

Molar incisor hypomineralization

Halenur Onat^{1*}, Gül Tosun¹

¹Department Pediatric Dentistry, Faculty of Dentistry, University of Selcuk, Konya, Turkey

ABSTRACT

Molar incisor hypomineralization (MIH) describes the clinical picture of hypomineralization of systemic origin affecting one or more first permanent molars that are associated frequently with affected incisors. Early diagnosis is essential since rapid breakdown of tooth structure may occur, giving rise to acute symptoms and complicated treatment. The purpose of this article is to review the diagnosis, putative etiological factors and to present a sequential approach to management of MIH.

Key words: Enamel Hypomineralization, Incisor, Molar, Molar Incisor Hypomineralization

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INTRODUCTION

Tooth development may be influenced by various factors (such as febrile illness, antibiotic use and excessive fluoride intake) during, before or after birth.^[1] Depending on the timing and duration of these factors, teeth may undergo various pathological conditions. During the developmental stages of the formation of teeth, is normal formation, but corrupt mineralization may result in “hypomineralization” while corrupt formation but normal mineralization might lead to “hypoplasia” occurs.^[2,3]

During the late 1970s, fragmentation, degradation or presence of fractures associated with extreme hypomineralization in severe conditions and often containing enamel defects in the molars and canine, white-yellow or yellow-brown or creamy yellow in color and with limited opacity, have been identified.^[4]

Such defects as they were observed were classified as developmental enamel defects of the molars are classified as “permanent hypomineralized first molars (FMs),” “idiopathic enamel hypomineralization of the FMs,” “cheese molars,” “hypomineralization irrespective of dental fluorosis,” and “demineralized FMs.”^[4-7]

During the European Academy of Pediatric Dentistry (EAPD) held in 2001, researchers have reached a consensus on a single description. The condition was described as the “molar incisor hypomineralization (MIH) in limited and qualitative defects of enamel origin affecting one of multiple molars with or without incisor retention.”^[8]

ETIOLOGY OF MIH

Factors causing MIH should be studied within three terms, which are prenatal, perinatal and postnatal periods.^[1]

Prenatal period

It was suggested that maternal disorders such as cardiologic diseases, infections of the urinary tract, A and D vitamin deficits, anemia, toxicity, diabetes mellitus and rubella embryopathy during pregnancy might result in developmental enamel defects in the child.^[9]

Perinatal period

During this term, various medical conditions may affect the infant’s health either combined, or individually. Cesarean section, prolonged delivery, preterm delivery and twin deliveries are among the very frequent perinatal problems/conditions. MIH is reported to be higher

*Address for correspondence

Miss Halenur Onat, Department Pediatric Dentistry, Faculty of Dentistry, University of Selcuk, Konya, Turkey.
E-mail: onat_2012@windowslive.com

compared to normal children in such cases.^[10] In addition, enamel defects in permanent teething, a difference in teeth development and extraction timings is also the case in premature children under the risk of miscarriage.^[11]

Prolonged respiratory suppression during preterm delivery may lead to hypoxia. Oxygen deficiency of the ameloblasts are considered as the cause of MIH or molar and incisor opacities in medical problems of this kind.^[7]

Hypocalcemia is a pathological condition encountered as a result of the plasma blood calcium levels less than 7.5/100 ml. In addition to the perinatal term, hypocalcemia may also occur during the prenatal and postnatal periods. Low calcium levels in MIH lesions are the result of degenerated calcium metabolism of ameloblasts.^[12] Hypocalcemia may occur in mothers with diabetes, in the case of vitamin D deficiency, prenatal and/or perinatal period and premature delivery.^[12]

Since a large portion of calcium and phosphate depot in neonates is formed during the last trimester, Ca and P are not sufficiently stored in preterm birth.^[13] Experimental studies demonstrate that the effect of hypocalcemia on the developing teeth tissues is related to the term of hypocalcemia. A diet poor in Ca is shown to reduce dentine thickness at the initial stage while a diet longer than 10 weeks increased hypoplastic response in secreted enamel.^[14]

Postnatal period

Several studies demonstrate that the medical problems faced during the postnatal term result in the occurrence of MIH. In a study conducted in Switzerland, the relationship between the diseases during the 0 and 1st years of age and MIH were found relevant in males only.^[15] Frequency of MIH in children with a systemic disease history in the first 3 years of age was higher than those without a disease history.^[16] Due to a large number of systemic etiologic factors during dental development, which occur simultaneously, it is highly difficult to isolate the factors or classify them in the order of relevance.^[17]

DIAGNOSIS AND TREATMENT OF MIH

MIH demonstrates an appearance that ranges between limited opacities in the enamel and loss of substance at various levels. Patients and parents were reported to complain of MIH with symptoms including esthetic problems, rapid dental wear and loss of enamel, inclination to caries, dental sensitivity and ultimate tooth loss.^[6,18]

Post-teething breaks (PTBs) in MIH may lead to the exposure of the porous sub-surface of the enamel or even the dentine, leading to the development of hypersensitivity

of the teeth to cold food, cool temperatures and brushing.^[18] Poor oral hygiene causes increased plaque retention in teeth with MIH, thereby supporting rapid caries development.^[6]

MIH defects not only cause clinical problems for children, but also the dentists. Due to sensitivity, patients may demonstrate dentist fear, concern and reluctance during treatment.^[18] “Acquired pain” resulting from behavioral problems; treatment using the insufficient anesthesia or local anesthesia are associated with their acquired experience.^[19]

Before starting treatment of teeth MIH, the following applications should be performed and treatment should be initiated in line with the obtained information:^[20]

- Permanent FMs and incisors (12 teeth) should be examined.
- During examinations performed for MIH, teeth should be examined in wet condition after sanitation.
- Eighth year of age is the optimum time for MIH examination.
- The following information should be recorded for each tooth:
 - Presence or absence of limited opacity.
 - Post-denting enamel breaks.
 - Atypical restoration.
 - Extraction due to MIH.
 - Failed denting of incisors or molars.

The approach to a six-step treatment developed by William *et al.* (2006) for the successful management of MIH teeth has been demonstrated [Table 1].^[21]

In addition to the six-step treatment approach by William *et al.*, a “treatment decision tree” based on the severity of the disease and the length of treatment duration has been prepared by Mathu-Muju and Wright (2006). According to the authors, the following clinical criteria are classifying the defect in three different degrees of severity [Table 2].^[22]

Preventive treatments

Directing children affected by MIH and their parents to suggestions for appropriate diets and preventive treatment is of crucial importance. Children with MIH, using toothpaste low in fluoride content should start using high fluoride toothpastes containing 1000 ppm F at minimum.^[23] Topical fluoride applications may be also useful. All of these methods ensure reduced dental sensitivity and assist to increased mineralization of the hypomineralization areas.^[1]

Another product for use would be casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), which is believed to be useful for teeth with MIH.^[21,23] Thanks to its

Table 1: A clinical management approach for permanent FMs affected by MIH

Steps	Recommended procedures
Risk identification	Assess medical history for putative etiological factor
Early diagnosis	Examine at-risk molars on radiographs if available Monitor these teeth during eruption
Remineralization and desensitization	Apply localized topical fluoride
Prevention of dental caries and PEB	Institute thorough oral hygiene home care program Reduce cariogenicity and erosivity of diet Place pit and fissure sealants
Restorations or extractions	Place intracoronal (resin composite) bonded with a self-etching primer adhesive or extracoronal restorations (stainless steel crowns) Consider orthodontic outcomes post-extraction
Maintenance	Monitor margins of restorations for PEB Consider full coronal coverage restorations in the long-term

FMs: First molars, MIH: Molar incisor hypomineralization, PEB: Post-eruption breakdown

Table 2: Treatment decision tree

Level of MIH	Symptoms
Mild MIH	Demarcated opacities are in non-stress-bearing areas of FPM Isolated opacities No enamel loss from fracturing is present in opaque areas No history of dental hypersensitivity No caries associated with the affected enamel Incisor involvement is usually mild if present
Moderate MIH	Intact atypical restorations Demarcated opacities are present on occlusal/incisal third of teeth without posteruptive enamel breakdown Posteruptive enamel breakdown/ caries are limited to 1 or 2 surfaces without cuspal involvement Dental sensitivity is generally reported as normal esthetic concerns
Severe MIH	Posteruptive enamel breakdown History of dental sensitivity Widespread caries is associated with the affected enamel Crown destruction can readily advance to involve the dental pulp Defective atypical restoration is present Esthetic concerns are expressed by the patient or parent

MIH: Molar incisor hypomineralization, FPM: First permanent molar

anti-sensitive property, CPP-ACP is a biocompatible source of Ca and P for MIH teeth in the extraction process.^[23] Lastly, the daily use of 0.4% stannous fluoride gel is recommended for reduced sensitivity in defective teeth.^[24]

Fissure sealant (FS)

Fissure sealant (FS) application on the defective surface is yet another treatment option recommended for eliminating sensitivity and preventing caries in moderate cases where the enamel integrity is not lost.^[24]

FS may be replaced by glass ionomer cement (GIC) in MIH affected teeth not completely erupted. This treatment provides temporary protection for the teeth against caries and sensitivity and minimizes PTBs. Due to their poor retention, materials of this kind should be replaced by a resin based FS after complete tooth eruption.^[21]

Restorative treatments

Decisions as to the use of restorative material for the hypomineralized permanent FM restoration should consider the width of defect, specific tooth structure intended for restoration, dentine sensitivity and eruption.^[25] Moreover, sufficient saliva isolation in hypomineralized permanent FMs as the teeth located at the distant back of the arch in patients 6-7 years of age is highly difficult. For this reason, the patient’s age and compatibility should be also taken into consideration.^[24]

The initial approach in defining cavity limits is the complete removal of the defective enamel. While it reduces the risk of failure of restoration, this option leads to increased loss of dental tissue. The second approach is the removal of the porous enamel only while leaving the enamel with increased resistance to drill intact. Although conservative, this option still bears the risk of break in the restored edges.^[24]

The porous exposed subsurface enamel or dentin may promote chronic inflammation of the pulp, complicating anesthesia.^[21] Apart from the restorative difficulties faced by clinicians, children with MIH have considerable behavior management problems; dental fear and anxiety are more commonly found in these children. Behavior problems can be related to pain experienced by the patients during multiple treatment appointments as many of them were either inadequately anesthetized or even had treatment without local analgesia.^[18]

GICs

Due to their adhesive property, GICs are good isolators and release fluoride. However, their use is restricted in hypomineralized permanent FMs with low wear resistance and affected by the chewing force in particular.^[24] GICs are recommended as cement for temporary restoration of MIH affected teeth or due to their properties close to dentine.^[24]

Resin composite materials

Compared with other restorative materials in MIH affected teeth, the composite resin material provides longer

stability (approximately 5.2 years and a success rate by 74-100%).^[26] Self-etching adhesive (SEA) was found to have better bond strength to MIH affected enamel than all-etch single-bottle adhesive (SBA) in a laboratory study.^[21] This was attributed to the omission of rinsing, thus eliminating any interference of residual water on the bond and to the presence of both micromechanical and chemical bonds between hydroxyapatite and SEA. Alternatively the hydrophilic properties of acetone included in some other SBA systems, may play the same role for eliminating the residual water from the etched enamel surface.^[26]

Amalgams

Amalgams mechanically adhering to the restoration cavity and demonstrating frequent leak in restored edges are bad isolators not chemically bound to the dental tissues. Due to these properties, they are not considered an effective treatment option for MIH affected teeth.^[24] While many studies conducted on MIH affected teeth showed that amalgam restorations are renewed more frequently than composites and that composites have better properties, there are also opinions suggesting their equal percentage of success.^[27,28]

Stainless steel crowns (SSC)

Stainless steel crowns (SSC) is the best option for restoration in teeth with severe coronal malformation and can be implemented temporarily until the adjacent permanent tooth erupts and reaches the occlusion plane.^[24] SSC is preferred for preventing material loss better than the caries-free, sound hypomineralized areas, ensuring control over sensitivity, providing proper contact in the interfaces and occlusal; cost-effectiveness and fast application.^[21]

Laboratory-fabricated crowns

Laboratory-fabricated crowns are oftentimes not preferred in clinical applications due to large pulps of young permanent teeth, short crowns, expensiveness, increased number of treatment sessions, causing wear of the opposing teeth and difficulty of finishing the crown edges in permanent teeth not completely erupted.^[29,30]

Extraction and orthodontic approach

Extraction in MIH affected teeth where severe coronal loss is the case should be considered as an option for treatment if the patient has crowding, in frequently repeated treatments and cases where the pulpal symptoms cannot be eliminated.^[8,18] Extractions should consider orthodontic complications. The optimum age range for extraction is 8.5 and 9 years of age in the condition is suitable in terms of orthodontic. The extracted first permanent molar is replaced with the second permanent molar by proper occlusion following extraction.^[18,31]

Orthodontic treatment is often required after the extraction procedure in the case of bad timing.^[8,18] Closing of the extraction gaps formed in the upper jaw are usually easier than those in the lower jaw. The best results in the extraction of the permanent FMs were obtained in the procedures performed between 8 and 10 years of age where the permanent lateral incisors begin to grow while the permanent second premolars and molars are not yet being erupted.^[32]

Restoration of defective teeth is recommended due to orthodontic restrictions such as the lack of crowding other than the age factor, improper skeletal structure or congenital dental deficit.^[24,31] However, defective teeth may be extracted in many cases despite the trial of all treatment options. Therefore, it was reported that a mutual decision between orthodontists and pediatric dentists would be more appropriate for the identification of the best treatment alternative of severely defective permanent FMs.^[31]

Incisor treatment

Although, it is recommended to wait until the patient's end of puberty for the treatment of defective incisors where loss of enamel and sensitivity are frequently observed, the increasing demand for esthetics should be satisfied starting from the growth of these teeth.^[24] Composite laminates implemented with or without removal of a certain amount of enamel tissue are more effective in improving the esthetic appearance.^[5,24]

CONCLUSIONS

Although the reason is not completely known, MIH is thought to occur as a result of the multifactorial reasons during the child's prenatal term or cases putting the health in danger such as systemic diseases and malnutrition during the child's first 3 years of age.

Clinical MIH is a serious problem for both, children and dentists. These teeth are sensitive to cold and hot temperatures and cause mild to severe pain. Therefore, the patient's concerns should be reduced and behavioral guidance should be provided. Factors causing the defect should be disclosed and proper treatment options should be developed for children affected by MIH, who undergo treatment more frequently than those unaffected.

REFERENCES

1. Alaluusua S. Aetiology of Molar-Incisor Hypomineralisation: A systematic review. *Eur Arch Paediatr Dent* 2010;11:53-8.
2. Clarkson J. Review of terminology, classifications, and indices of developmental defects of enamel. *Adv Dent Res* 1989;3:104-9.
3. Jälevik B, Norén JG. Enamel hypomineralization of permanent first molars: A morphological study and survey of possible aetiological factors. *Int J Paediatr Dent* 2000;10:278-89.

4. Koch G, Hallonsten AL, Ludvigsson N, Hansson BO, Holst A, Ullbro C. Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children. *Community Dent Oral Epidemiol* 1987;15:279-85.
5. Croll TP. Creating the appearance of white enamel dysmineralization with bonded resins. *J Esthet Dent* 1991;3:30-3.
6. Leppäniemi A, Lukinmaa PL, Alaluusua S. Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. *Caries Res* 2001;35:36-40.
7. van Amerongen WE, Kreulen CM. Cheese molars: A pilot study of the etiology of hypocalcifications in first permanent molars. *ASDC J Dent Child* 1995;62:266-9.
8. Weerheijm KL, Jälevik B, Alaluusua S. Molar-incisor hypomineralisation. *Caries Res* 2001;35:390-1.
9. Hall RK. Prevalence of developmental defects of tooth enamel (DDE) in a pediatric hospital department of dentistry population (I). *Adv Dent Res* 1989;3:114-9.
10. Lygidakis NA, Dimou G, Briseniou E. Molar-incisor-hypomineralisation (MIH). Retrospective clinical study in Greek children. I. Prevalence and defect characteristics. *Eur Arch Paediatr Dent* 2008;9:200-6.
11. Harila-Kaera V, Heikkinen T, Alvesalo L. The eruption of permanent incisors and first molars in prematurely born children. *Eur J Orthod* 2003;25:293-9.
12. Jälevik B, Norén JG, Klingberg G, Barregård L. Etiologic factors influencing the prevalence of demarcated opacities in permanent first molars in a group of Swedish children. *Eur J Oral Sci* 2001;109:230-4.
13. Seow WK. Effects of preterm birth on oral growth and development. *Aust Dent J* 1997;42:85-91.
14. Ranggård L, Norén JG. Effect of hypocalcemic state on enamel formation in rat maxillary incisors. *Scand J Dent Res* 1994;102:249-53.
15. Jälevik B, Klingberg G, Barregård L, Norén JG. The prevalence of demarcated opacities in permanent first molars in a group of Swedish children. *Acta Odontol Scand* 2001;59:255-60.
16. Kuscü OO, Caglar E, Aslan S, Durmusoglu E, Karademir A, Sandalli N. The prevalence of molar incisor hypomineralization (MIH) in a group of children in a highly polluted urban region and a windfarm-green energy island. *Int J Paediatr Dent* 2009;19:176-85.
17. Seow WK. Enamel hypoplasia in the primary dentition: A review. *ASDC J Dent Child* 1991;58:441-52.
18. Jälevik B, Klingberg GA. Dental treatment, dental fear and behaviour management problems in children with severe enamel hypomineralization of their permanent first molars. *Int J Paediatr Dent* 2002;12:24-32.
19. Weerheijm KL. Molar incisor hypomineralisation (MIH). *Eur J Paediatr Dent* 2003;4:114-20.
20. Weerheijm KL, Mejäre I. Molar incisor hypomineralization: A questionnaire inventory of its occurrence in member countries of the European Academy of Paediatric Dentistry (EAPD). *Int J Paediatr Dent* 2003;13:411-6.
21. William V, Messer LB, Burrow MF. Molar incisor hypomineralization: Review and recommendations for clinical management. *Pediatr Dent* 2006;28:224-32.
22. Mathu-Muju K, Wright JT. Diagnosis and treatment of molar incisor hypomineralization. *Compend Contin Educ Dent* 2006;27:604-10.
23. Willmott NS, Bryan RA, Duggal MS. Molar-incisor-hypomineralisation: A literature review. *Eur Arch Paediatr Dent* 2008;9:172-9.
24. Fayle SA. Molar incisor hypomineralisation: Restorative management. *Eur J Paediatr Dent* 2003;4:121-6.
25. Croll TP. Restorative options for malformed permanent molars in children. *Compend Contin Educ Dent* 2000;21:676-8, 680, 682.
26. Lygidakis NA. Treatment modalities in children with teeth affected by molar-incisor enamel hypomineralisation (MIH): A systematic review. *Eur Arch Paediatr Dent* 2010;11:65-74.
27. Kotsanos N, Kaklamanos EG, Arapostathis K. Treatment management of first permanent molars in children with Molar-Incisor Hypomineralisation. *Eur J Paediatr Dent* 2005;6:179-84.
28. Mejäre I, Bergman E, Grindefjord M. Hypomineralized molars and incisors of unknown origin: Treatment outcome at age 18 years. *Int J Paediatr Dent* 2005;15:20-8.
29. Zagdwon AM, Fayle SA, Pollard MA. A prospective clinical trial comparing preformed metal crowns and cast restorations for defective first permanent molars. *Eur J Paediatr Dent* 2003;4:138-42.
30. Koch MJ, García-Godoy F. The clinical performance of laboratory-fabricated crowns placed on first permanent molars with developmental defects. *J Am Dent Assoc* 2000;131:1285-90.
31. Williams JK, Gowans AJ. Hypomineralised first permanent molars and the orthodontist. *Eur J Paediatr Dent* 2003;4:129-32.
32. Thilander B, Skagius S. Orthodontic sequelae of extraction of permanent first molars. A longitudinal study. *Rep Congr Eur Orthod Soc* 1970:429-42.

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