Factors involved in the aetiology of molar-incisor hypomineralisation (MIH)

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ABSTRACT. Aim This study aimed to collect more information on factors associated with molar-incisor hypomineralisation (MIH), which is a frequent developmental enamel defect with unknown aetiology. Materials and methods A questionnaire was sent to the parents of 45 children (average age 9.9 years; SD±2.02), 24 with affected first molars and 21 controls. The two groups of children were similar in terms of age, gender and living conditions. Questions were asked about the health of mother and child during pregnancy, the birth and health of the child up to age four years. Results Birth weight and length in the two groups of children were similar, as was the duration of breast- and/or bottle-feeding and the incidence of complications during pregnancy and birth. The children with MIH were ill more frequently during the first four years of life. Conclusion Knowledge of the type of disease that might be involved in the development of such molars is still inadequate, but there appears to be an association with pneumonia, otitis media and high fevers.

Keywords: Dental enamel abnormalities, Molar-Incisor Hypomineralisation (MIH), Enamel hypomineralisation

Introduction

Developmental defects of tooth enamel are not uncommon, both in the primary and permanent dentitions [Koch et al., 1987], and can be divided into hypomineralisation and hypoplasia, [Clarkson, 1989]. Enamel hypomineralisation can be observed visually because of a different translucence, that is known as opaque enamel. The opacity may be diffuse or sharply defined, whereas in cases of hypoplasia parts of the enamel are absent or very thin with smooth borders adjacent to normal tissue.

The causes of developmental enamel defects may be congenital, acquired or unknown. Congenital defects, such as amelogenesis imperfecta, have a genetic basis [Small, 1978; Pindborg, 1982]. In the case of acquired defects, the aetiology is very often unknown. Examples of an acquired defect with a known cause are trauma or an excessive use of fluoride. Molar-Incisor Hypomineralisation (MIH) is an example of an acquired defect of unknown aetiology [Weerheijm et al., 2001a].

In cases of MIH, there is a hypomineralised defect of the first permanent molars, frequently associated with affected incisors. In the Netherlands the term “cheese molar” has been used for this particular enamel defect, based on clinical appearance [Van Amerongen and Kreulen, 1995]. In the literature MIH is also known as non-fluoride enamel opacities, internal enamel hypoplasia, non-endemic mottling of enamel, opaque spots, idiopathic enamel opacities and enamel opacities [Koch et al., 1987]. Recently Weerheijm et al. [2001a] suggested the term MIH for this enamel defect to facilitate comparing researches related to these types of molars.

In appearance the enamel defects are creamy-white to yellowish-brown and show a clear border between affected and sound enamel [Suckling et al., 1989]. The porous, brittle enamel can easily crumble away (Fig. 1). Sometimes the loss of enamel can occur so rapidly after the eruption that it seems as if the enamel was initially not formed. But this post eruptive enamel breakdown has to be differentiated from hypoplasia. The borders to the normal enamel are irregular in the case of posteruptive enamel breakdown. Histological examination has shown that the yellowish-brown defects are more porous than the creamy-white ones and cover the entire thickness of the enamel. The creamy-white defects are located in the internal part of the enamel [Jälevik and Norén, 2000].
The number of affected permanent first molars per patient varies from 1 to 4 and the expression of the defects may vary from molar to molar. In some cases, apart from defects in the permanent first molars, opacities may be found in the upper and sometimes in the lower incisors. The risk of defects to the upper incisors appears to increase with the number of first permanent molars that have been affected [Weerheijm et al., 2001b]. The enamel defects in incisors are mostly less serious than in molars because chewing forces are absent.

In the literature various causes for MIH, including respiratory tract problems, perinatal complications and dioxins in the mother milk have been suggested [Van Amerongen and Kreulen, 1995; Alaluusua et al., 1999]. Other causative factors mentioned are oxygen starvation of the child combined with a low birth weight, and environmental conditions [Johnson et al., 1984; Koch et al., 1987]. There are also suggestions that calcium and phosphate metabolic disorders might be a cause [Jontel and Linde, 1986]. Calcium and phosphate depletion can be the result of nutritional deficiencies, problems with the gastrointestinal tract, diarrhoea and high fever. The distribution of MIH in the population is non-random and seems to be child-related [Weerheijm et al., 2000b]. The cause might be a combination of factors.

Formation of enamel on the permanent first molars and the incisors is initiated shortly before birth and occurs over the first four years of life. The cause of abnormal enamel formation must then be present in this particular period. In order to be able to trace high-risk children earlier, it is essential to improve our understanding of this enamel defect. The aim of this study was to evaluate the risk factors and possible causes of MIH using a retrospective questionnaire based on causes reported earlier in the literature.

Materials and methods

A questionnaire was sent to the parents of 24 children with MIH (study group) and of 21 children with normal molars (control group) in the Amsterdam area. The parents signed an informed consent form permitting the use of data on their children. The children (average age: 9.9 years, SD ±2.02) were matched for age, gender and postal code. Those children in the study group had at least two first permanent molars with serious enamel defects. The criterion to include a first molar of a child in the study group was the presence of post-eruptive enamel loss across a large surface area of the molar. Due to the severe defects many of these molars were heavily restored. The treatment modalities included multisurface restorations (often including cusp capping), stainless steel crowns or extractions in consultation with the orthodontist. None of the children in the control group had MIH.

The questionnaire contained 55 questions, of which 25 were related to the medical history of the child in their first four years of age: ten were general questions, eight concerned the medical condition of the mother during pregnancy, five were about the specifics of childbirth relating to the mother and, finally, seven were about the specifics of childbirth relating to the child. The parents were asked only to record illnesses confirmed at that time by the family doctor. The medical histories of the children were reviewed retrospectively at intervals of 3 months across the first four years of life.

All data were processed using SPSS. To calculate the differences between the two groups in terms of birth weight and birth length t-tests were used and for the differences in the period of breast- and/or bottle-feeding the Mann Whitney U-test was used. The difference in number of illnesses between the two groups was checked using the Mann Whitney U-test, while the specific disease incidence between the two groups was checked using the Pearson’s chi-square test.

Results

No significant differences were found between the study and control groups in terms of birth weight, birth length and the period of breast- and/or bottle-feeding (p > 0.05). In all cases both
mother and child were healthy during pregnancy. There were no significant differences between the two groups relating to perinatal complications, such as a difficult and lengthy birth, premature birth or oxygen starvation ($p > 0.05$).

Not all parents were able to relate exactly what illnesses had occurred in each 3-month interval, but most could indicate it with adequate accuracy on a larger time scale. Figure 2 shows that in the first four years of life children with MIH were ill more frequently than the children without such molars (Mann Whitney U test: $z = -3.26$, $p = 0.001$). In addition, children with MIH have suffered a greater variation of illnesses (mean: 2.5; SD ±1.8) than children with normal molars (mean: 0.9; SD ±0.7).

Table 1 lists the most frequent illnesses which occurred in the study group and the control group. A difference in illnesses is apparent between the two groups. Significant differences were found for otitis media, ($\chi^2 = 5.47$, $p = 0.019$), pneumonia ($\chi^2 = 4.92$, $p = 0.027$) and high fever ($\chi^2 = 3.84$, $p = 0.05$).

**Discussion**

In the literature, oxygen starvation has been described as a possible cause of MIH [Johnson et al., 1984; Koch et al., 1987; Van Amerongen and

![Figure 2](image-url)  
**Fig. 2** - Number of diseases during the first four years in children with MIH and their controls.

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Patient group (n = 24)</th>
<th>Percentage</th>
<th>Control group (n = 21)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Otitis media</td>
<td>14</td>
<td>58%</td>
<td>5</td>
<td>24%</td>
</tr>
<tr>
<td>Bladder infection</td>
<td>2</td>
<td>8%</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>3</td>
<td>13%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Measles</td>
<td>2</td>
<td>8%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Chicken pox</td>
<td>19</td>
<td>79%</td>
<td>8</td>
<td>38%</td>
</tr>
<tr>
<td>Whooping cough</td>
<td>1</td>
<td>4%</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>5</td>
<td>21%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Airway infection</td>
<td>2</td>
<td>8%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Cara*</td>
<td>3</td>
<td>13%</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>Asthma</td>
<td>2</td>
<td>8%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Bronchitis</td>
<td>5</td>
<td>21%</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>Gastrointestinal infection</td>
<td>1</td>
<td>4%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>High fever</td>
<td>4</td>
<td>17%</td>
<td>0</td>
<td>0%</td>
</tr>
</tbody>
</table>

*non-specific respiratory disease

**Table 1** - The most frequent illnesses which occurred in the study group and the control group.
Kreulen, 1995] and may result from perinatal complications or from diseases that affect the oxygen balance in the head and neck area, such as respiratory tract infections, asthma, pneumonia, bronchitis and otitis media. In the current study, no significant difference in terms of perinatal complications was found between children with and without MIH. Otitis media, pneumonia and high fever episodes were, however, more frequent in the group of children with MIH, which can be considered as a support for this theoretical model.

The current study did not find any differences between the groups with regard to nutritional and/or gastrointestinal problems as suggested by Jontel and Linde [1986]. Prolonged breast-feeding was found to be associated with MIH in the Finnish study by A lauluusa et al. [1996]. Such an association could not be demonstrated in the present study. Both in this study and in a recent report by Leppäniemi et al., [2001], no differences in the period of breast- and/or bottle-feeding were found between children with and without MIH.

The use of antibiotics has also been suggested as a cause of MIH. No reference for this possible effect can be found in the literature, however, apart from discoloration due to tetracyclines [Schuurs, 1999]. In the current study no evidence for this link has been found either. Children taking prescribed antibiotics are usually seriously ill. It might be more probable that the actual disease, from which the child is suffering, is the cause of the developmental enamel defect.

The children in this study have been matched for age and living area. One of the reasons for this was that environmental conditions have been mentioned in the literature as a possible cause of MIH [Koch et al., 1987]. Because of the correction for living area in the present study, it is impossible to say anything about an influence of the environment on the development of MIH.

At the outset of the study, the questions were subdivided to relate to three months intervals. This was done in order to find a possible correlation between the severity of the "cheese molars" and the location of the enamel defect on the one hand, and the age and the length of time the child was ill on the other. As this study was retrospective, most parents found it difficult to accurately indicate the three months intervals in which their child had been ill. Therefore, these data have not been worked out in detail in the results section. The drawback of retrospective studies is that data are collected after the fact and rely on the memory of the people concerned, in this case the parents. It would be better to monitor a group of children medically, as a prospective study, from the date of birth to the time of eruption of the permanent first molars: this would make the study less dependent on the memory of the parents, and exclude the risk of retrospective attribution of a possible cause.

The aetiology of MIH still remains very unclear, partly because the defect is referred to under various names in the literature. For example, Jan and Vrbic [2000] in a recent paper lumped together MIH and enamel defects on permanent molars, premolars and canines. The use of a commonly accepted name for the defect would make it simpler to compare various study results [Weerheijm et al., 2001a].

When our knowledge of the development of MIH has been improved, it will be possible to trace patients at risk much earlier and initiate preventive treatment more rapidly. In the current study, children with MIH fell ill more frequently in the first four years of life than children in the same age group with normal molars. It appears that diseases concerning the head and neck area were relatively frequent. Therefore, in children with repeated illnesses in the first four years it seems useful to increase the frequency of dental check-ups during the period when the permanent first molars erupt. This would be a means to detect the clinical symptoms as early as possible.

MIH are fragile, and caries can develop very easily. This problem is aggravated because children tend to avoid the sensitive molars when brushing their teeth. If molars show signs of opacities and/or post-eruptive breakdown, a child should be seen every three months up until the time when the six-year molars have completely erupted. In order to minimise the loss of enamel and any damage due to caries, both preventive and interceptive treatment is required (fluoride application and glassionomer sealants). Figure 3 shows how rapidly substantial defects due to post-eruptive enamel loss and caries may emerge. The presence of MIH not only requires the dentist to identify problems at the earliest opportunity (Fig. 4), but also to thoroughly explain the problems to the parent and child. As only the permanent first molars (and sometimes the incisors) are affected by the developmental enamel defect, the parents can be reassured with respect to the quality of the remaining teeth that have not yet erupted.

Further research is required to gain more insight into the nature and extent of these
diseases. Prospective follow-up studies should focus on the medical situation of the child in the first years of life.

**Conclusion**

Children with MIH have experienced more diseases after birth. The type of disease is not clear but there seems to be a trend for otitis media, pneumonia and high fever to be related to MIH.

**References**