of enamel. According to Jalevik and Noren [2000] in MIH individuals, the ameloblasts seem to have been affected in early maturation. Some cells being irreversibly damaged, leading to greater porosity and to the development of a yellow or brownish-yellow demarcated opacity, these being mostly situated throughout the entire enamel bulk. Other ameloblasts may have the ability to recover from the insult
with an effect upon the maturation of the enamel being less marked such that the defects were creamy-yellow or whitish-creamy, with most being situated in the inner part of the enamel.6

Fearn et al. [2004] used a computer software package to analyse their X-ray Microtomography scans; line probes showed that the enamel of the full thickness hypomineralised area had a reduced mineral concentration of up to 20% compared with that of the apparently normal enamel of the same tooth.7 In contrast, Mahoney et al. [2004] using backscattered electron microscopy found a figure of only 5% mineral reduction for the affected enamel compared with unaffected apparently normal enamel.8

Jalevik et al. [2001b] also used X-ray microanalysis and found that the affected enamel had reduced calcium to phosphorus ratio (1:4) compared with that found in adjacent apparently normal enamel (1:8).9

CLASSIFICATION

Mathu-Muju and Wright [2006] gave following clinical criteria in order to divide the defects in the 3 different severity levels:

Mild MIH: Demarcated opacities are in non-stress-bearing areas of FPM, there are isolated opacities, no enamel loss from fracturing is present in opaque areas, there is no history of dental hypersensitivity, there are no caries associated with the affected enamel, and incisor involvement is usually mild if present.

Moderate MIH: Intact atypical restorations can be present, demarcated opacities are present on occlusal/incisal third of teeth without post eruptive enamel breakdown, post eruptive enamel breakdown/caries are limited to 1 or 2 surfaces without cuspal involvement, dental sensitivity is generally reported as normal, aesthetic concerns are frequently expressed by the patient or parent.

Severe MIH: Post eruptive enamel breakdown is present and frequently occurs as the tooth is emerging, there is a history of dental sensitivity, often widespread caries is associated with the affected enamel, crown destruction can readily advance to involve the dental pulp, defective atypical restoration is present, aesthetic concerns are expressed by the patient or parent.10,11

ETIOLOGY

Investigators have put forward a number of possible causes; asthma, pneumonia upper respiratory tract infections, otitis media, antibiotics, dioxins in mother’s milk, tonsillitis and tonsillectomy exanthematous fevers of childhood. Literature showed that teeth are affected indicates a systemic cause at around the time of birth.

The factors were grouped by age periods (prenatal, perinatal or postnatal) and by the putative factors likely to be present during those periods for better understanding.

Prenatal period: There was some evidence that medical problems during pregnancy were associated with MIH. In one study a specific illness, urinary infection, during the last trimester was associated with MIH-like lesions.12 In two other studies specific diseases were not associated with MIH but the authors reported that medical problems were more common in mothers of MIH children than in those mothers whose children did not have MIH.13,14

Perinatal period: In a Greek study, where the most common perinatal problems/conditions were Caesarian section, prolonged delivery, premature birth and twinning. In a prospective study it was found that MIH-like lesions and enamel hypoplasia were significantly more common in premature infants than in controls.15

Hypoxia: This can be associated with medical problems related to birth, such as prematurity, respiratory stress and excessively prolonged duration of birth. In an experimental study Hypoxia in rats was induced by maintaining the animals in a hypobaric chamber at 0.5 atm for 24 hrs This study suggested that short periods of hypoxia does not cause enamel defects.16

Postnatal period: Childhood illness/fever. In an experimental study it was possible to show that an exogenous pyrogen, turpentine, induced enamel hypomineralisation in rat incisors.17 The use of amoxicillin during the first year of life has been found to increase the risk of MIH.18 However, again it is not possible to be sure if childhood illness/fever or the treatment with an antibiotic is the causative factor or if both are involved.

Environmental toxicants: Accidental exposure to high levels of dioxins or polychlorinated biphenyls (PCBs) in early childhood has been found to be associated with demarcated opacity and/or hypoplasia.19,20 Those studies showed a dose-response relationship between the pollutant exposure (serum concentration) and developmental enamel defects in permanent teeth.

Breast feeding: A long duration of breastfeeding has been associated with MIH in a Finnish study. Those authors suggested that pollutants in human milk may be involved e.g. dioxins.21 Role of pollutants remained speculative.

Fluoride: It was thought to affect enamel crystal formation mainly during the maturation stage inducing defects described as diffuse opacities. In three studies the association between fluoride supplementation and MIH was studied. No significant association was found.13,22,23

Hypocalcaemia: This may occur in the perinatal period but also in prenatal and postnatal periods The finding that calcium, but not so clearly phosphate levels, was very low in MIH lesions suggests that they were caused by impaired calcium metabolism of the ameloblasts.9
CLINICAL APPEARANCE SIGNS AND SYMPTOMS

Clinically, MIH may present as discrete, opaque lesions, ranging from white to yellow-brown, distinct from the more diffuse linear opacities usually associated with fluorosis, and may be associated with post-eruptive enamel loss making it potentially difficult to distinguish from enamel hypoplasia. The distribution of the condition is often asymmetric, commonly with marked variation in severity within an individual. Lesions are fairly large demarcated opacities; these may or may not be associated with post-eruptive enamel breakdown and borders of enamel lost post-eruptively are irregular.

Rodd et al. investigated the pulps of non-carious hypomineralised FPM, comparing them to apparently sound FPM from MIH affected individuals. They acknowledged that their sample was small but concluded that the changes present were indicative of inflammatory changes.

MIH affected FPM can succumb to dental caries very rapidly and the presence of caries can act to mask the true diagnosis of MIH.

DIAGNOSIS

The European academy of pediatric dentistry seminar in 2003 agreed and published the following conditions

- Permanent first molars and incisors (12 index teeth) should be examined,
- Examination for MIH should be performed on wet teeth after cleaning
- Eight years of age was the best time for examination,
- Each tooth should be recorded for:
  > absence or presence of demarcated opacities,
  > posteruptive enamel breakdown,
  > atypical restoration,
  > extraction due to MIH,
  > failure of eruption of a molar or incisor.

Recently a Molar Hypomineralisation Severity index (MHSI) has been given by Kelly Oliver et al. [2013] and suggested that the MHSI characteristics were predictive of the treatment of the affected FPM’s and can guide management.

TREATMENT

A very useful 6-step management approach for MIH has been proposed recently by William et al.28

- Risk identification,
- Early diagnosis,
- Remineralisation (a better term may be mineralisation; the tooth was never ‘completely’ mineralised during development although there may also be an element of demineralisation from enamel caries, superimposed upon the hypomineralised areas) and desensitisation,
- Prevention of dental caries and post eruptive enamel breakdown,
- Restorations or extractions,
- Maintenance.

The first decision in relation to MIH- FPM that needs to be made is: Should the tooth be retained or lost? The decision to extract or restore will depend upon a number of different factors, some of these being the degree/extent of hypomineralisation, post eruptive breakdown, sensitivity, age and co-operation of the patient, any developing malocclusion and developmentally absent teeth.

Preventive advice. This is very important and should include sensible and appropriate dietary advice. If a child is still using a low-fluoride children’s toothpaste then the parents should be encouraged to change to one with a higher fluoride level of at least 1,000 ppm F. Casein Phosphopeptide-Amorphous Calcium Phosphate (CPPACP) creates and stabilises a super saturated solution of calcium and phosphate followed by deposition at the enamel surface; incorporated into sugar-free chewing gum this has been shown to remineralise sub-surface carious lesions. This product could be a material that may be useful for MIH patients; however, at the present time there is no evidence to support this.

Fissure sealants may be useful for FPM that are mildly affected, the enamel is intact and the teeth are not sensitive. Mathu-Muju and Wright [2006] suggested that if the fissures appeared opaque or yellow-brown then a 60 second pre-treatment with 5% sodium hypochlorite may be beneficial.

Restorative procedures. Restoration of the MIH affected FPM can be complicated by difficulties in defining the cavity margins; there are two approaches:

- Removal of all defective enamel to sound surfaces. This means that a lot of tooth material is lost but is better if an adhesive material is to rely upon bonding to enamel.
- The very porous enamel is removed, until resistance to the bur is felt. This is more conservative, but it can mean that the defective enamel may continue to breakdown.

There are many restorative materials/options available to the dental surgeon treating these patients:

- Glass Ionomer Cements,
- Resin Modified Glass Ionomer Cements,
- Polyacid modified composite resins,
- Composite resins,
• Preformed Metal Crowns (PMC),
• Indirect adhesive or cast restorations.

Amalgam is non-adhesive and its use in these atypically shaped cavities is probably best avoided.

In addition to existing types of treatment, minimally invasive Cast Adhesive Copings seems to be a feasible and useful method for restoration of FPMs with demarcated opacities and post-eruptive surface loss in children with MIH.30

Orthodontics: Dental age of 8.5-9 years is ideal for their extraction, this should coincide with the calcification of the bifurcation of the mandibular permanent second molars. This gives the best chance for the permanent second molars to come forwards into a good alignment.

Jalevik and Moller stated that extraction of severely affected FPM in patients diagnosed with MIH was a good treatment alternative to restorative care. They went on to state that spontaneous space reduction and favourable development could be expected if the extraction(s) was (were) undertaken prior to the eruption of the permanent second molar teeth.31

MIH Incisors: According to Jalevik and Noren the yellow or brownish-yellow defects are full thickness whilst those that are creamy-yellow or whitish-creamy are located in the inner part of the enamel. Because of these reasons micro-abrasion using pumice/acid is ineffective, bleaching with carbamide peroxide has been reported to produce some improvement, more so with the yellow-brown defects.5

The choice between direct and indirect veneers depends upon the clinician’s personal choice and skill. In addition, there is also a laboratory cost implication with indirect veneers.

Wakiaga et al. undertook a systematic review of direct versus indirect composite veneers for intrinsic dental stains and found six acceptable papers; they concluded that there was no reliable evidence to show that either approach was superior to the other with regard to longevity.32

CLINICAL IMPLICATIONS

• The affected teeth can be very sensitive to a current of air, cold or warm therefore a dentist has to pay serious attention to this sensitivity.
• Dentist can also face difficulty in obtaining adequate anaesthesia in affected children.
• Molars are fragile and caries can develop very easily.
• It is also difficult to identify which part is healthy.

CONCLUSION

MIH is an important clinical problem that often concerns physicians, general dentists and pediatric dentists. The present paper aims to further help clinicians dealing with the condition.

RECOMMENDATIONS

• Prospective studies starting around birth and extending to the time of eruption of FPM are needed to clarify etiology related issues since the etiology is still unclear.
• Expected mother and her health during pregnancy would need to be included in the study as a subject.
• General physicians/pediatricians should be made aware of the role of systemic illness in causing MIH.

REFERENCES


