Original Article

Molar Incisor Hypomineralization and Its Prevalence

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Abstract

Background: Molar incisor hypomineralization (MIH) is the hypomineralization of systemic origin of one to four permanent first molars, frequently associated with affected incisors. It is presented as demarcated enamel opacities of different colors, occasionally undergoing posteruptive breakdown. The characteristic feature of MIH is the clear demarcation between the affected and sound enamel. There is asymmetry of defects present in the molars and incisors where one molar or incisor can be severely affected, while the contralateral tooth may be clinically sound or have only minor defects. **Aim and Objective:** The aim of this study is to evaluate schoolchildren of 7–12 years of age with at least one of the first permanent molars fully or partially erupted from randomly selected government and private schools in Chennai using the European Academy of Pediatric Dentistry (EAPD) criteria for MIH, to determine the prevalence and characteristics of MIH. **Materials and Methods:** The dental examinations were performed in the classroom using a mouth mirror and explorer under a headlight. Teeth were wiped with gauze when necessary to remove plaque or the food accumulations. Surfaces that were examined were the buccal, lingual, palatal, and occlusal surfaces of permanent first molars and labial surfaces of upper and lower incisors. A single examiner was involved to avoid interexaminer bias. All the data were collected and scored using the EAPD criteria for MIH. **Results:** A total of 22 (12.9%) children out of the examined 170 had MIH. **Conclusion:** Distribution of MIH was more in males, more in 9 years of age. A total of 13 children had first molars affected and 9 children had both incisors and molars affected. Molars were affected more than the incisors. Mandible was affected more in comparison with the maxilla. Right side was affected more than the left side. The distribution of MIH was more in government schools compared to private schools.

Keywords: *Hypomineralization, molar incisor hypomineralization, permanent incisor, permanent molar, posteruptive breakdown, prevalence*

Introduction

Molar incisor hypomineralization (MIH) is the hypomineralization of systemic origin of one to four permanent first molars, frequently associated with affected incisors.[1-3] It is a qualitative defect of the enamel. It is also called as hypomineralized permanent first molars (PFMs), idiopathic enamel hypomineralization, nonfluoride hypomineralization, and dysmineralized PFMs. Koch *et al*. called these as cheese molars after carrying out the first epidemiological study.[4] When ameloblasts are affected in the late amelogenesis stage of mineralization or maturation, a defect in the enamel translucence can occur. These defects are called enamel hypomineralization. A common pattern of enamel hypomineralization affects molars and incisors. This dental defect is called the MIH.[5] It is clinically presented as white to yellow to brown demarcated enamel opacities of different colors, occasionally undergoing posteruptive breakdown due to soft and porous enamel.[2,3,6] Posteruptive enamel breakdown is a defect that indicates a decrease in the enamel depth after eruption. The posteruptive breakdown exposes the dentin which is very sensitive, and because of this, the tooth becomes vulnerable to rapidly progressing caries as the children cannot carry out oral hygiene very effectively.[1,7] Cavities and complete coronal distortion can occur requiring restorations.

The first report of MIH dates from the late 70's. In 2001, this defect was given a new name (MIH) with the definition of a "systemic hypomineralization" that affects one or more permanent first molars with or without permanent incisor involvement. The main characteristic of teeth with MIH is porous enamel that can be easily damaged due to masticatory forces. This can result in exposed dentinal tissues that

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may facilitate the development of carious lesions, hence MIH is associated with caries.[8] Children with MIH could present more intense dental sensitivity due to temperature variations. This is the result of the combination of the chronic pulp inflammation and innervation of the region right under the hypomineralized area. The children affected with MIH are at a risk of developing behavior management problems and dental fear because of the difficulties in achieving adequate anesthesia. The characteristic feature of MIH is the clear demarcation between the affected and sound enamel. There is asymmetry of defects present in the molars and incisors where one molar or incisor can be severely affected while the contralateral tooth may be clinically sound or have only minor defects. Rapid dental wear, loss of enamel, inclination to caries, dentin hypersensitivity, poor esthetics, anxiety, and tooth loss can occur in the long run.[7,9,10]

Restorative treatments for these hypomineralized teeth present with ten times more requirement than teeth without this condition. It ranges from prophylactic strategies to high complexity procedures.[11] Restorative treatments for these teeth are challenging for both the patient and the dentist due to the subclinical inflammation of pulpal cells and the altered porous enamel structure that makes bonding risky leading to defective and frequent loss of fillings and frequent retreatments which are painful because of anesthetizing difficulties.[7,12]

Knowledge of the magnitude of MIH is desirable as it is vulnerable for consequences such as rapid caries development, early enamel loss, and sensitivity. Hence, this study was conducted to evaluate the prevalence of MIH in schools of Chennai.

Materials and Methods

A descriptive cross-sectional study was done in four different schools of Chennai. The schools were randomly selected which include two private schools and two government schools. All the schoolchildren of 7–12 years of age were examined. The principal, teachers, and students were informed about the study and their consent was obtained. The research was conducted with approval from the Institutional Ethical Committee.

The inclusion criteria were children of 7–12 years of age with at least one of the first permanent molars and or incisors that are fully or partially erupted. Children with generalized development defects such as amelogenesis imperfecta, dentinogenesis imperfecta, hypoplasia, diffuse opacities, white spot lesions, tetracycline stains, erosion, fluorosis, and Turner's hypoplasia were excluded. Children wearing fixed appliances such as brackets and bands which interfere with evaluation of teeth and uncooperative children were also excluded.

The dental examinations were performed in the classroom using a mouth mirror and explorer under a headlight. Teeth were wiped with gauze when necessary to remove plaque or the food accumulations. Surfaces that were examined were the buccal, lingual, palatal, and occlusal surfaces of permanent first molars and labial surfaces of upper and lower incisors. A single examiner was involved to avoid interexaminer bias.

All the data were collected and scored using the European Academy of Pediatric Dentistry (EAPD) criteria for MIH.

EAPD criteria (2003):
- 0 – Normal
- 1 – Demarcated opacity
- 2 – Posteruptive enamel breakdown
- 3 – Atypical restorations
- 4 – Extracted molar due to MIH
- 5 – Unerupted molar due to MIH.

Results

A total of 170 children were examined from four different schools. Among them, 85 were male and 85 were female. Fifteen males and 7 females were affected with MIH [Graph 1]. A total of 22 (12.9%) children out of the examined 170 had MIH. Distribution of MIH was more in males, more in 9 years of age. A total of 13 children had first molars affected and 9 children had both incisors and molars affected [Graph 2]. Molars were more affected than incisors. Mandible was more affected. Right side was more affected [Figures 1 and 2]. The distribution of MIH was more in government schools compared to private schools.

Discussion

The children of age group 7–12 years were selected because at this age most children would have had all four first permanent molars and majority of incisors. These teeth would not have been exposed to the oral environment long enough to develop dental caries. At an older age, there would be a risk of posteruptive breakdown of enamel and caries initiation. Mandibular molars were more affected in this study which could be because they erupt earlier.

The prevalence in this study is less than that observed in Kavre district of Nepal.[1] This could be due to the difference in ethnicity and age groups, missing teeth, and unerupted teeth. The other findings were in accordance with the previous studies conducted in Chennai. The highest prevalence found till date is in Brazil of 40.2% in 2009.[2,6] Worldwide prevalence of MIH shows wide variation ranging from 2.4%–40.2%.[3,13,14] Weerheijm and Mejàre reported a prevalence rate ranging from 3.6% to 25% after carrying out studies in European countries.[15]

Hypomineralization is due to the combination of factors that may affect the ameloblasts resulting in abnormal enamel formation.[16,17,18] Systemic or environmental insults during the maturation stages of enamel development and genetic predisposition of ameloblasts to environmental

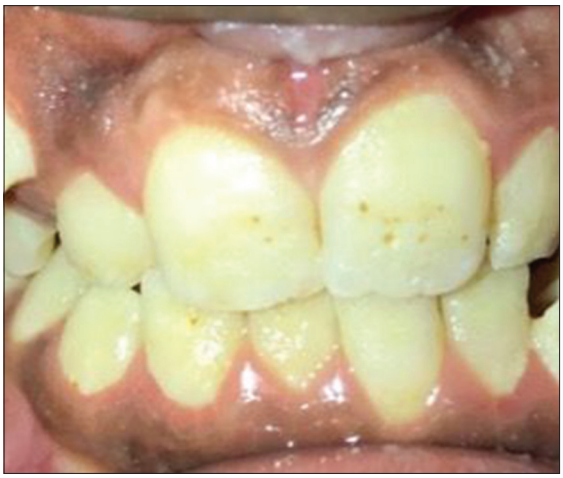


Figure 1: Hypomineralization in upper permanent central incisors

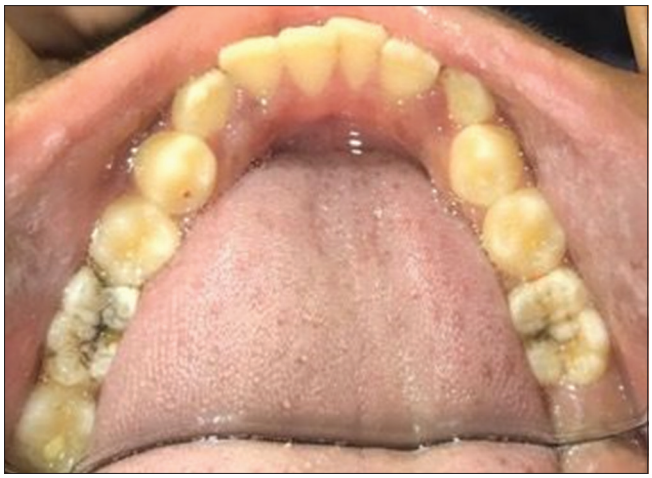
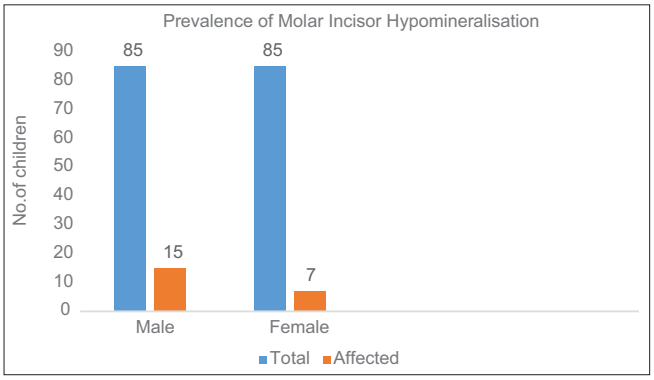
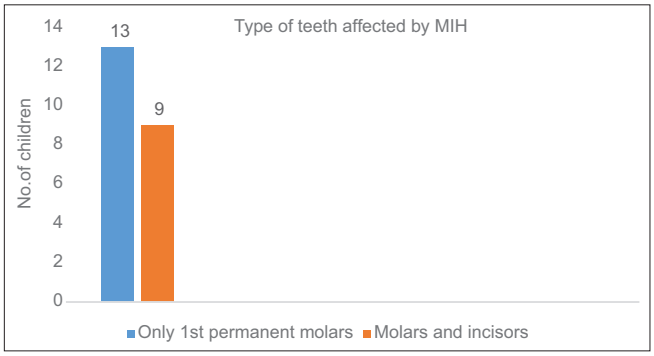


Figure 2: Hypomineralization in lower first permanent molars



Graph 1: Prevalence of Molar Incisor Hypomineralisation



Graph 2: Type of teeth affected by MIH

stressors have been reported to be a possible cause. Some authors have suggested MIH to be an autosomal recessive condition or localized amelogenesis imperfecta with possible association with genes related to enamel formation such as AMBN, TUFT1, and TFIP11.[19] MIH may be acquired as a result of prenatal, perinatal, and postnatal illness; low birth weight; antibiotic consumption; and toxins from breastfeeding.[1,11] Certain drugs such as chemotherapeutic, antibiotics, asthma, and antiepileptic drugs can cause MIH.[20,21]

Development of the first permanent molars and incisors begins at the 4th gestational month and hard tissue formation in them starts around or soon after birth. Enamel formation in the upper first incisors has been completed by the end of the 5th year of life and in the first molars at about 3 years. Accordingly, human permanent incisors and first molars are at greatest risk for defects caused by systemic environmental factors up to the first years of life.[22] MIH has no hypoplastic defects as there is no discernable reduction in enamel thickness. Any reduction in enamel thickness seen clinically is indicative of posteruption disintegration of enamel.[4]

Risk factors noted for MIH in the past were fever in the 1st year of birth, cyanosis, chicken pox, otitis media, ear infections, tonsillitis, urinary tract infections, GIT disorders, asthma, and allergies. Perinatal conditions such as infections during pregnancy, premature birth, birth complications, and family history of enamel defects could be a reason for MIH.[7,13,23] These conditions could probably cause alterations in the calcium phosphate balance or insufficient oxygen supply to ameloblasts resulting in enamel defects.[7,24] Conditions common in the first 3 years of life such as asthma, chicken pox, measles, and rubella are associated with MIH.[22] Other systemic illnesses associated with MIH are nutritional deficiencies, brain injury, cystic fibrosis, syndromes of epilepsy, dementia, and lead poisoning.

Premature delivery or preterm birth has been associated with increased prevalence of enamel defects in permanent dentition. The enamel defect severity increases with decreased gestational age and lower birth weight.[25] Hypoplasia in any form can predispose a child to develop MIH. Hypomineralization due to prolonged breastfeeding is due to the exposure to polychlorinated dibenzo-p-dioxins.[22]

Noncarious hypomineralized molars have underlying pulpal inflammation, as demonstrated by an increase in the pulpal innervation density and immune cell accumulation.[26] Studies conducted by Tobias *et al*

indicate the presence of bacteria in the dentinal tubules of the hypomineralized teeth. Since MIH is an enamel aberration which is seen at the time of eruption of the first molars, the dentinal tubules are still wide in which bacteria could easily penetrate.[27] The increased protein content present in the MIH enamel limits the access of acid to the hydroxyapatite crystallites. The high resistance of MIH enamel to acid etching is consistent with an increased organic content rather than carbonate substitution of the normal apatite lattice.[28] 22q11 deletion syndrome is found to be associated with MIH. These patients have many and complex medical problems including hypocalcemia and hypoparathyroidism.[29]

Enamel formation takes approximately 1000 days. Two-thirds of this time is devoted to the maturation stage of amelogenesis. The most critical period for enamel defects of the first permanent molars and incisors is during the 1st year of life coinciding with their early maturation. Ameloblasts are highly sensitive to various environmental disturbances during this period.[30] Patients with MIH-affected teeth suffer from dentine sensitivity to various thermal, mechanical, and osmochemical stimuli due to the porous nature of enamel sometimes, exposing the dentin. This can favor ingress of bacterial contaminants, thereby resulting in chronic inflammation of the pulp leading to a variety of morphological and cytochemical neuronal changes, with an overexpressed dentin sensitivity.[31] Microstructural analysis of sound and hypomineralization enamel shows marked changes which are less dense prism structure with loosely packed apatite crystals and wider sheath regions. These maybe responsible for the marked reduction in hardened and elastic modulus of the affected enamel.[32] The characteristic distinguishing feature of MIH from hypomaturation defects such as amelogenesis imperfecta or fluorosis is that these two contain high residual amelogenin protein. Pathogenically, this points to a preeruptive disturbance of mineralization involving overabundance of albumin, interfering with mineralization. An indicator of the severity of MIH-affected teeth is the actual organic content of its enamel whereas brown enamel, the most severe form of MIH, has the highest protein content, while the protein content of white/opaque and yellow enamel are both markedly higher than sound enamel.[33]

The management of MIH can be done by adhesive and fissure sealants for the molars, microabrasion and bleaching for the incisors, composite resin restorations, glass ionomer cement (GIC), resin-modified GIC, amalgam, preformed metal crowns, cast restorations, and extraction of severely affected teeth where restorations maybe impossible.[34,35]

Since this study was done using a very minimal sample size, further studies on larger populations are required to determine the complete prevalence.

Conclusion

MIH must be regarded as a public health problem which brings painful consequences, esthetic, and a negative impact on the quality of life of individuals suffering from it. It leads to a number of problems such as poor esthetics, food retention in the defective enamel areas, and higher sensitivity of the exposed dentin. The teeth with this condition can lead to rapidly progressing caries, and hence it is necessary for dentists to identify this condition at the earliest and treat appropriately. Early management of this condition helps in preventing first permanent molar morbidity and mortality so that remineralization as a preventive measure can be instituted. Application of sealants may be an effective approach to prevent carious lesions in MIH-affected first permanent molars. In all cases of MIH, it is essential to review the child on a regular basis to assure their long-term dental health.

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Conflicts of interest

There are no conflicts of interest.

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