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MOLAR INCISOR HYPOPLASIA-LITERATURE REVIEW

Paediatric Dentistry

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ABSTRACT

Molar incisor hypo mineralization (MIH) is defined as hypo mineralization of systemic origin of one to four first permanent molars, and incisors are also frequently affected. This disorder is a serious concern in pediatric dentistry. Teeth affected by MIH have many dental problems, such as hypersensitivity, poor aesthetics, and rapid progression of dental caries.The aetiology of MIH is unclear, but genetic and environmental factors have been proposed. The purpose of this article is to provide comprehensive overview of MIH, its diagnosis, illustration of various recent advances in treatment modalities, all of which may update pedodontist role in clinical management of this condition.

KEYWORDS

Hypo mineralization, molar incisor hypo mineralization, permanent incisor, permanent molar, post eruptive breakdown, prevalence

INTRODUCTION

Dental development and mineralization in humans start 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]

Disturbance in enamel formation from ameloblasts (mother cells) may occur in two phases-enamel matrix deposition (secretion phase) & enamel mineralization (maturation phase). If an unbalance occurs during the secretion phase, the enamel defect is called hypoplasia.[3]

In enamel hypoplasia, the defect is associated to smallest thickness of affected enamel, clinically presenting as shallow/deep fossae with horizontal/vertical grooves and with partial/total absence of enamel.[4]

On the other hand, if an unbalance occurs during maturation phase, the enamel defect is called hypo mineralisation. In hypo mineralisation, white or yellowish/brownish area can be seen and there is no thickness alteration.[5]

One alteration of great clinical importance is MIH (Molar Incisor Hypomineralization), which can be defined as an alteration of systemic aetiology, in which enamel disintegration of the occlusal tooth surface can be detected involving one to four first permanent molars. It can be associated with permanent teeth calcifying during the same formative period.[6]

The term molar-incisor hypomineralisation (MIH) was first introduced in 2001 by Weerheijm et al. and it was defined as 'hypomineralisation of systemic origin, presenting as demarcated, qualitative defects of enamel of one to four first permanent molars (FPMs) frequently associated with affected incisors.' [7] Earlier nomenclature included non-fluoride enamel opacities, internal enamel hypoplasia, non-endemic mottling of enamel, idiopathic enamel opacities and cheese molars.[8]

It is known that enamel formation begins about week 20 in utero for the crowns of the permanent first molars, 3–4 months for the central incisors and lower laterals and 10– 12 months for the upper lateral incisors. It is thought to take about 3 years for crown formation to complete. Therefore, research into the aetiology of MIH has concentrated on an environmental insult occurring in the first 3 years of life because of the pattern of molars and incisors affected.[6]

In 2003, MIH was further described as a developmental, qualitative enamel defect caused by reduced mineralisation and inorganic enamel components which leads to enamel discolouration and fractures of the affected teeth.[9]

In MIH, the FPMs show rapid caries progression starting shortly after eruption in the majority of cases, which causes serious problems to patients as well as treatment challenges to dentists.[9]

Prevalence of Molar Incisor Hypomineralisation

Today, abundant data on the prevalence of MIH is available. The prevalence of MIH has been shown to range from 2.8% to 44% in different studies [10].

Among studies with more than 1000 subjects, the prevalence of MIH ranges from 2.8% to 21%. Overall, the prevalence of MIH varies by country, region, and age group studied.[11]

Examination for MIH should be done at the age of 8 years, because at this age, all first molars and most incisors have erupted. Furthermore, the first molars are in relatively good condition at this point [12].

Of course, post-eruptive breakdown occurring before 8 years of age needs attention in clinical practice. Understanding the true prevalence of MIH requires a uniform calibration procedure, and the number of subjects included needs to be large enough to be representative of the studied population.[10]

Some studies reported that posteruptive breakdown occurs more frequently in boys. Children under the age of 10 are more commonly affected.[13]

Aetiological Considerations

The causative mechanism of MIH is still unclear.[15] The clinical presentation of localised and asymmetrical lesions suggests a systemic origin with the disruption in the amelogenesis process most probably occurring in the early maturation stage or even earlier at the late secretory phase.[9]

In general, the condition seems to be multifactorial and systemic factors such as acute or chronic illnesses or exposure to environmental pollutants during the last gestational trimester and first three years of life have been suggested as causative or contributing factors.[16]

The number of affected teeth was associated with the time when the potential systemic disturbance occurred; children with prenatal, perinatal and postnatal problems showing more affected teeth in increasing order.[17]

Multiple possible causes have been suggested in the literature, for instance, respiratory tract infections, perinatal complications, oxygen starvation, low birth weight, calcium and phosphate metabolic

disorders, frequent childhood diseases, use of antibiotics and prolonged breast feeding.[9]

In addition, some studies, raise the possibility of a genetic role in the aetiology of MIH, indicating that a genetic variation may interact with systemic factors leading to MIH.

Table 1: Various etiological factors of MIH [18]

Systemic	Environmental	Genetic	Medical
Severe malnutrition	Nutritional deficiency	D1X gene	Febrile illness
Maternal diabetes	Low socioeconomic status	RUNX2 gene	Respiratory and infectious diseases, chicken pox
Thyroid & parathyroid problems	Vaccines	Kallikrein 4	Preterm baby
Chronic-systemic diseases	Dioxins in breast milk	Mmp20 (enamelysin protein)	Prolonged-delivery cyanosis
Bilurubinemia	Antibiotics (amoxycillin)		Neonatal hypocalcemia, Vitamin D deficiency

Clinical Appearance, Signs and Symptoms: [19]

- Large demarcated opacities of altered enamel translucency, whitish-cream or yellowish-brown in colour, usually limited to incisal or cuspal one third of the crown, rarely involving the cervical one third.
- May or may not be associated with posteruption enamel breakdown
- Hypersensitivity
- Difficult to anesthetize
- Rapid caries progression

Diagnosis

The ideal time to diagnose MIH is as soon as it is clinically apparent either in primary or permanent dentition. [20]

The examination should be performed on clean wet teeth. The clinical presentation of MIH depends on its severity and can range from white-creamy opacities, yellow-brown opacities, post-eruptive enamel breakdown to atypical caries located on at least one FPM with or without incisor involvement.[8]

The lesions should be larger than 1 mm to be recorded as MIH.[20] When such clinical signs exist during examination, 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. [8]

A severity scale has been developed to classify MIH as mild, moderate, or severe at the tooth level, meaning that one tooth may be mild, and another tooth in the same patient may be severe, and seeing this amount of variability is a common occurrence. (Mathu-Muju & Wright, 2006).[21]

Table 2. Severity score of teeth affected with MIH [21]

	Mild	Moderate	Severe
Crown appearance	Demarcated opacities in non-stress-bearing area of molar	Intact atypical restoration present	Posteruptive enamel breakdown present
Enamel loss	Isolated opacities	Occlusal/incisal third of teeth without initial posteruptive enamel breakdown	Posteruptive enamel breakdown on erupting tooth that can be rapid
Caries	No caries associated with affected enamel	Posteruptive enamel breakdown/caries limited to one or two surfaces without cuspal involvement	Often develop widespread caries associated with affected enamel
Sensitivity	Normal dental sensitivity	Usually, normal dental sensitivity	Usually, history of dental sensitivity
Esthetics	Usually not an issue	Parents often express concern	Parents typically concerned

Table 3 According to EAPD placed in Athens in 2003, the scoring of MIH ranges from 0 to 10 as given below.[22]

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

MIH: Molar incisor hypomineralisation, PEB: Posteruptive enamel breakdown, EAPD: European academy of pediatric dentistry

Association between MIH and other hypomineralised teeth

The same demarcated defects that present on some molars and incisors in MIH have also been observed on other teeth such as second primary molars and tips of permanent canine cusps in some MIH cases.[7]

The studies had done to found out the association between hypomineralised second primary molars (HSPMs) and MIH. Approximately half of the FPMs with MIH were associated with HSPMs.[23]

Furthermore, Negre-Barber et al. in 2016, found that HSPM can be considered a predictor for MIH, indicating the need for monitoring, but the absence of HSPM does not rule out the appearance of MIH. [24]

A significant association between MIH and hypomineralised permanent canines has also been described in a paper by Schmalfuss et al. in 2017, where they found approximately one quarter of MIH-affected individuals had one or more permanent canines with signs of hypo mineralisation.[25]

Differential diagnosis

Conditions which can present with hypomineralised lesions and should be distinguished from MIH include.[26]

Fluorosis

This is associated with history of fluoride ingestion during enamel development. Clinically, fluorosis presents as diffuse, linear, patchy or confluent white opacities without a clear boundary. The severity can range from barely perceptible striations in the enamel to gross disfiguration with almost complete loss of the external part of the enamel. It affects teeth in a symmetrical, bilateral pattern unlike MIH which is asymmetrical. Moreover, teeth affected by fluorosis are caries-resistant while in MIH they are caries-prone. [26]

Enamel hypoplasia

This is a quantitative defect with reduced enamel thickness. The borders of hypoplastic enamel lesions are mostly regular and smooth, indicating developmental and pre-eruptive lack of enamel. The margins in MIH with post-eruptive enamel breakdown are sharp and irregular due to post-eruptive shearing of weakened enamel. [26]

Amelogenesis imperfecta

This is a genetic condition which results in enamel that is hypoplastic, hypomature, or hypo mineralised. In this condition, all teeth in both dentitions are affected and a familial history is often present.

White spot lesion

This is the earliest clinical sign of caries. The lesions appear chalkier, matt or opaquer than the adjacent sound enamel. They can be distinguished from MIH because they occur in areas of plaque stagnation, such as the cervical margin of the tooth.[26]

Traumatic hypo mineralisation
This is associated with a history of dental trauma to the primary predecessor tooth. Periapical infection of the primary tooth can disturb mineralisation of the underlying tooth germ. It has a wide variety of clinical presentations differing in shape, outline, localisation and colour. It is often limited to one tooth and asymmetrical.

Treatment modalities:
Identification of patients at risk of MIH and early diagnosis can lead to more effective and conservative management. [24]

Based on the available evidence, children at risk of MIH are those with poor general health during early childhood and/or those with HSPM(s).[20] [24]

More recently, the Würzburg MIH work group (an international working group with representatives from universities in Germany, Austria and Switzerland) introduced a treatment need index for MIH (MIH TNI).[24]

The MIH TNI is unique as it is not only based on the extent of the destruction of tooth structure but also the possibility of hypersensitivity.[24]

Table 4 MIH-TNI by Steffen et al. The MIH treatment needs index (MIH TNI), R. Steffen, N. Krämer, K. Bekes,2017[24]

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

Treatment approaches thus tend to be predicated on the severity of the MIH and the presence or absence of dental sensitivity.[25]

Six step management approach by William et al. 2006 suggests the following:[25]

Steps	Recommended procedures
Risk identification	Assess medical history for putative etiological factors
Early diagnosis	Examine at risk molars on radiograph if possible. Monitor these teeth during eruption.
Remineralisation & desensitisation	Apply localized topical fluorides
Prevention of dental caries & PEB	Institute through oral hygiene home care program. Reduce cariogenicity and erosivity of diet. Place pit and fissure sealants.
Restorations and extractions	Place intracoronal (resin composite) bonded with self-etching primer adhesive or extracoronal restorations (SSCs). Consider orthodontic outcomes postextraction.
Maintenance	Monitor margins of restorations for PEB. Consider full coronal coverage restorations in the long term

MILD MIH:
Prevention and maintaining the dentition [26]
- Topical fluoride application.
- Desensitizing toothpaste.
- Apply a CPP-ACP topical crème daily using a cotton bud.[31]
- Glass ionomer cement (GIC) sealants can provide caries protection and reduce surface permeability. [27]
- Where the enamel is intact and the patient does not report any sensitivity, sealants are the current treatment of choice. 60-second pre-treatment with 5% sodium hypochlorite to remove intrinsic enamel proteins may be beneficial.[27]

MODERATE MIH:
- Preventive measures previously outlined. Intervention may be required.
- Anterior teeth with isolated demarcated opacities that are of esthetic concern can be treated with NaOCl or other bleaching techniques, microabrasion or resin restorations. Yellow or yellow/brown spots in incisors or molars can lighten and become less noticeable with bleaching, but whitish opacities may become more prominent after applying the bleach. [34]
- For posterior teeth with enamel loss or decay limited to 1 or 2 surfaces that does not involve cuspal tooth structure, resin is the material of choice if the tooth can be adequately isolated. [35]
- Cavity margin placement- all defective enamel is removed. Only the very porous enamel is removed, until good resistance of the bur to enamel is felt.
- GIC restorations- Conventional GIC and RMGIC.It has propert of adhesion with both enamel and dentin and long-term fluoride release. Poorer mechanical properties therefore it is not recommended to be used in stress bearing areas and be used as an intermediate restoration. [28]
- Composite resin restorations- longer term stability compared with other restorative materials. Polyacid modified resin composites have good handling characteristics, release and take up fluoride and have tensile and flexural strength properties superior to GIC and RMGIC, but inferior to that of resin composite. Use of PMRCs in permanent teeth is restricted to non-stress bearing areas.[35]

SEVERE MIH:
- Once the molar has erupted, preformed SSCs are the treatment of choice for severely hypoplastic molars.[36]

I. Prevent further tooth deterioration.
ii. Control tooth sensitivity.
iii. Establish correct interproximal contacts and proper occlusal relationships.
iv. Are not as technique sensitive or costly as cast restorations.
v. Require little time to prepare and insert.
vi. If not adapted properly, may produce an open bite, gingivitis or both.
vii. Properly placed, SSCs can preserve permanent first molars with MIH until cast restorations are feasible.

- Partial and full coverage indirect adhesive or cast crown and onlays. Cast restorations [37]

I. Require minimal tooth reduction.
ii. Minimize pulpal trauma.
iii. Protect tooth structure.
iv. Provide high strength for cuspal overlays.
v. Control sensitivity.
vi. Maintain periodontal health due to their supragingival margins

Anterior teeth can be managed with veneers or crowns should they be indicated for severe cases of enamel defects, and where esthetic concerns continue to be an issue.[38]
- Timely extraction is a feasible treatment option in cases of: [39]

I. Severe hypomineralisation.
ii. Severe sensitivity or pain.
iii. Large multi surface lesions
iv. Difficulty of restoration.
v. Inability to achieve local anesthesia.
vi. Behavior management problems preventing restorative treatment.
vii. Apical pathosis.
viii. Orthodontic space requirements, where first permanent molars are heavily restored in the presence of healthy premolars.
ix. Crowding distally in the arch and third permanent molars reasonably positioned. - Financial considerations precluding other forms of treatment.
x. If the orthodontic condition were favourable, the ideal dental age for extracting the defective permanent first molars would be 8.5 to 9 years of age.[40]

RECENT ADVANCES: [41]
Elmex gel is a drug registered with the OICM for intensive anti-decay prophylaxis, and is especially indicated in the event of high levels of decay activity, sensitive tooth necks, and in the event of enamel demineralization. Effects are stimulation of remineralisation, reduction in solubility in an acidic medium, homogeneous action across all dental surfaces, and inhibition of plaque formation obtained through anti-microbial action. Composition –amine fluoride (0.25%,

2500ppm), sodium fluoride (1%, 1ppm), excipients, water and a humectant.

Since the quality of enamel supporting the restoration is frail, the prognoses of restorations are poor. The need for evaluating restoration at regular intervals becomes mandatory. However, failing restorations always necessitate treatment planning for techniques and materials that last longer; hence, emphasis should be more on radical tooth preparations in contrast to the usual conservative preparations.[42]

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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