ORIGINAL ARTICLE

Prevalence and Clinical Characteristics of Molar–Incisor Hypomineralization in 8–16-year-old Children in Industrial Town of Solan District of Himachal Pradesh

Hemlata Thakur[1], Avninder Kaur[2], Neetika Singh[3], Reetu Singh[4], Sanchit Kumar[5]

Abstract

Aim: The aim of the present study was to investigate the prevalence and etiology of molar–incisor hypomineralization (MIH) in 8- to 16-year-old children from town (Baddi) of Himachal Pradesh.

Materials and methods: A cross-sectional study was conducted with 2000 children from various schools of Baddi. Molar–incisor hypomineralization was diagnosed on the basis of EAPD 2003 criteria revised in 2009.

Results: Prevalence of MIH found was to be 2.9%. Demarcated opacities were predominant among all the defects. Postnatal illnesses were highly associated with MIH. The prevalence of dental caries reported in MIH patients was 82.7%.

Conclusion: Postnatal illnesses are significantly associated with MIH. Molar–incisor hypomineralization predisposes the tooth to dental caries.

Keywords: Demarcated opacities, Enamel defects, Molar–incisor hypomineralization.

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Introduction

Molar–incisor hypomineralization (MIH) was earliest documented by Swedish dentists in 1970.[1] Molar–incisor hypomineralization is defined as the defect that involves at least one of the four permanent molars that may or may not be associated with permanent incisors.[2] The term molar–incisor hypomineralization was introduced in 2001. Other terms used for MIH are "hypomineralized" PFMs, "idiopathic enamel hypomineralization," "dysmineralized" PFMs, "nonfluoride hypomineralization," and "cheese molars." Molar–incisor hypomineralization occurred due to disturbances in ameloblastic activity in transitional and maturational phase of amelogenesis.

Various etiological factors such as respiratory tract disorders, perinatal conditions, and dioxins via breastfeeding have been suggested for MIH. Other contributory factors are low level of oxygen, birth weight less than 2.5 kg, gastrointestinal tract disorders, diarrhea, high fever, and environmental conditions. MIH may be caused by reduced levels of calcium and phosphate. Calcium and phosphate reduction can occur because of nutritional deficiencies. Diseases that are common during early years of life are asthma, otitis media, tonsillitis, chicken pox, measles, and rubella which may cause MIH.[3,4] Molar–incisor hypomineralization may also be caused by prolonged breastfeeding due to presence of polychlorinated dibenzo-p-dioxins (PCDDs) in mother's milk.[3]

Regarding respiratory disorders such as pneumonia, chest infection, and asthma, these all have been reported to generate systemic metabolic stress linked to defects in the dental structure. Respiratory disorders lead to hypoventilation that results in metabolic acidosis and lower oxygen level. This results in pH change of enamel matrix that inhibits proteolytic enzymes and development of hydroxyapatite crystals leading to hypomineralization.[4,5]

There are variations in the MIH prevalence throughout the world, and it varies between 2.4% and 40.2% with normal child population.[6,7] Hence, the present study was aimed to find the prevalence, severity, and clinical characteristics of MIH and to determine the potential etiological factors of the same.

[1–5]Department of Pediatric and Preventive Dentistry, Bhojia Dental College and Hospital, Baddi, Nalagarh, Himachal Pradesh, India

Corresponding Author: Avninder Kaur, Department of Pediatric and Preventive Dentistry, Bhojia Dental College and Hospital, Baddi, Nalagarh, Himachal Pradesh, India, Phone: +91 9814319434, e-mail: avninder21@yahoo.com

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Materials and Methods

The present study was approved by the Institutional Ethical Committee of Bhojia Dental College and Hospital (Bhud) Baddi, Distt. Solan, Himachal Pradesh. Final sample of 2,000 children in the age range of 8–16 years was selected to evaluate the prevalence, clinical characteristics, and etiological factors of MIH. The children were selected by simple random sampling method from both Govt. and private schools of Baddi, Nalagarh Distt. Solan (HP).

A total of 15 schools were selected from the list of various schools in Baddi, and the purpose of this study was explained to the heads of the schools (Correspondent, Principal, Headmaster, Headmistress, etc.), and prior permission was obtained to conduct the study in their respective schools. Informed consent was obtained from the parent or legal guardian of each participant. The participants were made to sit on furniture chair facing away from direct sunlight, and a trained assistance recorded the observations on a specially designed proforma. Sterilized mouth mirror and probe were used for clinical

examination. The probe was used to remove plaque. The teeth were cleaned and examined wet to distinguish opacities from incipient carious lesions. The children with MIH were examined thoroughly for the defects and dental caries and questionnaire pertaining to etiology was filled by the examiner.

Molar–incisor hypomineralization was diagnosed based on EAPD 2003 criteria revised in 2009. The severity of hypomineralization was recorded according to the Wetzel and Reckel scale.

A self-prepared questionnaire containing 21 questions concerning the prenatal, perinatal, and postnatal history of the child to determine the possible risk factors involved in the etiology of MIH was filled by the examiner. Questionnaires were filled either by asking questions directly from the parents face to face or by telephonic conversation. All completed questionnaires and recording proforma were analyzed, and the data were transferred on the Microsoft Office Excel sheet. The data were subjected to SPSS, version 13, and statistically analyzed using Chi-square test.

Results

Two thousand children (1,033 males and 967 females) aged 8 to 16 years were examined. The data of 58 children diagnosed with MIH were statistically analyzed giving a prevalence rate of 2.9%. A higher prevalence of 1.65% was observed in males compared to 1.25% in females. Of a total of 291 affected teeth with MIH, 71.5% teeth had demarcated opacities, 19.6% had posteruptive enamel breakdown, 8.9% had atypical restorations, and only 0.34% were extracted due to MIH. Age-wise prevalence of MIH affected molars was evaluated which was statistically very highly significant (Table 1).

Of a total of 291 affected teeth with MIH, 208 (71.5%) teeth had demarcated opacities (Fig. 1), 57 (19.6%) teeth had posteruptive enamel breakdown (Fig. 2), 26 (8.9%) teeth had atypical restorations (Fig. 3), and only 1 (0.34%) tooth was extracted due to MIH (Fig. 4). Therefore, demarcated opacities were predominant among all the defects. Extraction due to MIH was least, that is, only 1 tooth was extracted due to MIH.

Table 1: Age-wise prevalence of MIH

Age (years)	*Total no. of children examined (n = 2,000)*	*Children with MIH*		*p value*
		n	*(%)*	
8	267	19	32.8	0.000*
9	269	10	17.2	
10	311	9	15.5	
11	215	6	10.3	
12	293	12	20.7	
13	209	2	3.4	
14	164	0	0	
15	153	0	0	
16	113	0	0	

*Very highly significant

Fig. 1: Demarcated opacities on mandibular permanent first molars

Mandibular teeth were significantly more affected with MIH than maxillary teeth with *p* value of 0.001. Out of 291 affected teeth in 58 children, 58.8% were molars and 41.2% were incisors. The mandibular molars (62%) were more affected than maxillary ones (38%), and the difference is statistically very highly significant difference with a *p* value of 0.000. Maxillary incisors (51.7%) were affected more than mandibular incisors (48.3%), and the difference was not statistically significant. In contrast, mandibular lateral incisors (66.6%) were more affected than maxillary lateral incisors (33.3%).

According to severity of MIH, out of 291 affected teeth, 206 teeth were presented with mild form of opacities. Moderate defects found were 20.6%, and severe defects were 8.6% only.

Pattern of MIH affected children based on the involvement of number of hypomineralized first permanent molars and incisors is given in Table 2. Out of 58 children with MIH, 44 were in the age range of 8–11 years. Only 13 children presented with HSPM (29.5%). Overall there were 176 teeth present in 44 children. Fifteen maxillary and 22 mandibular second primary molars were hypomineralized i.e., 8.5% and 12.5%, respectively. The prevalence was higher in mandible than in the maxilla and the difference was not statistically significant (0.19). Association of MIH with dental caries has been given in Table 3.

The analysis of the questionnaire-based interview indicated that no relevant medical history was found in 5.2% cases, and remaining 94.8% reported various medical conditions putatively associated with MIH. In these cases, there were either postnatal events alone or combinations of prenatal and postnatal and/or perinatal and postnatal. None of the patients reported with prenatal and perinatal events alone.

Amongst the prenatal health conditions a few were significantly more associated with the development of a hypomineralized defect i.e., pregnancy illnesses including hypotension-related anemia (34.5%), psychological stress (13.8%), 3rd trimester gestational ill health (27.7%) and ultrasounds taken more than three times during the last trimester (22.4%).

Amongst the perinatal health conditions presented a few were more significantly associated with MIH i.e., low birth weight (68.2%),

Fig. 2: Posteruptive enamel breakdown on mandibular right permanent first molars

Fig. 3: Atypical restoration on 46 and SSC on 36

Fig. 4: Extraction due to molar–incisor hypomineralization

Table 2: Pattern of MIH-affected children based on number of hypomineralized PFMs and incisors (I)

Teeth affected					
Molars with:	*One molar, n (%)*	*Two molars, n (%)*	*Three molars, n (%)*	*Four molars, n (%)*	*Total, n (%)*
Alone	1 (1.7)	4 (6.8)	0 (0)	5 (8.5)	10 (17)
+1 incisor	0 (0)	2 (3.4)	3 (5.1)	6 (10.2)	11(18.7)
+2 incisors	2 (3.4)	8 (13.6)	3 (5.1)	7 (11.9)	20 (34)
+3 incisors	1 (1.7)	2 (3.4)	0 (0)	2 (3.4)	5 (8.5)
+4 incisors	1 (1.7)	3 (5.1)	2 (3.4)	3 (5.1)	9 (15.3)
+5 incisors	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
+6 incisors	0 (0)	0 (0)	0 (0)	1 (1.7)	1 (1.7)
+7 incisors	0 (0)	0 (0)	0 (0)	1 (1.7)	1 (1.7)
+8 incisors	0 (0)	0 (0)	0 (0)	1 (1.7)	1 (1.7)
Total	5 (8.5)	19 (32.3)	8 (13.6)	26 (44.2)	58 (100)

Table 3: Association of dental caries with MIH

Frequency of children (%)	*DMF score (n = 58)*	*Total DMF score*	*Mean DMF score ± SD*
10 (17.2)	0	53	0.91± 2.4
7 (12.1)	1		
13 (22.4)	2		
8 (13.8)	3		
10 (17.2)	4		
3 (5.2)	6		
2 (3.4)	7		
1 (1.7)	8		
1 (1.7)	10		
2 (3.4)	12		

preterm birth (29.3%), neonatal jaundice (37.9%), and respiratory distress (18.9%).

Among the postnatal health conditions and other general variables, the chance of MIH was significantly more likely to happen with following acute health illnesses: unexplained high fever (34.4%), chest infections other than pneumonia (31.2%), pneumonia (17.2%), and tonsillitis (6.8%). Exposure of antibiotics during the first year of life increased the probability of defect occurrence more than three times compared to non-use (94.8%). Amongst other general risk factors, length of breastfeeding was of significant importance. Children breastfed up to the first 6 months (8.65%) or up to the first year of life (62.1%) were highly more likely to have MIH compared to children who had been breastfed constantly for >2 years that found to have a protective role against defect occurrence (20.7%).

Discussion

The present study assessed the prevalence, etiology and clinical features of MIH and its association with dental caries in 8–16 years old children. Molar–incisor hypomineralization is a qualitative defect i.e., defects during maturation stage of amelogenesis. In the present study, a lower prevalence of MIH (2.9%) was observed in the Himachal population. A higher prevalence was observed in Karnataka,[8] Gujarat,[9] Udaipur[1] i.e., 8.9%, 9.4% and 9.2%, respectively. This disparity in prevalence in different regions of India might be due to population examined or sample selected or the diagnostic criteria used. Enamel demineralization due to other reasons like dental caries may conceal the actual prevalence of MIH. In case of older children occlusal wear due to mastication and restorations of carious teeth may lead to diagnostic error.[3,10–12]

The gender predilection was higher with respect to males as compared to females i.e., 1.3:1. Similar to the findings that were seen in Jordanian[13] (1.05:1), Udaipur[1] (1.3:1), Gujarati populations[9] (1.2:1).Whereas in other studies like Karnataka and Brazil, females were affected more compared to males.

Demarcated opacities found in MIH cases are variable in degree with clear demarcation from the adjacent enamel. Demarcated opacities (71.5%) were the frequent finding of MIH in the present study, which is in accordance with previous studies such as Brazilian

children[14] (73.1%) and Iraqi children[15] (76.4%). The prevalence of post eruptive enamel breakdown was seen higher in the study (19.6%) than reported in Sweden[16] (2.5%) and Libya[17] (1.9%,). The present study showed a higher percentage of demarcated opacities which are easy to diagnose in direct vision and well illumination.

The mandibular teeth were more frequently affected with MIH than the maxillary teeth with a very highly significant difference ($p = 0.001$). This was similar with other studies like Leppäniemi et al.,[18] Lygidakis et al.[19] and Chawla et al.[20] The reason might be its better accessibility and visibility to diagnose the case. Mandibular teeth are easy to examine in direct vision. Since maxillary molars are examined in indirect vision. This could be the reason for lower prevalence in maxillary teeth.

Frequency of MIH was more as compared to MH alone i.e., 82.8% and 17.2%, respectively (Table 2). Similar pattern was found in the studies conducted in Gujarat[7] (MIH 72.6% and MH 22.1%), and Spain[21] (MIH 81.6% and MH 19.4%). Contrary to the findings of the present study, higher prevalence of MH was found as compared to MIH i.e., Udaipur[1] (MIH 29% and MH 71%), Jordan[13] (MIH 32 % and MH 68 %).

Different possible combinations and clinical features were observed in the present study e.g. involvement of only one molar or all molar with or without involvement of incisors (Table 2). The most common pattern observed was the combination of two molars and two incisors (13.6%). The findings are similar in other studies on Lithuanian,[22] Greek,[19] Brazilian,[23] Jordanian[13] and Gujarati[9] Indian population. Hence, it indicates the versatility in clinical characteristics.

The severity of the MIH was also evaluated as per Wetzel and Reckel scale. Most of the defects belonged to mild form of MIH (70.8%). These findings are consistent with the study done by Preusser et al.[24]

In the present study, no significant association was found between MIH and hypomineralized second primary molar (HSPM). This was according to the study done by Costa-Silva et al.[25]

The teeth with MIH are more susceptible to caries because of more plaque accumulation and fast carious progression in a defectively mineralized tooth. The present study also investigated the correlation of dental caries with MIH, and it was found to be statistically nonsignificant (Table 3).

The involvement of the teeth calcified almost at the same age indicates its systemic origin. Calcification of first permanent molar starts before birth. Therefore, prenatal, perinatal, and postnatal history is important to correlate the defect with possible etiological factors. Till date, no conclusive etiology is found, rather combinations of factors may lead to development of MIH. A questionnaire was filled relevant to etiology of MIH. Some cases of MIH were related to maternal illness in the prenatal phase compared to perinatal and postnatal period. In prenatal health conditions, hypotension-related anemia, psychological stress, third trimester ill health, and number of ultra sounds taken did not show any statistically significant results. This was in accordance with other studies such as Kusku et al.[26] and Lygidakis et al.[19]

No statistically significant association was found between MIH and factors such as gestational age of baby at birth and maternal age at child's birth. This was in accordance with other studies such as Jalevik et al.,[16] Beentjes et al.,[27] Whatling and Fearne[28] who were also unable to report the specific etiology of MIH. In the present study, a low birth weight was found in 68.2% children which is significantly associated with MIH. Similar findings were observed in the other studies such as Johnsen et al. 1984 and Seow et al. 1987.[29,30] It has been observed that low birth weight is associated with low level of serum calcium, phosphorus, and oxygen due to incompletely developed lungs. This can lead to reduced oxygen supply for the ameloblasts during active amelogenesis and thus can result in defective enamel. However, neonatal complications such as jaundice (37%) and respiratory distress (18.9%) have also shown an association with development of MIH.

Postnatal illness in children does affect the mineralization of enamel during infancy as explained by Tunk et al.[31] Their study showed disorientation of enamel prisms and crystal-free area due to persistent high-grade fever. A persistent high fever of up to 38.5°C temporarily impairs ameloblastic activity during initial calcification stage. Kreshover[32] stated that a high-grade fever may lead to devastating effect on amelogenesis, ranging from enamel hypoplasia, hypomineralization, extending to complete cellular degeneration. The present study showed a significant correlation between MIH and postnatal illness. Consistent with the earlier studies, acute health illness such as unexplained fever (34.4%), chest infections other than pneumonia (31.2%), and pneumonia (17.2%) were the most common reasons for MIH.

The frequency and duration of antibiotics had strong correlation with the development of MIH (94.8%). The antibiotic therapy during the first three years of birth may alter the secretary ability of the ameloblasts. Whether the defect is due to antibiotics or the disease involved is difficult to explain. A study by Hong et al.[33] also found a correlation between amoxicillin use during infancy and fluoride-like defects in permanent molar and incisors. Laisi et al.[34] showed that amoxicillin used in early childhood may increase the risk of MIH.

Molar–incisor hypomineralization has also been found to be associated with exposure to the polychlorinated dibenzo-p-dioxins (PCBs) during early childhood via breastfeeding. The PCBs are environmental pollutants also known as polyhalogenated aromatic hydrocarbans. In the present study, prevalence of mineralization defects following breastfeeding for up to the first 6–12 months after birth was high, indicating that early weaning could be one of the risk factor for mineralization defects. Early weaning, as explained by the World Health Organization, compromises the child's nutritional status could be a risk factor for developmental defect of enamel. This was in agreement with the findings of Alaluusua et al.[35] and Fagrell et al.[36] who showed that breastfeeding for more than 6 months significantly increased the possibility of defect development. There is, however, disagreement with some findings such as Jälevik B, Norén,[37] Beentjes et al.[27] and Whatling and Fearne,[28] where the length of breastfeeding was not found a potential causative factor for hypomineralized first permanent molar. The breastfeeding for a longer period (>2 years) gives protection against the defect occurrence.

Contradictory to the previous findings, the present study showed no relation to birth order, as most of children were first or second born in the family.

To conclude, MIH is an important health issue that predisposes the teeth to dental caries. No conclusive health issues can predict the chances of MIH in a child. Maternal health as well as perinatal and postnatal illnesses can increase the chances of developmental defects in enamel.

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