The prevalence and aetiology of Molar-Incisor Hypomineralisation in a group of children in Istanbul

O. ONDER KUSCU, E. CAGLAR, N. SANDALLI

ABSTRACT: Aim To our knowledge, the prevalence and aetiology of molar-incisor hypomineralisation has not been discussed nor investigated in Turkish children in Istanbul. Therefore the aim of the present study is to investigate the prevalence and aetiology of MIH in a group of children in Istanbul. Design Between April and July 2007, a retrospective clinical study was initiated at the Dept. of Paediatric Dentistry, School of Dentistry, Yeditepe University, Istanbul, Turkey. A total of 147 children aged 7-9 years visiting our clinic were examined by two calibrated paediatric dentists (kappa: 0.89). The examiners used the criteria for the diagnosis of demarcated opacities, post-eruption breakdown, atypical restorations, and extracted PFMs due to MIH developed by Weerheijm et al. Results In the present study, prevalence of MIH was 14.9%. Of the 22 children affected with MIH, 17 (77.2%) had only demarcated opacities, but no breakdown or atypical restorations. Regarding diseases in the first 3 years of life, 55% of MIH and 19.4% of non-MIH children had a disease history. 27% of MIH children had suffered from upper and lower respiratory tract infections including bronchitis. This was significantly different from non-MIH children (p<0.001). Conclusion MIH was common among a group of 7-9 years old children, Istanbul.

KEYWORDS: Hypomineralisation; Molars; Incisors; MIH; Prevalence.

Introduction

The term molar incisor hypomineralisation (MIH) was introduced in 2001 to describe the clinical appearance of enamel hypomineralisation of systemic origin affecting one or more permanent first molars (PFMs) that are frequently associated with the affected incisors [Weerheijm et al., 2001]. It has also been referred to as “hypomineralised” PFMs [Jalevik and Noren, 2000] “idiopathic enamel hypomineralisation” [Koch et al., 1987; Fearne et al., 2004], “dysmineralised” PFMs (“nonfluoride hypomineralisation”) [Holtta et al., 2001; Leppaniemi et al., 2001] and “cheese molars” [van Amerongen and Kreulen, 1995; Weerheijm et al., 2001b]. The condition is attributed to disrupted ameloblastic function during the transitional and maturational stages of amelogenesis [Fearne et al., 2004; Kuscu et al., 2007]. The developmental defects observed in MIH can create considerable discomfort to the child (reports of shooting pains when they are eating ice cream, or even breathing cold air shortly after the eruption of the affected teeth has started), concern to the parents, and problems to the clinician regarding the management of the affected teeth [Jalevik and Klingberg, 2002; Fayle, 2003; Kuscu et al., 2007]. Current information suggests that children presenting with this type of defects require extensive and often repeated restorative treatment [Leppaniemi et al., 2001; Jilevik and Klingberg, 2002]. The limited prevalence data for MIH reflects several diagnostic classifications. Using the criteria of Weerheijm et al. [2003], the prevalence ranges from 4% to 25% [Koch et al., 1987; Alaluusua et al., 1996a; Jalevik et al., 2001a; Leppaniemi et al., 2001; Weerheijm et al., 2001b; Dietrich et al., 2003; Jasulaityte et al., 2007]. The number of hypomineralised PFMs in an individual can vary from 1 to 4, affecting particularly 2 or more PFMs including the contralateral tooth, where the teeth are moderately or severely affected [Alaluusua et al., 1996a; Jalevik et al., 2001a; Weerheijm et al., 2001a; Dietrich et al., 2003, Lygidakis et al., 2003]. The risk of
involvement of the permanent maxillary incisors appears to increase when more PFM are affected.

By the age of 9, children affected with MIH generally have to undergo dental treatment of their PFM nearly 10 times more often than the healthy controls [Jalevik and Klingberg, 2002]. Recently Kotsanos et al. [2005] reported that children exhibiting MIH have a probability 11 times higher of undergoing restorative treatment in their PFM compared with children of a control group. Moreover, fillings and sealants in MIH affected children have over 3 times a higher probability of requiring re-treatment than the control group [Kotsanos et al., 2005].

This situation shows the importance of preventing children from getting MIH and attempts to understand its aetiology are critical. In the literature, factors associated with the development of MIH include systemic conditions and environmental insults influencing natal and early development [van Amerongen and Kreulen, 1995; Jalevik et al., 2001b]. To our knowledge, prevalence and aetiology of MIH has not been fully discussed nor investigated in Turkish children. Therefore the aim of the present study is to investigate the prevalence and aetiology of MIH in a group of children in Istanbul.

**Methods**

Between April and July 2007, a retrospective clinical study was conducted at the Department of Pediatric Dentistry of the School of Dentistry of Yeditepe University, Istanbul (Turkey). The study protocol was approved by the School of Dentistry Ethics Committee of the University of Yeditepe. A total of 147 children aged 7-9 years visiting our clinic were examined. Dental examinations were carried out by two calibrated paediatric dentists (EC, OOK) (kappa: 0.89). After thorough cleaning, the 4 PFMs and 8 erupted permanent incisors were examined wet for demarcated opacities, post-eruption breakdown (PEB), and atypical restorations [Weerheijm et al., 2003]. Criteria for the diagnosis of demarcated opacities, PEB, atypical breakdown (PEB), and atypical restorations [Weerheijm et al., 2003]. Criteria for the diagnosis of demarcated opacities, PEB, atypical restorations, and extracted PFMs due to MIH were developed by Weerheijm et al. [2003]. Demarcated opacities were defined as defects of altered enamel translucency; the defective enamel is white-cream or yellow-brown in colour, of normal thickness with a smooth surface, and has a distinct boundary adjacent to normal enamel [Commission on Oral Health Research & Epidemiology, 1992; Jalevik and Noren, 2000]. Dentitions with generalised opacities present on all teeth (such as in several forms of amelogenesis imperfecta), rather than limited to the PFMs and permanent incisors, are not considered to have MIH [Weerheijm et al., 2003; Kuscu et al., 2007]. The location of demarcated opacities and enamel breakdown was recorded on specially designed data sheets. Details of medical history and various confounding factors (such as birth details, place of residence during tooth development, duration of breast-feeding, childhood diseases including respiratory, ear or other infections, hospitalisation, fever >39° C, medications, fluoride exposure, and environmental (toxin exposure) of MIH patients had been recorded after personal interview with patients’ mothers. Patients’ health data booklet with all medical information recorded by the paediatrician was also examined. It should be noted that only children aged 7-9 years having 4 PFMs and 8 permanent incisors erupted were included in the study. At this age, PFM’s have just erupted, caries prevalence is still low, and thus minimises the probability that hypomineralisation lesions were masked by carious lesions. The collected data were analysed using SPSS software version 10.0 for Windows and the level significance was set at 5%. Chi Square, Mann-Whitney U, and Fisher exact test were applied for statistical analysis.

**Results**

A total of 147 children (67 F, 80 M) visiting the department of Pediatric Dentistry and fulfilling the study criteria during a 4 months period were examined. In the present study, prevalence of MIH was 14.9% (n. 22). The majority of children (85%, n. 125) were not affected with MIH. Of the 22 children affected with MIH, 17 (77.2%) had only demarcated opacities, but no breakdown or atypical restorations. At least one tooth with breakdown was present in 3 of the children (13.6%) and 2 children (9%) had both atypical restoration and an extraction of PFM. Of the 22 children with MIH, only one had lesions confined to the PFMs and 21 (95.4%) had lesions in both incisors and molars. 5 children (22.8%) had a single molar and incisors affected (Table 1).

<table>
<thead>
<tr>
<th>Affected teeth</th>
<th>Girls</th>
<th>Boys</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single molar</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Single molar + incisors</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Only two molars</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Molars + incisors</td>
<td>9</td>
<td>7</td>
<td>16</td>
</tr>
<tr>
<td>Total MIH children</td>
<td>14</td>
<td>8</td>
<td>22</td>
</tr>
</tbody>
</table>

**Table 1 - Distribution of MIH in different tooth groups in a group of 22 children.**
Tooth 16 was the most affected PFM, and tooth 46 the least. Tooth 16 was significantly more affected from MIH than both other PFMs and incisors (p<0.001). Tooth 21 was the most affected incisor and tooth 31 the least affected incisor by MIH. Tooth 21 and 11 were significantly more affected from MIH than tooth 12, 22, 32, 31, 41 and 42 (p <0.001).

Distribution of affected teeth is presented in Table 2.

Regarding gender, there were no statistical differences between MIH (14 F, 8 M) and non-MIH (53 F, 72 M) groups (p >0.05).

Regarding aetiological factors such as diseases in the first three years of life, none of the mothers reported childhood diseases. Regarding diseases in first 3 years of life, 55% of MIH and 19.4% of non-MIH children had a disease history (Table 3). In the present study, 27% of the MIH children had suffered from upper and lower respiratory tract infections including bronchitis. This was significantly different than the non-MIH children (p <0.001) (Table 3). 13.6% of the MIH children had also suffered from renal infections. This was also significantly different to the non-MIH children (p <0.01) (Table 3).

Regarding systemic diseases reported in MIH and non-MIH children, no statistical differences were recorded (p>0.05).

Regarding preterm birth data, only one child with MIH (4.5%) was reported to have been delivered preterm with very low birth weight, while 8 of non-

**Table 2 - Distribution of affected teeth (n. 22).**

<table>
<thead>
<tr>
<th>Tooth no</th>
<th>1</th>
<th>26</th>
<th>36</th>
<th>46</th>
<th>12</th>
<th>11</th>
<th>21</th>
<th>22</th>
<th>32</th>
<th>31</th>
<th>41</th>
<th>42</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>13*</td>
<td>12*</td>
<td>10*</td>
<td>4*</td>
<td>15**, 14**, 5**, 4**, 3**, 7**, 9 (59 (45.4%) (18.1%) (68.1%) (63.6%) (22.7%) (18.1%) (13.6%) (18.1) (31.8%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*,** p<0.001

**Table 3 - Diseases which occurred in a group of 147 children.**

<table>
<thead>
<tr>
<th>Diseases in the first 3 years of life</th>
<th>MIH</th>
<th>Non-MIH</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systemic disease</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Cardiology</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>- Rotated arthritis</td>
<td>1 (0.8 %)</td>
<td>1 (0.8 %)</td>
</tr>
<tr>
<td>- Immune deficiency</td>
<td>1 (0.8 %)</td>
<td>1 (0.8 %)</td>
</tr>
<tr>
<td><strong>Upper airway infections</strong></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>- Respiratory</td>
<td>4 (18.2 %)**</td>
<td>2 (1.6%)**</td>
</tr>
<tr>
<td>- Bronchitis</td>
<td>2 (9.1%)***</td>
<td>1 (0.8 %)NS</td>
</tr>
<tr>
<td>- Otitis media</td>
<td>1 (0.8 %)NS</td>
<td>3 (2.4%)NS</td>
</tr>
<tr>
<td><strong>Renal infections</strong></td>
<td>3 (13.6 %)**</td>
<td>1 (0.8 %)**</td>
</tr>
<tr>
<td><strong>Urinary infections</strong></td>
<td>-</td>
<td>3 (2.4%)NS</td>
</tr>
<tr>
<td><strong>Gastrointestinal infections</strong></td>
<td>-</td>
<td>1 (0.8 %)NS</td>
</tr>
<tr>
<td><strong>Viral infections</strong></td>
<td>1 (4.5%)NS</td>
<td>1 (0.8 %)NS</td>
</tr>
<tr>
<td><strong>Fever &gt;39o C</strong></td>
<td>2 (9 %)NS</td>
<td>8 (6.4%)NS</td>
</tr>
<tr>
<td><strong>Allergy, Asthma</strong></td>
<td>-</td>
<td>2 (1.6%)NS</td>
</tr>
<tr>
<td><strong>Fire burn</strong></td>
<td>-</td>
<td>1 (0.8 %)NS</td>
</tr>
<tr>
<td><strong>No disease history</strong></td>
<td>10 (45 %)*</td>
<td>102 (81.6 %)*</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>22 (100 %)</td>
<td>125 (100 %)</td>
</tr>
</tbody>
</table>

* p<0.001, **p< 0.01, *** p<0.05, NS p>0.05
MH children (6.4%) were born preterm.

With regard to breastfeeding duration, there were no significant differences (p > 0.05) between MIH (8.9 ± 8 months) and non-MIH (10.4 ± 8.3 months) children.

With regard to place of residence of mothers and their children up to the time of the present study: MIH children (born, breastfed, current residence) and their mothers (5 years before pregnancy and gestation up to time of study) had resided in a total of 6 urban areas in Turkey while the majority of them (77.2%) were in Istanbul. Non-MIH children (born, breastfed, current residence) and their mothers (5 years before gestation up to time of study) had resided in a total of 9 urban areas in Turkey and 3 foreign urban areas while majority of them (89.6%) were in Istanbul. There were no statistical differences between MIH and non-MIH children, regarding where their mothers had lived for the last 5 years before gestation and breastfeeding periods and where children had lived up to date (p > 0.05).

Discussion

Teeth develop as a result of a series of inductive, sequential, and reciprocal interactions between the ectoderm and the subjacent mesenchyme [Thesleff et al., 1995]. Tooth development is genetically regulated, but sensitive to environmental disturbances. Aberrations in the function of tooth-forming cells lead to permanent morphologic consequences [Sahlberg et al., 2007]. The last few years have seen a series of reports on the developmental defects observed and suggested as MIH [Weerheijm et al., 2003].

It has been stated that children with poor general health and systemic conditions are more likely to have developmental enamel defects [Hall, 1989; Pascoe and Seow, 1994]. In the present study, regarding diseases in first 3 years of life, 55% of MIH and 19.4% of non-MIH children had a disease history which may cause poor general health. From MIH children, 27% had suffered from upper and lower respiratory tract infections including bronchitis and this was significantly different from non-MIH children. In the present study, MIH children were also significantly more related to renal infections.

The systemic conditions implicated in MIH to date include nutritional deficiencies, heart defects, brain injury and neurologic defects, cystic fibrosis, syndromes of epilepsy and dementia, nephrotic syndrome, atopia, lead poisoning, cleft lip and palate, radiation treatment, rubella embryopathy, epidermolysis bullosa, ophthalmic conditions, coeliac disease, and gastrointestinal disorders [Hall, 1989; Kirkham et al., 2000; Klingberg et al., 2002; Martinez et al., 2002]. In the present study, regarding systemic diseases reported in MIH and non-MIH children, no statistical differences were recorded.

Determination of aetiological factors of MIH is complicated as the child may have many medical problems in the first 3 years of life after birth. Frequent preschool age infections such as upper respiratory diseases, otitis media, tonsillitis, chicken pox, measles, and rubella, appear to be associated with MIH [van Amerongen and Kreulen, 1995; Jalevik and Noren, 2000; Jalevik et al., 2001b]. In the present study, none of the mothers reported about childhood diseases such as chicken pox, measles, and rubella as the primary concern. It could be that vaccinations have prevented the destructive effects of some childhood diseases.

Preterm birth has been associated with increased prevalence of enamel defects, including hypomineralisation and hypoplasia in the permanent dentition [Seow, 1996; Seow, 1997; Aine et al., 2000; Martinez et al., 2002]. The status of the primary and permanent teeth was evaluated in 32 preterm children and in 64 control children in Finland. The prevalence of enamel defects in children born preterm was significantly higher as compared with controls in both the primary (78% vs. 20%) and permanent (83% vs. 36%) dentitions [Aine et al., 2000]. A study of 40 children born preterm with very low birth weight (< 1,500 g) in Brisbane (Australia) showed a significantly higher percentage of enamel defects in their PFMs (17%) than in a matched sample of normal birth weight children (8%) [Seow, 1996].

In the present study, only one child with MIH (4.5%) was reported to have been born preterm with very low birth weight and no relation could be associated with MIH.

In the present study, it could be concluded that the risk of involvement of the permanent maxillary incisors appears to increase the more PFMs are affected. This is also parallel to the results of the recent studies [Jalevik et al., 2001a; Weerheijm et al., 2001a; Lygidakis et al., 2003]. While tooth 16 was the most affected tooth from MIH, tooth 21 and 11 were significantly more affected from MIH than other incisors.

Children’s developing teeth may be sensitive to environmental toxins such as polyhalogenated and polychlorinated aromatic hydrocarbons which have adverse health effects on human body [Kuscu et al., 2007]. Twenty-five years after the dioxin accident in Seveso (Italy), subjects from the contaminated areas were found to show enamel defects, and
Developmental dental aberrations were associated with childhood exposure to toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) [Alaluusua et al., 2004]. Recently, associations have been made between the presence of polychlorinated aromatic hydrocarbons, mainly polychlorinated dibenzo-p-dioxins (PCDDs) in breast milk and enamel hypomineralisation in both clinical and laboratory studies [Alaluusua et al., 1996a,b]. Persistence and accumulation of PCDDs in tissue lipids and in the food chain may result in chronic low-level exposure in humans [Gao et al., 2004]. Likewise, in a Slovenian epidemiologic study [Jan and Vrbic, 2000] on 8-15 year olds found that pre- and postnatal exposure to polychlorinated biphenyls, mainly PCBs, resulted in significantly more developmental defects of enamel in comparison to children in a control area. Recently, the effects of long-term exposure to PCBs on developmental dental defects of deciduous and permanent teeth in children in eastern Slovakia demonstrated a dose–response relationship between PCB exposure and developmental enamel defects of permanent teeth in children [Jan et al., 2007]. However, other clinical studies have not found associations between dioxin compounds in breast milk and hypomineralised enamel [Holatta et al., 2001; Jalevik et al., 2001b]. In the present retrospective study, it is not possible to examine breast milk retrospectively, nor did we receive ethical clearance from the Yeditepe University Ethics Committee to examine the PCDD serum levels of MIH children. However, there were no significant differences between MIH and non-MIH children regarding breastfeeding duration. To our knowledge, none of the children and their mothers had ever been exposed to a main toxic hazard, or any dioxin or environmental accident.

Environmental pollution may be another onset factor for MIH. Every year 370,000 people in Europe die prematurely from diseases linked to environmental pollution [Commission of the European Communities, 2007]. Urban life in western world may be a factor for MIH as a North African study showed that MIH was rare (only 1.1%) in Libya [Fleita et al., 2006]. This prevalence was clearly lower than in comparable studies performed in Nordic countries (4% to 25%) [Koch et al., 1987; Alaluusua et al., 1996a; Jalevik et al., 2001a; Leppaniemi et al., 2001; Weerheijm et al., 2001b; Dietrich et al., 2003; Jasulaityte et al., 2003]. In the present study, prevalence of MIH was 14.9%. The present concern regarding the increasing level of tropospheric sulfur dioxide and other gases in urban areas is essentially due to their role in causing detrimental effects on human health and aquatic and terrestrial ecosystems. There were no statistical differences between MIH and non-MIH children, regarding where their mothers had lived for the last 5 years before gestation and breastfeeding periods and where children had lived up to date. Environmental factors and genetics should be examined further as among the 22 MIH children, there were 2 sister couples (n. 4) having the same dental findings.

Regarding fluoride; The Turkish Ministry of Health (2003) stated the F level in the areas where the present study children had been gestated and breast-fed is ‘negligible’ (< 0.25 mg/l). None of the mothers stated that they had used fluoride supplements. The F level in the 3 foreign cities where only two children had been gestated and breast-fed was over 0.8 mg/l.

Conclusions
MIH was common among a group of 7-9 years old children in Istanbul. Although the aetiology may be unclear, children with poor general health in the first 3 years or those who were exposed to certain environmental contaminants may be at risk for MIH.

Acknowledgements
We would like to thank Prof. Dr. Richard Welbury (Dept. of Paediatric Dentistry, School of Dentistry, Glasgow Dental Hospital and School, Glasgow, UK) for revising the English text, and Mr. Gunay Can (Istanbul) for his statistical expertise.

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In the group with intellectual disabilities, molar incisor hypomineralization was observed in eight (11.1%) children, i.e., six (75%) boys and two (25%) girls. In the control group, one (1.4%) boy had MIH. The prevalence of MIH in males and females was not significantly different in children with intellectual disabilities (Fisher’s exact test, p = 0.702) or in the control group (Fisher’s exact test, p = 1.000). Children with intellectual disabilities had significantly more MIH defects than children in the control group (Fisher’s exact test, p = 0.033).


Molar incisor hypomineralisation (MIH) is a type of enamel defect affecting, as the name suggests, the first molars and incisors in the permanent dentition. MIH is considered a worldwide problem and usually occurs in children under 10 years old. This developmental condition is caused by the lack of mineralisation of enamel during its maturation phase, due to interruption to the function of ameloblasts. Many factors have been suggested, such as genetics and medical problems during pregnancy, but only... Molar Incisor hypomineralisation prevalence has been reported with a wide variation of about 2.4 to 40.2% around the world. The lowest prevalence (2.5%) of MIH was observed among Chinese children and the highest (40.2%) prevalence was reported in Brazil (Table 1) [10,12-17]. In India, the first study was conducted. 45. The prevalence of Molar Incisor Hypomineralisation seems to be increasing in children all around the world. There are high chances of misdiagnosing MIH affected teeth in adults as it gets masked by caries. Prevalence and etiology of molar-incisor hypomineralization (MIH) in the city of Istanbul. Journal of Dental Sciences 13.4 (2018): 318-328. 17. Saitoh Masato., et al.