Journal of Oral Science, Vol. 55, No. 3, 203-207, 2013

Original

Dental development in children with severe molar-incisor hypomineralization in Samsun, Turkey

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(Received March 28, 2013; Accepted May 28, 2013)

Abstract: Anomalies in amelogenesis may be due to developmental defects or abnormalities in different components of developing teeth and can affect dental development. We compared dental development in a group of children with molar-incisor hypomineralization (MIH) with that in age- and sex-matched controls. Dental age was determined using panoramic radiographs of 105 children (59 girls, 46 boys) aged 7-11 years with severe MIH, and the findings were compared with those from 105 healthy age- and sex-matched controls. Although accelerated dental development was noted in the MIH group, the difference between the MIH and control groups was not statistically significant ($P < 0.05$). Furthermore, no relationship was found between number of affected teeth and the difference between dental and chronological age. In conclusion, children with severe MIH had slightly accelerated dental development as compared with controls. (J Oral Sci 55, 203-207, 2013)

Keywords: children; dental development; molar-incisor hypomineralization (MIH).

Introduction

Amelogenesis is the orchestrated and genetically conserved process of enamel formation (1). However, this process can be disrupted by numerous influences: well-known causes of developmental enamel defects include inheritance (as in amelogenesis imperfecta [AI]), excess fluoride intake, and systemic disturbance or trauma during amelogenesis (2-4). Moreover, idiopathic developmental enamel defects have been reported in the literature since the 1970s (5). In 2001, the term molar–incisor hypomineralization (MIH) was introduced to describe a specific abnormality caused by a disturbance during the early phase of enamel maturation in one or more permanent first molars, frequently associated with affected incisors (6). Studies of the cause of MIH have not produced clear results, although it has been linked to several pre-and perinatal conditions (7-10).

Clinically, the enamel defects of MIH present as opaque lesions varying in color from white to yellow or brown, with a sharp demarcation between affected and sound enamel. In severe cases, post-eruptive enamel breakdown is so rapid that it appears clinically as if the enamel has not formed at all (1,11,12).

Recent research has emphasized the close interaction between the different components of the developing tooth germ during odontogenesis (13), and findings suggest that defects in amelogenesis are associated with developmental defects or abnormalities in one or more components of the developing tooth, as well as in dentoskeletal development (e.g., taurodontism, agenesis, delayed eruption, and frontal open bite). In light of these findings, it is possible that the dental development of children with MIH is also affected. To our knowledge, no study has examined dental development in children with MIH. Therefore, we compared dental development in a group of children with severe MIH with that in a group of age- and sex-matched healthy controls.

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Materials and Methods

Study population

The research protocol was approved by the Ondokuz

Mayıs University Human Subjects Ethics Committee (190/2013) and informed consent was obtained from the parents of all study participants.

All children aged 7-11 years with severe MIH who presented at the Ondokuz Mayis University Faculty of Dentistry's Pediatric Dentistry Clinic in Samsun, Turkey during a 2-year period were invited to participate in the study. Most children had been referred from general practice, but children under routine care in the department were also included. Controls were selected from among patients without signs of hypomineralization who sought routine care at the department during the study period and were matched with MIH cases by sex and chronological age (± 0.1 years) at the time of panoramic radiography. Because our clinical protocol does not include routine panoramic radiographic examination for mild and moderate MIH cases, only severe MIH cases were included in the present study. Moreover, we included only those children whose parents gave their informed consent and who had a panoramic radiograph of sufficient quality for assessment of dental development. Children with developmental enamel defects caused by AI, dental fluorosis, local trauma, or major general health conditions, and those currently undergoing orthodontic treatment, were excluded from the study. A total of 105 children (59 girls, 46 boys) were identified as having severe MIH (> 0.1 mg/l *F* water content).

Dental examinations

All dental examinations were conducted by one of the authors (A.T.U.), an experienced pediatric dentist, at the Department of Pediatric Dentistry under optimal lighting using a mirror and probe. All teeth were dried before inspection.

MIH was diagnosed according to criteria proposed by the European Academy of Paediatric Dentistry (14). Hypomineralized lesions of < 1 mm in diameter were not recorded. MIH lesion severity was graded as follows: mild MIH, demarcated opacities not requiring treatment; moderate MIH, lesions associated with rough and broken enamel; severe MIH, lesions associated with loss of dental structure affecting both enamel and dentin and/or atypical restorations replacing affected hard tissue as well as teeth extracted because of severe hypomineralization. For each child, MIH severity was recorded according to the most severe defect observed among the permanent first molars and incisors (15).

A data-collection document was developed to obtain information on patient code, age, sex, date of birth, date of panoramic radiography, and number/location/severity of teeth with MIH.

Assessment of dental development

Dental development was assessed from panoramic radiographs, using the dental age (DA) estimation method of Demirjian et al. (16), which determines DA based on the radiographic appearance of the seven permanent mandibular teeth on the left side, supplemented by the corresponding teeth on the right side when a left tooth was missing. Tooth formation is divided into eight stages, and detailed written criteria and supplementary illustrations, separated by tooth and sex, are provided. For each tooth, a biologically weighted score is allocated according to development stage, and the sum of the seven scores is recorded as the estimate of a subject's dental maturity. This overall maturity score is then converted to DA using available tables and/or percentile curves (16,17).

Dental development of the study sample was assessed by one of the authors (E.S.T.), a trained examiner, blinded to subject age and sex. Assessment was performed in a darkened room using a radiographic illuminator, to provide contrast enhancement of dental images. After determining DA, chronological age (CA) for each subject was calculated by subtracting the date of the panoramic radiograph from the date of birth, after converting both to decimal ages. The difference between DA and CA (DA-CA) was also recorded.

Reproducibility

A total of 22 radiographs (10% of the sample) were reassessed after a 1-month interval, and κ coefficients were calculated to determine level of agreement.

Statistical analysis

The data were analyzed using the software program SPSS version 16.0 (SPSS Inc., Chicago, IL, USA). Normality of distribution was evaluated using the Shapiro-Wilk test, after which the Mann-Whitney *U* test was used to compare mean differences in DA and CA between the MIH and control groups. Spearman rank correlation analysis was performed to determine the relationship between the number of teeth affected by MIH and DA-CA values. The level of significance was set at 0.05.

Results

Intra-examiner consistency

An unweighted κ of 0.96 confirmed good intra-examiner consistency.

Demographic characteristics

Table 1 shows the distribution of MIH patients according to age and sex. Among the 105 children (59 girls, 46 boys) in the MIH group, mean (±SD) CA and DA were

Table 1 Age and sex distribution of patients with MIH

Age group (years)	Girls	Boys
7.0-7.9	3	8
8.0-8.9	13	15
9.0-9.9	9	15
10.0-10.9	10	13
11.0-11.9	11	8
Total	59	46

Table 3 Difference in DA-CA between control and MIH groups

	Control group – Hypodontia group	(years)
Girls	0.63-0.51	0.12
Boys	0.61-0.47	0.14

Table 2 Descriptive data for MIH and control groups

Groups		Chronological age (CA) mean ± SD	Dental age (DA) mean ± SD	Dental age – Chronological age (DA-CA) mean ± SD
MIH group (n = 105)	Girls	9.42 ± 1.24	9.94 ± 1.67	0.51 ± 1.21
	Boys	9.68 ± 1.33	10.15 ± 1.65	0.47 ± 0.89
Control group (n = 105)	Girls	9.42 ± 1.24	10.04 ± 1.87	0.63 ± 1.06
	Boys	9.68 ± 1.32	10.25 ± 1.61	0.61 ± 0.67

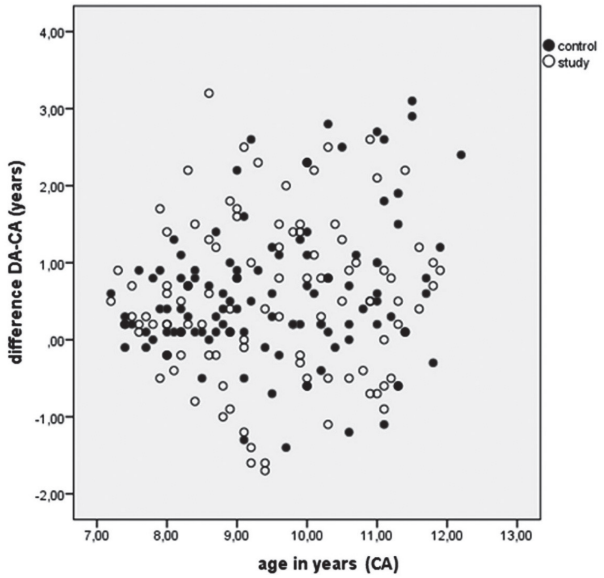

Fig. 1 Scatterplot of DA-CA for control and MIH groups.

9.42 ± 1.24 years and 9.68 ± 1.33 years, respectively. The mean number of affected molars/incisors was 7.63 for girls and 7.46 boys.

Assessment of dental development

Table 2 shows the mean (SD) of CA, DA, and DA-CA for the MIH and control groups by sex. The highest mean difference in DA-CA was observed among girls in the control group; however, it did not exceed 0.63 years. The difference in DA-CA between the experimental and control groups was not statistically significant among girls (P = 0.653) or boys (P = 0.594). However, the MIH group showed a tendency toward accelerated dental development among boys (+0.14) and girls (+0.12) (Table 3). Scatterplots were constructed to compare CA and DA-CA in the MIH and control groups (Fig. 1). The distribution pattern of DA-CA was similar for both groups and ranged from 0-1 years in most cases.

Spearman rank correlation analysis showed no relationship between number of teeth affected by MIH and DA-CA values (r = 0.028, P = 0.777).

Discussion

As the prevalence of caries in children and adolescents continues to decrease, developmental enamel defects are attracting increasing attention in terms of scientific theory and clinical practice (18). Children with MIH are a particular challenge for clinicians and researchers, as variation in MIH prevalence was reported to vary between 2.4% and 40.2% in general populations of children (19). To date, most research on MIH has focused on prevalence and possible etiological factors (7,11-19).

Teeth with MIH are often very sensitive and may rapidly break down. In some cases, early extraction is the treatment of choice (19-21). Accurate prediction of tooth maturation and eruption is essential for satisfactory treatment planning and is especially important in the treatment of MIH because of the clinical and pathological implications of the condition.

In diagnosing MIH, it is important to differentiate it from other developmental disturbances of the enamel. In the present study, strict case selection and examination criteria were used. Individuals with generalized enamel defects in primary or permanent dentition were considered to potentially have AI or a systemic condition that extended over a period of time long enough to affect tooth

development at different time periods and were therefore excluded from the study. We did not examine the causes of MIH among the study participants. However, given that a familial component has been reported in some MIH cases (1), patient family histories were examined, and those patients who had family members with enamel defects or dental anomalies were also excluded from the study. MIH cases were selected according to EAPD criteria, and MIH severity was determined according to the criteria of Leppaniemi et al. (15). Clinical examination was performed under optimal lighting using a mirror and probe, and all teeth were dried before inspection.

Dental age can be determined from dental emergence or from the radiographic appearance of tooth-formation stages. However, radiographic analysis is preferred because tooth emergence, i.e., the initial appearance of a tooth in the oral cavity, occurs over a short period of time (22) and can be affected by local factors such as lack of space (23) and systemic factors such as nutritional status (24,25). Several methods have been described for determining dental development stage from radiographs (16,17,23,26). One widely used method is that of Demirjian et al. (16), which was first described in a large-scale study of French-Canadian children, conducted in 1973. This method was used in the present study because its applicability and reproducibility have been confirmed in earlier studies (27,28).

Although we found that dental development in children with MIH did not significantly differ from that in controls, there was a trend toward accelerated dental development among both boys (+0.14) and girls (+0.12) in the MIH group. In a review of the literature we found no studies of dental development in children with MIH. However, because MIH has been suggested to result from various environmental factors that act systemically to disrupt ameloblasts during the enamel-production phase (9), the results of the present study are consistent with those of a previous study of dental development and AI (29), which found accelerated dental development and hypocalcified enamel in patients with AI. The author of that study suggested that accelerated dental development could reflect a lower level of enamel mineralization, which would require less time for crown development as compared with normal enamel. Possible changes in root development rates were also mentioned in relation to enamel defects.

We found no correlation between number of MIH-affected teeth and the extent of the delay in dental development, perhaps because we enrolled only severe MIH cases.

The results showed no significant difference between MIH and controls but showed a trend toward accelerated dental development in MIH patients as compared with controls. Studies of larger samples and other populations will be necessary to confirm our result.

References

1. Mathu-Muju K, Wright JT (2006) Diagnosis and treatment of molar incisor hypomineralization. Compend Contin Educ Dent 27, 604-610.
2. Small BW, Murray JJ (1978) Enamel opacities: prevalence, classifications and aetiological considerations. J Dent 6, 33-42.
3. Pindborg JJ (1982) Aetiology of developmental enamel defects not related to fluorosis. Int Dent J 32, 123-134.
4. Sundell S, Koch G (1985) Hereditary amelogenesis imperfecta. I. Epidemiology and clinical classification in a Swedish child population. Swed Dent J 9, 157-169.
5. Koch G, Hallonsten AL, Ludvigsson N, Hansson BO, Holst A, Ullbro C (1987) Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children. Community Dent Oral Epidemiol 15, 279-285.
6. Weerheijm KL, Jälevik B, Alaluusua S (2001) Molar-incisor hypomineralisation. Caries Res 35, 390-391.
7. Whatling R, Fearne JM (2008) Molar incisor hypomineralization: a study of aetiological factors in a group of UK children. Int J Paediatr Dent 18, 155-162.
8. Crombie F, Manton D, Kilpatrick N (2009) Aetiology of molar-incisor hypomineralization: a critical review. Int J Paediatr Dent 19, 73-83.
9. Kuscu OO, Caglar E, Aslan S, Durmusoglu E, Karademir A, Sandalli N (2009) The prevalence of molar incisor hypomineralization (MIH) in a group of children in a highly polluted urban region and a windfarm-green energy island. Int J Paediatr Dent 19, 176-185.
10. Alaluusua S (2010) Aetiology of molar-incisor hypomineralisation: a systematic review. Eur Arch Paediatr Dent 11, 53-58.
11. William V, Messer LB, Burrow MF (2006) Molar incisor hypomineralization: review and recommendations for clinical management. Pediatr Dent 28, 224-232.
12. Ghanim A, Morgan M, Mariño R, Bailey D, Manton D (2011) Molar-incisor hypomineralisation: prevalence and defect characteristics in Iraqi children. Int J Paediatr Dent 21, 413-421.
13. Thesleff I (2006) The genetic basis of tooth development and dental defects. Am J Med Genet A 140, 2530-2535.
14. Weerheijm KL, Duggal M, Mejàre I, Papagiannoulis L, Koch G, Martens LC et al. (2003) 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 4, 110-113.
15. Leppäniemi A, Lukinmaa PL, Alaluusua S (2001) Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. Caries Res 35, 36-40.
16. Demirjian A, Goldstein H, Tanner JM (1973) A new system

of dental age assessment. Hum Biol 45, 211-227.
17. Demirjian A, Goldstein H (1976) New systems for dental maturity based on seven and four teeth. Ann Hum Biol 3, 411-421.
18. Heitmüller D, Thiering E, Hoffmann U, Heinrich J, Manton D, Kühnisch J et al. (2013) Is there a positive relationship between molar incisor hypomineralisations and the presence of dental caries? Int J Paediatr Dent 23, 116-124.
19. Jälevik B (2010) Prevalence and diagnosis of molar-incisor-hypomineralisation (MIH): a systematic review. Eur Arch Paediatr Dent 11, 59-64.
20. Jälevik B, Klingberg GA (2002) Dental treatment, dental fear and behaviour management problems in children with severe enamel hypomineralization of their permanent first molars. Int J Paediatr Dent 12, 24-32.
21. Jälevik B, Möller M (2007) Evaluation of spontaneous space closure and development of permanent dentition after extraction of hypomineralized permanent first molars. Int J Paediatr Dent 17, 328-335.
22. Eid RM, Simi R, Friggi MN, Fisberg M (2002) Assessment of dental maturity of Brazilian children aged 6 to 14 years using Demirjian's method. Int J Paediatr Dent 12, 423-428.
23. Moorrees CF, Fanning EA, Hunt EE Jr (1963) Age variation of formation stages for ten permanent teeth. J Dent Res 42, 1490-1502.
24. McGregor IA, Thomson AM, Billewicz WZ (1968) The development of primary teeth in children from a group of Gambian villages, and critical examination of its use for estimating age. Br J Nutr 22, 307-314.
25. Infante PF, Owen GM (1973) Relation of chronology of deciduous tooth emergence to height, weight and head circumference in children. Arch Oral Biol 18, 1411-1417.
26. Haavikko K (1970) The formation and the alveolar and clinical eruption of the permanent teeth. An orthopantomographic study. Suom Hammaslaak Toim 66, 103-170.
27. Nykänen R, Espeland L, Kvaal SI, Krogstad O (1998) Validity of the Demirjian method for dental age estimation when applied to Norwegian children. Acta Odontol Scand 56, 238-244.
28. Baghdadi ZD, Pani SC (2012) Accuracy of population-specific Demirjian curves in the estimation of dental age of Saudi children. Int J Paediatr Dent 22, 125-131.
29. Seow WK (1995) Dental development in amelogenesis imperfecta: a controlled study. Pediatr Dent 17, 26-30.