

Could Molar-Incisor Hypomineralization (MIH) Existence be Predictor of Short Stature?

Abstract

Background: Molar--incisor hypomineralization (MIH) could be appeared in condition of calcium (Ca^{2+}) disorders. Body height is an index of growth health monitoring in child that may be assumed by calcium metabolism. This study was designed to compare the body height of 8--9 years old schoolchildren with MIH and control group. **Methods:** This cross-sectional study was carried out by examination of 606 Iranian healthy schoolchildren for recording enamel defects and body height measurements by a single trained examiner. Putative etiological factors were evaluated using the structured questionnaire. The questionnaire was about maternal, prenatal, and postnatal factors. Statically analysis was done using *t*-test and Chi-square test in SPSS 22. **Results:** The prevalence of MIH in the schoolchildren was 52.9%. Prevalence of MIH significantly was higher in girls. Most of maternal and child's parameters appeared to have no significant correlation with MIH except birth weight, antibiotic therapy, maternal disease in pregnancy, and medication ($P < 0.05$). **Conclusions:** In spite of lower body height in schoolchildren with MIH, there was not any significant correlation between them.

Keywords: Body height, dental enamel hypoplasia, incisor, Iran, prevalence

Introduction

During the process of odontogenesis, each alteration resulting from diverse disturbances is named as developmental defect of enamel (DDE). It may be seen with or without other hard tissue dysplasia.^[1] Disturbances in hard tissue matrix and mineralization could lead to enamel hypoplasia (EH) or enamel opacities (EO). EH according to the quality of mineralization classified to EO with two subgroups, demarcated or diffused.^[2,3]

In spite of normal mineralization of matrix, thickness of enamel is reduced in EH. In EO some parts of enamel matrix fail to mineralize completely and appear as demarcated or diffused opacities.^[4]

Prevalence of DDE in developed countries has been reported in range of 9-63% in permanent teeth.^[2]

The most common involved teeth maxillary was central and first molar, respectively.^[5] Nowadays MIH is a hot topic in pediatric dentistry. Dental enamel would be formed in three interrelated phases. In addition to

genetic factors, ameloblasts are sensitive to environmental changes like temperature rise, PH changes, and hypocalcemia.^[5-9] Also some medical problems (parental, postnatal) have been implicated.^[7,10,11] Two-thirds of stress lead to EH occurring in first year of life.^[12]

Other than esthetic aspect, susceptibility to caries, bacterial plaque accumulation, occlusal problems, and tooth sensitivity are the main side effects of MIH.^[13]

EH provides an inevitable marker of physiologic stress during parental and childhood periods.^[14]

Scientist believe that stature is an excellent measure of conditions in childhood. It is the outcome of complex interaction between genetic, socioeconomic conditions, and environmental factors. Although genetic analysis have shown hundreds of genes are involved in stature control but its expression is influenced upon some factors during growth and development.^[15]

This study was designed to compare the body height of 8--9 years old children with MIH and control group.

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Methods

Study design and participants

A randomized cluster sample of 606 children between 8 and 9 years old, who had their first permanent molars and incisors, were evaluated. The date of ethical committee is 1394(2015). The students with history of serious illness or chronic medical conditions such as diabetes mellitus, seizure or asthma, syndromic disorder, or taking unusual drugs like corticosteroids or cytotoxic were excluded from the study.

Study instruments and assessment of variables

Patient consent form was completed with the questionnaire as a part of it. Information regarding demographic data, medical history, pregnancy, birth weight, duration of breastfeeding and antibiotic therapy was obtained through a structured questionnaire. This study was carried out in two steps. A cross-sectional study was initially conducted to estimate the frequency of MIH in 8--9 years old students of both sexes. Clinical criteria according to Weerheijm's study were used to diagnose MIH, a demarcated defect composing the different enamel translucency. The involved enamel has normal thickness with a smooth surface and may appear white, yellow, or brown.^[16,17] In second phase, a case-control study was performed to compare the average of body heights of students with MIH and control group.

Dental examinations were conducted by a single trained last term students of dentistry. She was trained in use of MIH through color photographs. Only permanent teeth were selected for observation, clinical examinations were done after brushing and drying teeth with a sterile gauze using artificial light. Body heights of students were measured by standing height index.

Statistical analysis

Descriptive and analytical statistics were computed by the software SPSS version 22. Initially, descriptive statistics were calculated for the characteristics of the subjects, frequency and percentages for categorical variables, mean, SD, and percentages.

The Kolmogorov–Smirnov test was used to analyze the normality of data. Using Chi-square test categorical variables were compared. Furthermore, quantitative variables were compared through *t*-test. A probability value of $P < 0.05$ was considered statistically significant.

Results

There were 12 refusal to join the study, while 640 signed informed consent from parents were returned by children. However, 34 children did not fulfill inclusion criteria. Therefore, 606 school children participated in the study, composed of 296 males and 310 females.

Table 1 shows the prevalence of EH and EO between two sexes. Of 606 examined schoolchildren aged 8--9 years, 321 had some form of DDE. Diffused opacity was the least common type of DDE in both sexes [Table 1].

The frequency of environmental and maternal factors of MIH was shown in Table 2. 30 (9.3%) schoolchildren in MIH group and 29 (10.2%) in group without MIH had history of allergy. There was no statically significant relation between history of allergy and MIH ($P = 0.73$).

12 (3.7%) children with MIH and 19 (6.7%) without MIH had previous frequent fever. Analysis showed relationship between frequent fever and MIH was not statically meaningful ($P = 0.1$).

Of 321 schoolchildren with MIH, 33 children (10.3%) had low birth weight (<2500 g). The difference in frequency among the study groups is statistically significant. $P = 0.01$ indicating the prevalence of MIH varies in direct relation to birth weight [Table 2]. Although the rate of long breastfeeding (more than 6 month) was higher in MIH group, 291 (90.7%) rather than 250 (87.7%), but the difference was not statically significant ($P = 0.5$). The following variables related to the child were reported below: positive history of allergy ($P = 0.73$), recurrent fever ($P = 0.1$), and previous sever infection with antibiotic taken during childhood ($P = 0.004$), and low birth weight ($P = 0.01$). There was a statistically significant relationship between occurrence of MIH and antibiotic taking and low birth weight [Table 2].

Cross-sectional analysis indicated no significant association between MIH and body height [Table 3].

Discussion

This cross-sectional study focused on the putative factors identified in an Iranian schoolchildren with MIH to MIH occurrence was omitted by evaluating simultaneous involvement of incisor and molar teeth.^[18-20]

Molar and incisor teeth were selected because permanent teeth were affected more than primary

Table 1: Distribution of types of developmental defects of enamel (DDE) in permanent teeth according to the number of affected teeth

Group	Diffused opacity <i>n</i> (%)	Demarcated opacity <i>n</i> (%)	Enamel hypoplasia <i>n</i> (%)	Total <i>n</i> (%)
Girls with DDE*	24 (14.1)	73 (42.9)	73 (42.9)	170 (100)
Boys with DDE	19 (12.6)	62 (41.1)	70 (46.3)	151 (100)

*Developmental defects of enamel, Chi-square test, $P=0.001$

Table 2: Frequency of environmental and maternal factors of MIH

Problems		With MIH	Without MIH	P
Allergy	Yes	30 (9.3)	29 (10.2)	0.731*
	No	291 (90.6)	256 (89.8)	
Recurrent fever	Yes	12 (3.7)	19 (6.7)	0.102*
	No	309 (96.3)	266 (93.3)	
Antibiotic therapy	Yes	14 (4.4)	30 (10.5)	0.004**
	No	307 (95.6)	255 (89.5)	
Low birth weight	<2500 g	33 (10.3)	43 (15.1)	0.014**
	2500-3900 g	259 (80.7)	201 (70.5)	
	>3900 g	29 (9)	41 (14.4)	
Breast feeding	<6 months	16 (5)	19 (6.7)	0.506
	>6 months	291 (90.7)	250 (87.7)	
	Dry milk	14 (4.4)	16 (5.6)	

*Chi-square test significant, **Significant

Table 3: Descriptive statistics for stature in the schoolchildren

Groups	With MIH* Mean±SD	Without MIH Mean±SD
Girls	125.23±1.3	128.08±0.82
Boys	131.17±0.73	133.23±0.76
P	0.115	0.054

*Molar incisor hypoplasia. *t*-test

teeth by DDE, and molar and incisor teeth are most commonly involved.^[4,7]

Whole permanent teeth except third molars are calcified completely before 8 years old, therefore 8-9 year-old schoolchildren participated in the study.^[21]

According to the results, the lower the birth weight of child, the greater chance to develop MIH. It could be constructed that MIH is a sign of calcium disorders associated to childhood period.^[4,13,22,23]

Regarding sex, the greater prevalence of MIH in girls was similarly documented by others^[24,25] but in contrast with results of Basha.^[22]

History of infectious disease during childhood was more in MIH group. it might be related to elevated temperature in sever infectious that affect the ameloblasts.^[26] Of course antibiotic use following infectious disease could impact on ameloblasts in addition to fever.^[27]

Maternal factors such as use of medications and infection during the parental period have been associated with a higher prevalence of MIH. Hypocalcemia and vitamin D mal absorption associated to above maternal problems could damage to ameloblasts.^[4]

Prevalence of MIH was similar in both groups, regarding premature birth and type of nutrition. These factors could influence on vital odontoblasts more than non-vital ameloblasts.^[28]

On the other hand, in critical period of teeth calcification, first 6 months of newborn, maternal antibody are accessible and environmental factors almost would be ineffective.^[29]

It is noteworthy that premature birth and dry milk fed children are points of more attention to take supplements.^[30] The authors suggested to compare future study.

There was no meaningful difference between two groups in view of history of allergy and trauma to teeth. Of course, ameloblasts are sensitive to hypoxia, but most of the schoolchildren have mild to moderate allergy without need to hospitalize. Therefore, this factor could not impact ameloblasts. Simultaneous evaluation of body height and MIH was a new topic that was measured in this study by Clark *et al.*^[31] Data showed although average of body height in MIH group was lesser than control group, it was not statistically meaningful. Multifactorial process terminate to MIH and body height and some of them were inevitable in this study. It could be suggested to design similar studies with larger sample size, after growth mutation and dentin disorders in future studies. Some of the limitation of our study was inability to determine severity of hypoplasia in different tooth surface.

Conclusions

Despite our limitations, the present data showed schoolchildren who had history of severe infection in early childhood, low birth weight, and maternal problem are predisposed to MIH.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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References

- Jalevik B, Noren JG. Enamel hypomineralization of permanent first molars: A morphological study and survey of possible aetiological factors. *Int J Paediatr Dent* 2000;10:278-89.
- Kim Seow W, Ford D, Kazoullis S, Newman B, Holcombe T. Comparison of enamel defects in the primary and permanent

- dentitions of children from a low-fluoride District in Australia. *Pediatr Dent* 2011;33:207-12.
3. Martinez Gomez TP, Guinot Jimeno F, Bellet Dalmau LJ, Giner Tarrida L. Prevalence of molar-incisor hypomineralisation observed using transillumination in a group of children from Barcelona (Spain). *Int J Paediatr Dent* 2012;22:100-9.
 4. Correa-Faria P, Martins-Junior PA, Vieira-Andrade RG, Marques LS, Ramos-Jorge ML. Perinatal factors associated with developmental defects of enamel in primary teeth: A case-control study. *Braz Oral Res* 2013;27:363-8.
 5. Mahmodian J, Kousari A, Mortazavi S. The prevalence of enamel defects in students 7-12 years of age in Isfahan. *J Dent Med* 2000;13:43-51.
 6. Crombie F, Manton D, Kilpatrick N. Aetiology of molar-incisor hypomineralization: A critical review. *Int J Paediatr Dent* 2009;19:73-83.
 7. Ramezani J, Mirkarimi M. A review of molar-incisor hypomineralization (MIH): Diagnosis, etiology and treatment. *J Isfahan Dent Sch* 2011;7:344-54.
 8. Simmer JP, Hu JC. Dental enamel formation and its impact on clinical dentistry. *J Dent Educ* 2001;65:896-905.
 9. Wong HM. Aetiological factors for developmental defects of enamel. *Austin J Anat* 2014;1:1003.
 10. Robles MJ, Ruiz M, Bravo-Perez M, Gonzalez E, Penalver MA. Prevalence of enamel defects in primary and permanent teeth in a group of schoolchildren from Granada (Spain). *Med Oral Patol Oral Cir Bucal* 2013;18:e187-93.
 11. Mehran M, Jalayer Naderi N, Hosseini M. The prevalence of enamel defects and its influencing factors among incisors and permanent first molars in 8-9 year-old children of Tehran in 2004. *J Islam Dent Assoc* 2004;17:114-20.
 12. Goodman AH, Armelagos GJ. The chronological distribution of enamel hypoplasia in human permanent incisor and canine teeth. *Archives of Oral Biology* 1985;30:503-7.
 13. Kim Seow W, Humphrys C, Tudehope DI. Increased prevalence of developmental dental defects in low birth-weight, prematurely born children: A controlled study. *Pediatr Dent* 1987;9:221-5.
 14. Guatelli-Steinberg D, Larsen CS, Hutchinson DL. Prevalence and the duration of linear enamel hypoplasia: A comparative study of Neandertals and Inuit foragers. *J Hum Evol* 2004;47:65-84.
 15. Vercellotti G, Piperata BA, Agnew AM, Wilson WM, Dufour DL, Reina JC, *et al.* Exploring the multidimensionality of stature variation in the past through comparisons of archaeological and living populations. *Am J Phys Anthropol* 2014;155:229-42.
 16. Garg N, Jain AK, Saha S, Singh J. Essentiality of early diagnosis of molar incisor hypomineralization in children and review of its clinical presentation, etiology and management. *Int J Clin Paediatr Dent* 2012;5:190-6.
 17. Weerheijm KL, Duggal M, Mejare I, Papagiannoulis L, Koch G, Martens LC, *et al.* Judgement criteria for molar incisor hypomineralisation (MIH) in epidemiologic studies: A summary of the European meeting on MIH held in Athens, 2003. *Eur J Paediatr Dent* 2003;4:110-3.
 18. Arrow P. Prevalence of developmental enamel defects of the first permanent molars among school children in Western Australia. *Aust Dent J* 2008;53:250-9.
 19. Vejdani J, Kaboodan M. Prevalence of enamel defects and associating factors in permanent incisors and first molars in 8-9 years-old children. *J Guilan Univ Med Sci* 2010;19:13-9.
 20. Procaccini M, Campisi G, Bufo P, Compilato D, Massaccesi C, Catassi C, *et al.* Lack of association between celiac disease and dental enamel hypoplasia in a case-control study from an Italian central region. *Head Face Med* 2007;3:25.
 21. Proffit WR, Fields HW, Sarver DM. *Contemporary Orthodontics*. 5th ed. St. Louis: Elsevier; 2012.
 22. Basha S, Mohamed RN, Swamy HS. Prevalence and associated factors to developmental defects of enamel in primary and permanent dentition. *Oral Health Dent Manag* 2014;13:588-94.
 23. Kakoei S, Bahman Bijari B. Assessment of the enamel defects in primary teeth of premature infants. *J Isfahan Dent Sch* 2009;5:81-7.
 24. Elfrink ME, ten Cate JM, Jaddoe VW, Hofman A, Moll HA, Veerkamp JS. Deciduous molar hypomineralization and molar incisor hypomineralization. *J Dent Res* 2012;91:551-5.
 25. Ghanim A, Bagheri R, Golkari A, Manton D. Molar-incisor hypomineralisation: A prevalence study amongst primary schoolchildren of Shiraz, Iran. *Eur Arch Paediatr Dent* 2014;15:75-82.
 26. Ford D, Kim Seow W, Kazoullis S, Holcombe T, Newman B. A controlled study of risk factors for enamel hypoplasia in the permanent dentition. *Pediatr Dent* 2008;31:382-8.
 27. Tariq A, Alam Ansari M, Owais Ismail M, Memon Z. Association of the use of bacterial cell wall synthesis inhibitor drugs in early childhood with the developmental defects of enamel. *Pak J Med Sci* 2014;30:393-7.
 28. Murray PE, About I, Lumley PJ, Franquin JC, Remusat M, Smith AJ. Human odontoblast cell numbers after dental injury. *J Dent* 2000;28:277-85.
 29. Nanci A. *Ten Cate's Oral Histology-E-Book: Development, Structure, and Function*. 9th ed. St. Louis: Elsevier Health Sciences; 2017.
 30. Kumar RK, Singhal A, Vaidya U, Banerjee S, Anwar F, Rao S. Optimizing nutrition in preterm low birth weight infants-consensus summary. *Front Nutr* 2017;4:20.
 31. Clark AL, Tayles N, Halcrow SE. Aspects of health in prehistoric mainland Southeast Asia: Indicators of stress in response to the intensification of rice agriculture. *Am J Phys Anthropol* 2014;153:484-95.