

Molar Incisor Hypomineralization: A Study of Prevalence and Etiology in a Group of Iranian Children

Rahil Ahmadi^{1*}, DDS, MSc; Nahid Ramezani^{2,3}, DDS, MSc; Rahmatollah Nourinasab², DDS

1. Pediatric Dentistry Department, Dental School, Shahed University, Tehran, Iran
2. Pediatric Dentistry Department, Dental School, Zahedan University of Medical Sciences, Zahedan, Iran
3. Children And Adolescents Health Research Center, Zahedan University of Medical Sciences, Zahedan, Iran

Received: May 13, 2011; Final Revision: Jan 08, 2012; Accepted: Jan 27, 2012

Abstract

Objective: The aim of this study was to investigate the prevalence of molar incisor hypomineralization (MIH) and its relationship with systemic conditions in a group of Iranian children.

Methods: The study population comprised of 433 7-9 year olds, from four schools in Zahedan, Iran. Subjects were evaluated clinically by one examiner, and at a separate session, their mothers completed a coded medical history questionnaire. Hypo-mineralized molars and incisors were recorded based on DDE (developmental defects of enamel) index and DMFT (number of decayed, filled and missing teeth) was determined. Statistical analyses were performed using Chi-square and independent sample t-tests.

Findings: Fifty-five (12.7%) children showed MIH. The overall mean number of affected teeth was 0.2. The mean value of DMFT in MIH children was greater than in normal children. Demarcated opacities were the most frequent (76%) enamel defect. Mother's and child's medical problems during prenatal, perinatal and post natal period were significantly remarkable in MIH children.

Conclusion: The prevalence of MIH in a group of Iranian children was 12.7%. Prenatal, perinatal and post natal medical conditions were more prevalent in children affected by MIH.

Iranian Journal of Pediatrics, Volume 22 (Number 2), June 2012, Pages: 245-251

Key Words: Developmental enamel defects; Molar incisor hypomineralization; Hypoplasia

Introduction

Enamel hypomineralization in the permanent first molars (PFMs) and incisors was first described in 1970^[1]. Since then different diagnostic terms have been used to define this developmentally-derived enamel defect. Some of these definitions are linked to the clinical characterization of enamel, such as cheese molars and some are describing its etiology as non-fluoride hypo-mineralization^[2,3]. In 2001 the term molar incisor hypomineralization (MIH)

was recommended for PFMs with white or yellow-brown discoloration, which is frequently associated with hypomineralized permanent incisors^[4,5].

Due to soft and porous enamel of teeth affected with MIH, unusual cavitation and enamel disintegration on the occlusal surface may occur. This defective enamel structure may cause hypersensitivity, secondary caries, atypical restorative treatments, loss of fillings and, in severe cases, extraction of the affected teeth^[6,7].

* Corresponding Author;

Address: Department of Pediatric Dentistry, Dental School, Shahed University, Italia st, Vesal Ave, Tehran, Iran

E-mail: rahilsh@yahoo.com

© 2012 by Pediatrics Center of Excellence, Children's Medical Center, Tehran University of Medical Sciences, All rights reserved.

Despite the higher treatment demands, restorative treatments for these teeth are challenging for both patient and dentist^[8]. Children suffering from MIH often demonstrate behavior management problems and dental fear because of the difficulties in achieving adequate anesthesia. Moreover, the altered and porous enamel structure makes bonding risky, leading to defective fillings and frequent re-treatments^[8,9]. Because of these severe clinical problems occurring with MIH, knowledge about its occurrence and etiological factors are essential.

A wide range of prevalence (4-25%) has been reported for MIH by various investigators in different countries^[7]. However, most of these studies were conducted in European countries^[1,2,10,11]. To our knowledge, at the time of this study no report on the prevalence of MIH in Iran has been published.

Every systemic physiological stress within the prenatal period and the first 3 years of life may disrupt the ameloblasts' activity during secretory and/or maturation phase. Since the formed enamel does not re-model, environmental disturbances may leave detectable defects in the mature enamel^[5,7,11,12]. Harmful medical or environmental conditions may affect enamel formation. Von Amerongen stated that any factor influencing the oxygen supply of ameloblasts may affect the enamel mineralization^[13]. Fever, chicken pox, ear infection, birth prematurity, the mode of delivery, and environmental toxins are mentioned as possible causal factors for MIH in different studies^[7,14]. However, doubt exists about the exact relationship of common medical and environmental conditions and the incidence of MIH^[14,15].

Thus, the aim of this study was to investigate the prevalence of MIH and its relationship with systemic conditions in a group of Iranian children.

Subjects and Methods

Four-hundred and fifty-seven first to third graders, from 4 different elementary schools distributed in 2 districts of Zahedan, were invited

to participate in our study. Of those invited, 433 children (7-9 years) participated with informed parental consent. They were all born and lived in Zahedan, a large city in eastern Iran. To minimize the effect of different socioeconomic status, the schools were chosen such that 2 were public and 2 private schools.

All subjects used municipal water supply and the local government measurements showed an optimal fluoride level in their drinking water (0.7-1 ppm). Children with generalized developmental enamel defects such as amelogenesis imperfecta, not fully erupted molars and incisors and those suffering from a chronic disease were excluded. Each child was given a code to avoid bias.

Dental examination:

The examinations were performed by one calibrated pediatric dentist. After theoretical training for MIH, 30 photographs of 18 patients with MIH and 12 cases with other enamel defects were used to calibrate the examiner. All photographs were diagnosed correctly on the first day and 4 weeks later. The examiner was prepared for DMFT evaluations according to WHO guidelines for oral health surveys^[16]. The reproducibility for DMFT assessments was evaluated by examination and re-examination of 10% of the subjects in a 4 weeks interval. Dental examinations were performed in schools' medical room using a mirror, under day light supported by a head light. Teeth were not dried. When necessary, gauze was used to remove food accumulations.

DDE index was implemented to classify the affected teeth. This index included information about color, type and extent of the enamel defects, which consisted of 3 parts: demarcated opacities, diffuse opacities and hypoplasia. Demarcated opacities were defined as opacities with different colors and distinct margins to the adjacent enamel. Whereas diffuse opacities were alterations in enamel translucency without any clear boundary with adjacent enamel, and may be observed in 3 patterns: linear, patchy and confluent. Teeth with reduced enamel thickness demonstrated enamel hypoplasia.

At the end of the examination, caries were assessed using the DMFT index under day light in

accordance with the World Health Organization caries diagnostic criteria for epidemiological investigations. The DMFT index was recorded for each child in a dental charting.

Questionnaire:

A detailed coded questionnaire comprised of 40 questions was designed to investigate mother's medical history during pregnancy and child's medical condition within the first 3 years of his/her life and perinatal period, family history of enamel defects and duration of breast feeding. The part of medical history of the questionnaire contained questions on the

prinatal and postnatal periods as follow:

Prenatal period: infections or illnesses of mother during pregnancy, hypocalcaemia and Vitamin D deficiency, hypertension, gestational diabetes, pre-eclampsia. Prinatal period: complications during birth, premature birth, type of delivery. Postnatal period: childhood illnesses and infections: Otitis media, asthma, urinary tract infection, chicken pox, rubella, tonsillitis, high fever, allergies, antibiotic usage, renal failure, cardiac problems, epilepsy.

In order to evaluate the reliability of the questionnaire, a pilot study has been performed. Forty five mothers were randomly asked to complete the questionnaire twice, with a 2 weeks interval. The test-retest repeatability of the questionnaire was measured by kappa coefficient, and our results showed an excellent agreement between the test-retest responses and a weighted kappa of 0.93.

The validity of the questionnaire was assessed by an expert committee consisting of 2 pediatric dentists, 2 pediatricians, 1 oral pathologist and 1 specialist dentist in dental public health and their responses were used to improve the questionnaire

precision and clarity and to decrease the ambiguity.

Statistical analysis:

Chi-square and independent sample t-tests were used for testing the differences between 2 groups of affected and unaffected children. The probability value of less than 0.05 was considered as significant.

Findings

The sample comprised of 215 (49.7%) boys and 218 (50.3 %) girls. Of the 433 pupils examined, 55 (12.7%) children exhibited MIH (Table 1). However, the Chi-square statistics did not reveal any significant differences between boys (14%) and girls (11.5%) ($P=0.4$).

The overall mean number of affected teeth was 0.21 (SD=0.09), while the mean number of affected teeth per affected child was 2.1 (SD=0.8). The mean value of DMFT in affected children (1.46, SD=0.99) was higher than normal children (0.76, SD=1.33; $P<0.001$). Demarcated opacities were observed in 67.5% first permanent molars and incisors, whereas diffused opacities and hypoplasia were present in 25.6% and 6.9% of teeth with MIH, respectively (Table 2).

As it is shown in Tables 3 and 4, mother's and child's medical histories during the prenatal, perinatal and postnatal periods were significantly remarkable in MIH children compared to unaffected children ($P<0.001$). Postnatal factors such as renal failure, chicken pox, asthma and allergic reactions and the use of amoxicillin were higher in MIH affected children than in normal children. Children who were longer breastfed

Table 1: Molar incisor hypomineralization distribution according to gender*

Gender	Affected n (%)	Unaffected n (%)	Total n (%)
Female	25 (11.5)	193 (88.5)	218 (100)
Male	30 (14)	185 (86)	215 (100)
Total	55 (12.7)	378 (87.3)	433 (100)

P. value= 0.4

Table 2: Prevalence of various enamel defects in molar incisor hypomineralization affected dentitions

Enamel developmental defects	Tooth type				Total n (%)
	Upper incisors	Lower incisors	Upper molars	Lower molars	
Demarcated opacities	16	8	28	27	79 (67.5)
Diffuse opacities	6	0	13	11	30 (25.6)
Hypoplasia	1	0	3	4	8 (6.9)
Total	23	8	44	42	117 (100)

demonstrated MIH more commonly ($P=0.005$) than those who were breastfed for a short period. Family history of enamel defects was not significant in MIH children compared to unaffected children ($P=0.56$).

Discussion

The present study showed a prevalence of 12.7% for MIH in a group of Iranian children. This was greater than that observed in Chinese children^[11], and comparable to the data obtained from two investigations in Italy and Bosnia and Herzegovina^[17,18], and lower than that reported for Swedish and Danish children^[10,19]. These variations may be partly due to differences in the ethnicity and age groups of the samples studied.

We found no significant difference in the number of girls and boys with MIH, which is comparable with the findings of some other studies^[3,10,11]. We report that the prevalence and severity of enamel defects in molars were more than those found in incisors. This was in accordance with other studies^[5,11]. Our results

showed that 6 molars had hypoplastic lesions, while only 1 incisor was hypoplastic. The mean number of teeth with MIH per affected child was 2.1 in our study, lower than that observed in a study of Chinese children, which reported 2.6 teeth per child with MIH^[11].

The DMFT value in affected children was higher than in unaffected pupils. This difference was expected because of the abnormal enamel structure, which may lead to rapid caries formation and progression, more complicated treatments and defective composite-enamel bonding, resulting in a higher DMFT^[5,7,20].

In this study, MIH was significantly more observed among children who had prenatal, perinatal and postnatal medical problems. Mothers of affected children experienced more diseases during pregnancy compared to unaffected subjects ($P<0.001$). This was in agreement with some other preceding studies, which reported a higher prevalence of MIH in children whose mothers suffered from different ailments during their prenatal period^[21,22,23]. The duration of breast feeding was significantly longer in MIH children compared to normal children; however, all of the subjects in both groups were breast fed. This was in line with the findings of

Table3: Distribution of pre natal and perinatal variables in molar incisor hypomineralization affected and unaffected children

	Variables		Affected n (%)	Unaffected n (%)	Total n (%)	P value
Prenatal	Problems during pregnancy	yes	11 (58)	8 (42)	19 (100)	<0.001
		No	44 (10.7)	370 (89.3)	414 (100)	
Perinatal	Birth delivery	normal	18 (6)	286 (94)	304 (100)	<0.001
		cesarean	26 (23)	88 (77)	114 (100)	
	Birth complications		5 (100)	0	5 (100)	
			6 (60)	4 (40)	10 (100)	

Table 4: Distribution of postnatal variables in molar incisor hypomineralization affected and unaffected children

Variables		Affected n (%)	Unaffected n (%)	Total n (%)	P value
Ear infection	yes	4(36.4)	7(63.6)	11	0.017
	no	51(12.1)	371(87.9)	422	
Urine tract infection	yes	3(27.3)	8(72.8)	11	0.14
	no	52(12.3)	370(87.7)	422	
Fever illness	yes	2(25)	6(75)	8	0.29
	no	53(12.5)	372(87.5)	425	
Chicken pox	yes	7(36.8)	12(63.2)	19	0.001
	no	48(11.6)	366(88.4)	414	
Renal failure	yes	9(100)	0(0)	9	<0.001
	no	46(10.8)	378(89.2)	424	
Allergies	yes	4(40)	6(60)	10	0.009
	no	51(12.1)	372(87.9)	423	
Asthma	yes	7(77.8)	2(22.2)	9	<0.001
	no	48(11.3)	376(88.7)	424	
Amoxicillin usage	yes	8(50)	8(50)	16	<0.001
	no	47(11.3)	370(88.7)	417	
Average breast feeding period	0-12 months	7(5)	115(95)	122	0.005
	12-18 months	35(13.8)	218(86.2)	253	
	>18 months	13(22.5)	45(77.5)	58	

Alaluusua et al^[24]. On the contrary, Whalting found no association between the groups regarding breast feeding and its duration and MIH^[14]. This finding may be supported by the fact that some environmental threatening factors such as mother's Vitamin D deficiency or malnourishment may be transferred to the breastfed child. These deficiencies may lead to the MIH appearance with no clinical or skeletal signs of rickets^[12]. Illnesses during early childhood period were more common in MIH children ($P<0.001$). Among all the questioned ailments, chicken pox, renal failure, asthma and allergic reactions were more common in affected children, which was in accordance with the findings of some previous investigations^[12,14,25]. It has been suggested that any factor, which may cause oxygen deficit in active ameloblasts may be responsible for MIH development. Respiratory diseases occurring within the first years of life may affect tooth formation by oxygen deficiency^[15]. Chicken pox is caused by *Varicella zoster*, which infects the epithelial surfaces. It is probable that this virus spreads to epithelial derived ameloblasts during the enamel maturation phase and affects their activity^[14,15]. Antibiotic therapy, particularly

amoxicillin, during the first 3 years of child's life was significantly more observed in MIH group. This may be because of direct effect of amoxicillin on active ameloblasts or may be attributed to the infectious disease that antibiotic was prescribed to alleviate the illness^[14,25,26].

In our study we attempted to minimize the probable bias. The examiner was calibrated using photographs. This calibration method in studying enamel defects was confirmed and utilized by Soviero et al and Sabieha et al^[5,27]. Extensively dried enamel may result in overestimated data^[5,28]. To ensure appropriate accuracy, the moisture was controlled with gauze and the index teeth remained wet^[10]. Children of 7-9 years were selected for this study since caries or attrition may mask the enamel defects in older children. In addition, at 7-9 years the first permanent molars and incisors are usually erupted^[10,12]. The patients' criteria used in this study have been confirmed and recommended by EAPD in 2003^[29]. Although several studies have stated that the fluoride is not associated with the etiology of MIH^[11,30,31], however, in our study all participating children resided in areas with optimal fluoride level in their drinking water and no one had

received systemic fluoride supplements.

The present study revealed that different medical and environmental factors may be associated with MIH development. This is logical since enamel formation is a long and sensitive procedure, which may be affected by many environmental insults. In addition, various childhood diseases commonly happen in young children. Thus it is unlikely that a single factor would be responsible as an etiological cause^[14]. Presence of a relationship between child's medical history and defective enamel formation puts emphasis on the role of pediatricians in initial diagnosis of MIH. Since children with health problems firstly seek medical treatments rather than their dental needs, pediatricians are of a great help in informing parents about possible dental defects and referring them to a dentist for current or future dental desires.

Some limitations were noted in this study: the information on the etiological factors was based on the memories of mothers interviewed, which may be affected by recall bias. To minimize this type of bias, an extensive questioning was performed and mothers were not informed about the possible etiological effects of the questioned disease on the MIH formation. However, despite our attempt to reduce the bias, the acquired data will not completely reflect the child's medical history in his/her first years of life. Obtaining the child's health data from medical documents recorded by a health center, will help to access to more accurate and precise information, yet this will be difficult if not impossible to achieve for all subjects in a retrospective study and illustrates the need for prospective evaluations. This assessment has been performed on a group of 433 children and we found a relationship between some of the investigated factors and MIH. Nevertheless, this study provided baseline data in an Iranian population and authors suggest further prospective longitudinal observational investigations on larger populations to declare the exact factors responsible for this type of enamel defect. We found a prevalence of 12.7% in our study which means this enamel defect may serve as a frequent public health problem and its prevention, diagnosis and treatment needs special consideration.

Conclusion

Within the limitation of this cross-sectional study, these conclusions may be drawn:

1. The prevalence of MIH in a group of Iranian children was 12.7%.
2. The DMFT value was higher in MIH children compared to the normal children.

Prenatal, perinatal and postnatal medical conditions were more prevalent in children affected by MIH in current study. Thus, pediatricians play an important role in initial diagnosis of MIH and informing the parents about this type of enamel defects.

Acknowledgment

This study was approved by the Ethics Committee for Research of the Zahedan University of Medical Sciences (Ref: 90-1001).

Conflict of Interest: None

References

1. Koch G, Hallonsten AL, Ludvigsson N, et al. Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children. *Community Dent Oral Epidemiol* 1987;15(5):279-85.
2. Weerheijm KL, Groen HJ, Beentjes VE, Poorterman JH. Prevalence of cheeses molars in eleven year old Dutch children. *ASDC J Dent Child* 2001;68(4):259-62, 229.
3. Leppäniemi A, Lukinmaa PL, Alaluusua S. Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. *Caries Res* 2001;35(1):36-40.
4. Weerheijm KL, Jälevik B, Alaluusua S. Molar-incisor hypomineralisation. *Caries Res* 2001; 35(5):390-1.
5. Soviero V, Haubek D, Trindade C, et al. Prevalence and distribution of demarcated opacities and their sequelae in permanent 1st molars and incisors in 7 to 13-year-old Brazilian children. *Acta Odontol Scand* 2009;67(3):170-5.

6. Fagrell TG, Lingström P, Olsson S, et al. Bacterial invasion of dentinal tubules beneath apparently intact but hypomineralized enamel in molar teeth with molar incisor hypomineralization. *Int J Paediatr Dent* 2008;18(5):333-40.
7. Chawla N, Messer LB, Silva M. Clinical studies on molar-incisor-hypomineralisation part 2: development of a severity index. *Eur Arch Paediatr Dent* 2008;9(4):191-9.
8. 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(1):24-32.
9. Kotsanos N, Kaklamanos EG, Arapostathis K. Treatment management of first permanent molars in children with Molar-Incisor Hypomineralisation. *Eur J Paediatr Dent* 2005;6(4):179-84.
10. Jälevik B, Klingberg G, Barregård L, et al. The prevalence of demarcated opacities in permanent first molars in a group of Swedish children. *Acta Odontol Scand* 2001;59(5):255-60.
11. Cho SY, Ki Y, Chu V. Molar incisor hypomineralization in Hong Kong Chinese children. *Int J Paediatr Dent* 2008;18(5):348-52.
12. Ogden AR, Pinhasi R, White WJ. Nothing new under the heavens: MIH in the past? *Eur Arch Paediatr Dent* 2008;9(4):166-71.
13. 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(4):266-9.
14. Whatling R, Fearne JM. Molar incisor hypomineralization: a study of aetiological factors in a group of UK children. *Int J Paediatr Dent* 2008;18(3):155-62.
15. Alaluusua S. Etiology of molar incisor Hypomineralization: A systematic review. *Eur Arch Paediatr Dent* 2010;11(2):53-8.
16. World Health Organization. Individual tooth status and treatment need. In: Oral Health Surveys: Basic Methods, 3rd ed. Geneva, Switzerland. World Health Organization. 1987; Pp: 34-39.
17. Calderara PC, Gerthoux PM, Mocarrelli P, et al. The prevalence of Molar Incisor Hypomineralisation (MIH) in a group of Italian school children. *Eur J Paediatr Dent* 2005;6(2):79-83.
18. Muratbegovic A, Markovic N, Ganibegovic Selimovic M. Molar incisor hypomineralization in Bosnia and Herzegovina: etiology and clinical consequences in medium caries activity population. *Eur Arch Paediatr Dent* 2007;8(4):189-94.
19. Wogelius P, Haubek D, Poulsen S. Prevalence and distribution of demarcated opacities in permanent 1st molars and incisors in 6 to 8-year-old Danish children. *Acta Odontol Scand* 2008;66(1):58-64.
20. Lygidakis NA. Treatment modalities in children with teeth affected by molar-incisor enamel hypomineralisation (MIH): A systematic review. *Eur Arch Paediatr Dent* 2010;11(2):65-74.
21. 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(4):230-4.
22. Chawla N, Messer LB, Silva M. Clinical studies on molar-incisor-hypomineralisation part 1: distribution and putative associations. *Eur Arch Paediatr Dent* 2008;9(4):180-90.
23. Lygidakis NA, Dimou G, Marinou D. Molar-incisor-hypomineralisation (MIH). A retrospective clinical study in Greek children. II. Possible medical aetiological factors. *Eur Arch Paediatr Dent* 2008;9(4):207-17.
24. Alaluusua S, Lukinmaa PL, Koskimies M, et al. Developmental dental defects associated with long breast feeding. *Eur J Oral Sci* 1996;104(5-6):493-7.
25. Jälevik B, Odelius H, Dietz W, Norén J. Secondary ion mass spectrometry and x-ray analysis of hypomineralized enamel in human permanent first molars. *Arch Oral Biol* 2001;46(3):239-47.
26. Lai S, Ess A, Sahlberg C, et al. Amoxicillin may cause Molar incisor Hypomineralization. *J Dent Res* 2009;88(2):132-6.
27. Sabieha AM, Rock WP. A comparison of clinical and photographic scoring using the TF and modified DDE indices. *Community Dent Health* 1998;15(2):82-7.
28. Jälevik B. Prevalence and diagnosis of molar incisor hypomineralization (MIH): A systematic review. *Eur Arch Paediatr Dent* 2010;11(2):59-64.
29. Weerheijm KL, Duggal M, Mejäre I, et al. Judgment criteria for molar incisor hypomineralization (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. *Eur J Paediatr Dent* 2003;4(3):110-3.
30. Balmer RC, Laskey D, Mahoney E, et al. Prevalence of enamel defects and MIH in non-fluoridated and fluoridated communities. *Eur J Paediatr Dent* 2005;6(4):209-12.
31. Balmer R, Toumba J, Godson J, et al. The prevalence of molar incisor hypomineralisation in Northern England and its relationship to socioeconomic status and water fluoridation. *Int J Paediatr Dent* 2011. doi: 10.1111/j.1365-263X.2011.01189.x [Epub ahead of print]