10.5005/jp-journals-10024-1898

REVIEW ARTICLE

Molar Incisor Hypomineralization

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ABSTRACT

Molar incisor hypomineralization (MIH) is a developmental defect affecting teeth. High prevalence rates of MIH and its clinical implications are significant for both the patients and clinicians. A wide variation in defect prevalence (2.4–40.2%) is reported. It seems to differ with regions and various birth cohorts. Some of the recent prevalence studies are tabulated.

Patient implications include hypersensitive teeth, rapid progression of caries, mastication impairment due to rapid attrition, and esthetic repercussions. Implications for clinicians include complexity in treatment planning and treatment implementation, poor prognosis of the restorations, difficulty in achieving pain control during treatment, and behavior management problems. Intention of this paper is to review the etio-pathogenesis, prevalence, clinical features, diagnostic features, and eventually present a sequential treatment approach, i.e., in accordance with current clinical practice guidelines.

Keywords: Dental enamel hypoplasia, Incisor pathology, Molar incisor hypomineralization, Molar pathology.

How to cite this article: Rao MH, Aluru SC, Jayam C, Bandlapalli A, Patel N. Molar Incisor Hypomineralization. J Contemp Dent Pract 2016;17(7):609-613.

Source of support: Nil

Conflict of interest: None

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INTRODUCTION

Tooth hypoplasia is a defect in quality and quantity of tooth structure due to developmental origin.[1] Molar incisor hypomineralization (MIH) is a specific form of tooth hypoplasia. Defined MIH as a "hypomineralization of one to four permanent first molars frequently associated with affected incisors and is due to systemic origin."[2,3]

SYNONYMS

Other phrases used to describe MIH include idiopathic enamel hypomineralization in the permanent first molars, idiopathic enamel opacities of permanent first molars, nonfluoride enamel hypomineralization of permanent first molars, and nonendemic mottling of enamel of permanent first molars. Molar incisor hypomineralization is today the proposed phrase for this condition.[4]

A similar condition that shows hypomineralization of permanent first molars alone with noninvolvement of permanent incisors is termed as molar hypoplasia (MH). It is seen that MIH and MH form part of an MIH spectrum, where MIH is a more severe form of the condition than MH.[5]

Another condition that has been noticed is deciduous molar hypomineralization (DMH), presenting itself as hypomineralization of deciduous molars (DH). Deciduous molar and DMH share a common etiology and hence presence of DMH in deciduous dentition can be used to predict MIH in the permanent dentition. Interception of future undesired sequel can be planned early.[6]

Ghanim et al,[7] in their study, showed MIH-affected children significantly seek dental care more often than normal children (82.4, 68.2% respectively). They were over three times more likely to visit the dentist complaining of pain and were over six times more likely to seek dental care due to tooth sensitivity than normal children.

Teeth affected with MIH are very porous, leading to increased plaque formation and caries susceptibility. Additionally, these porous teeth are very sensitive,

making maintenance of effective plaque control difficult. Both factors increase the risk of caries. It is seen these teeth are very sensitive to cavity preparation procedures; thus, a patient may show greater anxiety and behavioral problems.[8]

The work done by European Academy of Pediatric Dentistry (EAPD) with regard to MIH is to be acknowledged. Recognizing the lack of existing knowledge regarding MIH, it promoted further research in this regard. In 2003, European Archives of Pediatric Dentistry (EAPD) set up a Defect Diagnostic Criteria. It published various papers and a whole issue assigned to the defect in the European Archives of Pediatric Dentistry (2008). European Archives of Pediatric Dentistry also called for an Interim Seminar and Workshop on MIH in Helsinki in 2009.[2,4,5,7]

ETIOLOGY

Investigators have put forward a number of possible causes; the etiologies were divided into five groups: (1) Exposure to environmental contaminants, (2) pre/peri and neonatal problems, (3) exposure to fluoride, (4) common childhood illnesses, and (5) medically compromised children.[3]

Willmott et al[9] suggested asthma, pneumonia, upper respiratory tract infections, otitis media, antibiotics, dioxins in mother's milk, tonsillitis and tonsillectomy, and exanthamatous fevers of childhood. Crombie et al[10] opined polychlorinated biphenyl/dioxin nutrition, birth and neonatal factors, and acute or chronic childhood illness/treatment, fluoride or breastfeeding. Alaluusua[11] in his review suggested high fever, hypoxia, hypocalcemia, exposure to antibiotics (amoxicillin), and dioxins as risk factors.

Combined effect of several factors should also be taken into account.

Despite existing knowledge regarding etiological factors, adequate proof to validate the effect of etiological factors is still required. Experimental dose/response studies on molecular mechanism of ameloblasts are essential to deepen our knowledge of presently presumed factors. Also, prospective studies are needed to reveal new factors that might be involved.[9-11]

With regard to the timing of the defect formation, the review of the literature has shown systemic factors act around the time of birth or during the child's first 3 years (corresponds to formative stages of incisors and molars).

PREVALENCE

A wide variation in defect prevalence (2.4–40.2%) is reported. It seems to differ with regions and various birth cohorts.[2] Cross comparison of results of various studies are difficult because of use of different indices and criteria, examination variability, methods of recording, and different age groups.[12] Some of the recent prevalence studies are tabulated (Table 1).

Table 1: Different studies and prevalence rates in various populations

Study	*Population*	*Prevalence*
Kukleva et al (2008)[13]	Plovdiv, Bulgaria	3.58% (2.43–7.84%)
Mahoney and Morrison (2009)[14]	Wellington, New Zealand	14.9%
Biondi et al (2011)[15]	Buenos Aires	15.9% (13.8–18.2%)
Balmer et al (2012)[16]	Northern England	15.9% (14.5–17.1%)
Parikh et al (2012)[17]	Gujarat, India	9.2%

Molar incisor hypomineralization was more common in preterm children than in controls (38% *vs* 16%), as were enamel developmental defects (69.5% *vs* 51%). Low gestational age and low birth weight increased the risk of MIH.[18]

CHIEF COMPLAINT

Children and/or accompanying guardians usually complain of presence of defective molar teeth and/or incisors. These are commonly referred to as "cheese molars," because the lesions clinically resemble cheese in color and consistency.

Yet again, depending on the severity of disease, age of interaction of etiological agent, and age of presentation of patient to dental clinic, a wide variety of complaints include sensitivity to air, cold, warm, and mechanical stimuli; inability to chew food; carious teeth and its sequel. In severe conditions, inability to brush teeth due to tooth ache has also been reported.

Patients may even complaint of repeated or failing restorations.

CLINICAL DIAGNOSIS

On examination, there may be hypoplasia involving one or more permanent first molars and incisors. Lesions are more frequent in the upper jaw than in the lower jaw. Occlusal surfaces are most commonly affected.[2-4,19]

Affected teeth show demarcated enamel opacities, ranging from white to brown, according to the severity of the disease and the hypoplasia that can be associated (Figs 1 to 5). Hypomineralized enamel is soft and porous and occasionally it undergoes to posteruptive breakdown, resulting in anomalous noncarious cavities. This rapid breakdown of the teeth often calls for extensive restorative procedures. Molar incisor hypomineralization molars are fragile, and caries can develop very easily in these molars. This can give raise to higher DMF index.

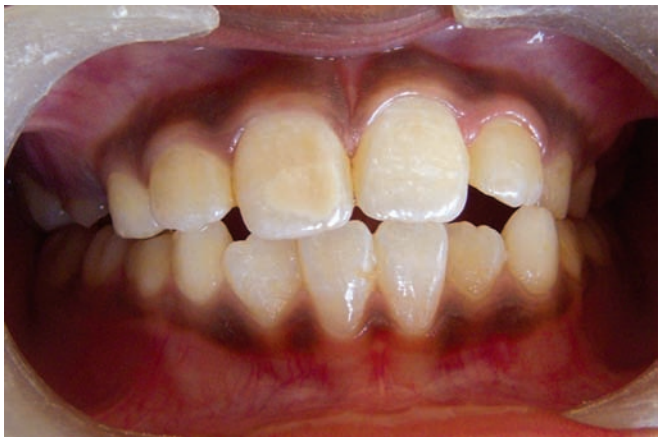

Fig. 1: Labial aspect showing hypoplastic incisors

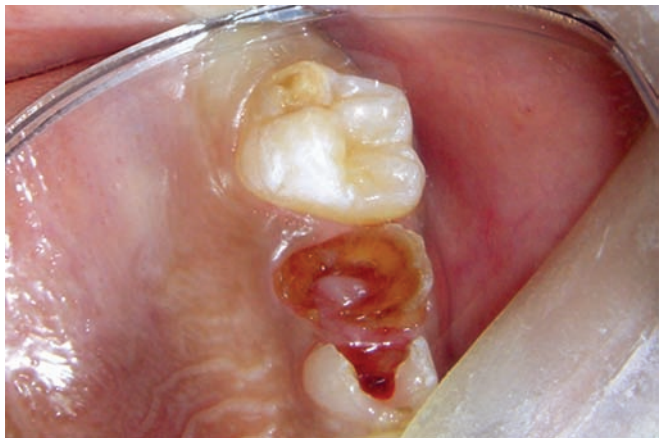

Fig. 2: Right maxillary view showing hypoplastic tooth 16

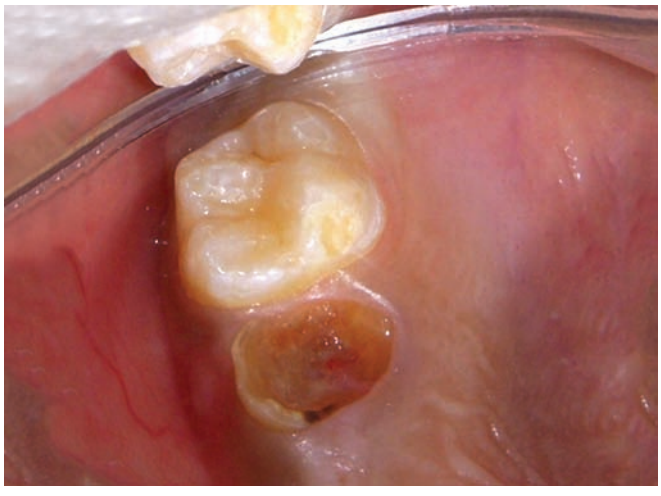

Fig. 3: Left maxillary view showing hypoplastic tooth 26

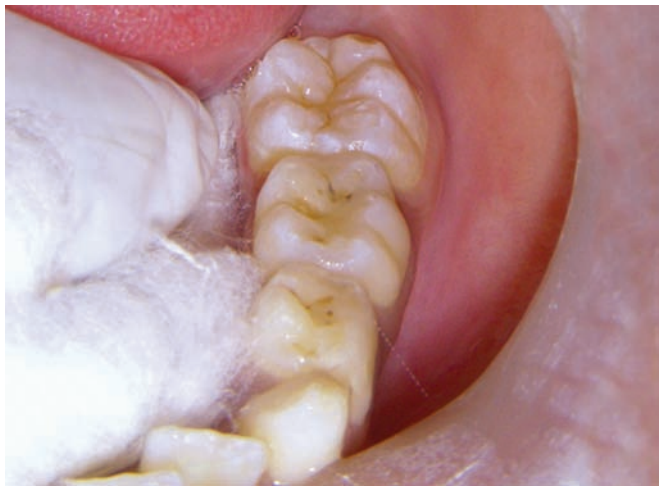

Fig. 4: Left mandibular view showing hypoplastic tooth 36

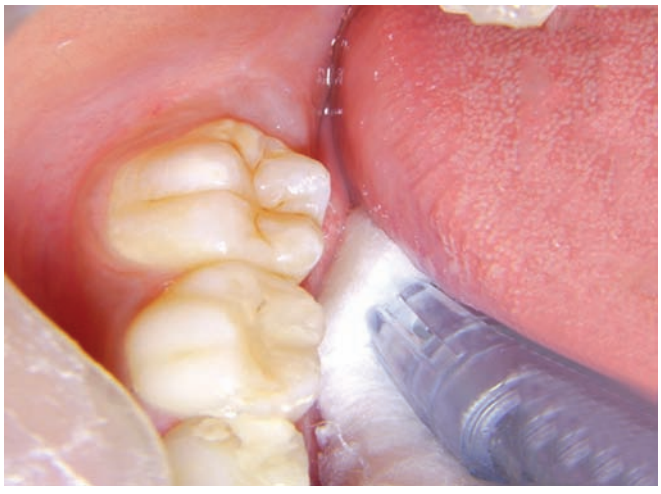

Fig. 5: Right mandibular view showing hypoplastic tooth 46

The affected teeth are very sensitive to air, cold, warm, and mechanical stimuli. Rodd et al[20] showed increased expression of transient receptor potential ion channel (TRPV1), a noxious heat receptor within the pulps of hypomineralized teeth, indicative of an underlying pulpal inflammation and also explained the heat sensitivity experienced by some patients with this condition.

European Archives of Pediatric Dentistry has put forward specific criteria for diagnosis of this phenomenon. This includes presence of demarcated opacity, posteruptive enamel breakdown, atypical restoration, extracted molar due to MIH, and unerupted teeth.[3]

INVESTIGATIONS FOR STRUCTURAL AND MECHANICAL CHARACTERISTICS

The microstructure of hypomineralized enamel in MIH has been described in several studies. Microstructural changes take place during enamel maturation. Microstructure of enamel shows loosely packed hydroxyapatite crystals. Enamel rods are unorganized with rod sheaths that are less distinct. This may be responsible for the dramatic reduction in mechanical properties of the affected regions.[21]

Comparative evaluations for mechanical properties, hardness, and modulus of elasticity of the hypomineralized enamel have shown lower values when compared with normal enamel structure.[22] Clinically this is of significance; attrition of the hypoplastic molars can lead to decrease in vertical dimension and patient having an collapsed bite.

Histologically affected teeth show enamel porosities of varying degree. Studies on chemical makeup of MIH enamel have shown that mean Ca/P ratio in hypomineralized areas was significantly lower (1.4) than the mean Ca/P ratio in the adjacent normal enamel (1.8).[23]

PREVENTION AND INTERCEPTION

An early diagnosis and treatment planning as well as prognostication of hypomineralized first molars are desirable. A six-step approach to management is described: (1) Risk identification, (2) early diagnosis, (3) remineralization and desensitization, (4) prevention of caries and posteruption breakdown, (5) restorations and extractions, and (6) maintenance.[24]

Da Costa-Silva et al[25] determined risk factor calculation based on color of enamel opacities. In their longitudinal study, they recorded enamel opacities according to color shades of white, yellow, and brown, allowing assessment of susceptibility to structural loss over time. It was concluded that teeth with yellow and brown opacities were at high-risk than lighter ones. These results can help clinicians determine a risk-based treatment for children with MIH. Also, DMH can be used clinically as a future predictor for MIH. Early planning and necessary intervention can intercept disease progression.[6]

Interception of further breakdown and caries progression can be done by regular use of fluorides, amorphous calcium phosphate solutions, and pit and fissure sealants. Since the patients are on soft pureed diet, diet counseling is of utmost importance. Enhancement of regular oral hygiene procedures should be recommended to prevent decay and enhance the prognosis of restoration following treatment procedures.

Risk factors, such as malnutrition and childhood diseases are seen to enhance MIH. The author opines that, "prevention in true sense" for the disease might require a better effort on the part of health care providers to provide healthier ante- and postnatal care and decrease the ill effects of associated risk factors.

TREATMENT

Molar incisor hypomineralization is an important clinical problem that often concerns both general dental practitioners and pediatric dentists. The author opines that pediatric dentists especially should be aware of MIH condition because (1) These children are more likely to visit dental service at a younger age because the condition presents early in childhood, (2) severe morbidity leading to handicapped dentition at early age, (3) complexity of disease as well as its treatment, (4) poor prognosis of restorations and long-term follow-up required, and (5) associated behavioral problems.

The complex care involved in treating affected children must address their behavior and anxiety, aiming to provide a durable restoration under pain-free conditions. The challenges include inadequate pain control, complex cavity designs, and preference of restorative materials (restorations in hypomineralized molars appear to fail repeatedly). There are no consensus to facilitate clinical decisions regarding cavity design and material choice.[26,27]

Restorations with glass ionomer cement, composite, stainless steel crowns, full veneer metal-ceramic crowns, fixed-removable partial dentures, and/or implants are the different treatment options that are discussed in various studies.

Restorative options for affected molar vary from adhesive intracoronal restorations (composite is the material of choice) to extracoronal restorations (e.g., preformed metal crown). Esthetic solutions for affected incisors include microabrasion with resin composite or porcelain veneer to full veneer crowns in childhood and metal/metal-ceramic full veneer crowns in adulthood.

Furthermore, adhesion of composite resins to hypomineralized tissue is clinically significant. The acid-etched hypomineralized enamel that appeared on SEM studies is shown to be covered with a structureless layer and, enamel prisms appeared disorganized with thick prism sheaths and loosely packed crystallites. Bacteria were also found deep in porous hypomineralized enamel close to the enamel–dentin junction.[22]

Due to the presence of chronic pulpitis that is often seen with hypomineralized teeth, behavior problems occur.[28] It is reasonable to assume that behavior management problems and dental fear and anxiety on repeated occasions are related to experiences of pain due to chronic pulpitis.[29] Local anesthesia and other pain-reducing techniques, for example, sedation be used when treating these teeth.[29,30]

Extraction should be considered if teeth are nonrestorable. In extraction cases, moreover, an interdisciplinary approach with an orthodontist should be planned for space management and restoration of function in these young children.

PROGNOSIS

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.

CONCLUSION

There is still need for further studies to shed light on causative factors and their effect as well as on improving the permanence of restorations.

Until then, the key for a successful treatment is early diagnosis, prompt treatment, and intense follow-up as soon as the teeth erupt.

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