Molar Incisor Hypomineralisation (MIH)

K.L. WEERHEIJM

ABSTRACT. Review. Molar Incisor Hypomineralisation (MIH) is defined as a hypomineralisation of systemic origin of one to four permanent first molars frequently associated with affected incisors. MIH molars are fragile and caries can develop very easily in those molars. Although MIH molars are well known by paediatric dentists and their occurrence is related in severe cases to major clinical problems, only limited data of the size of the problem are available. The prevalence of MIH ranges in the literature from about 3.6 to 25% and seems to differ in certain regions and birth cohorts. Unfortunately more complete comparable valid data are lacking at the moment. It seems that several aetioloical factors can cause the enamel defects and that their occurrence is child related. Conclusion. For children with repeated illnesses in the first years after birth and children with opacities on erupted molars or incisors it seems useful to increase the frequency of dental check-ups during the period of erupting first permanent molars.

KEYWORDS: Dental enamel abnormalities, Molar Incisor Hypomineralisation (MIH), Enamel hypomineralisation.

Introduction

Besides hereditary enamel developmental disorders such as amelogenesis imperfecta, other acquired enamel disorders with known cause, such as fluorosis or Turner’s teeth, can be found. For some enamel (idiopathie) disorders the cause is still unknown. An example of the latter is the first permanent molars with demineralised defects (Fig. 1). In the literature, a lot of different descriptions (Table 1) can be found for molars with white yellow or yellow brown demarcated opacities sometimes even in combination with enamel breakdown (Fig. 2) [Koch et al., 1987; Alaluusua et al., 1996b, 1999; Jälevik and Norén, 2000; Leppäniemi et al., 2001; Weerheijm et al., 2001a; Höltä et al., 2001].

In order to focus the research concerning such molars, Weerheijm et al. [2001b] defined the phenomenon as a hypomineralisation of systemic origin of one to four permanent first molars frequently associated with affected incisors and suggested the name Molar Incisor Hypomineralisation (MIH) for it. In this article the clinical presentation, implications, prevalence and aetiology of MIH will be described.

Clinical presentation

In MIH clinically demarcated opacities of different colour, that is opacities with a clear and distinct border to the adjacent enamel, can be seen. Figure 3 shows a demarcated yellow opacity on the palatal surface of a maxillary first permanent molar. Clinically a hypomineralisation can be seen as an abnormality in the translucency of the enamel. The enamel of MIH molars looks soft and porous and has the appearance of discoloured chalk or old Dutch cheese. Hence the condition has been termed ‘cheesy molar’. Some opacities have significant subsurface porosity, leading to breakdown of the surface after eruption (Fig. 4).

The expression of the phenomenon can vary in severity between patients but also within a mouth. This means that not all molars will be affected to the same extent. Figures 5a and 5b show the mandible and maxilla of a child with MIH. The asymmetrical occurrence of MIH in the molars can be clearly noticed in these two figures. When a severe defect is found within a subject it is likely that the contralateral tooth is also affected.

Though not always occurring, the hypomineralised porous enamel can chip off easily, leading to unprotected dentine and also to an unexpectedly rapid caries development. The loss of enamel can occur immediately after eruption as the result of masticatory forces on the fragile enamel and has to be discerned from hypoplasia. Enamel hypoplasia is...
Fig. 1 - Maxillary molar with large opaque and discoloured distal, occlusal and palatal areas with posteruptive enamel loss and cavity formation.

Fig. 2 - Maxillary molar with white opacity and posteruptive enamel breakdown at the mesio-palatal surface.

Fig. 3 - Maxillary molar with demarcated yellow opacity on the palatal surface.

Fig. 4 - Mandibular first permanent molar with occlusal sealant and loss of enamel at the buccal surface. Notice the yellow and yellow/brown opacities on the occlusal surface and at the borders of the areas with the enamel loss.

Fig. 5 - a) maxilla of a boy with MIH. Notice the large affected areas with enamel loss and cavity formation in the 16 and the yellow opacities with the beginning of enamel loss in the 26; b) mandible of the same boy as in Figure 5a. Besides opacities, enamel loss is larger and rapid cavity formation can be noticed in the 36 while only a mild opacity is present on the lingual surface of the 46.
a consequence of a deficient enamel matrix formation (due to a disturbance in the ameloblast function during the secretory phase), which appears as a superficial defect resulting from reduced enamel thickness. In cases of hypoplasia the borders of the normal enamel are mostly smooth, while in MIH molars, where the enamel matrix is initially formed to its normal shape, the borders of the normal enamel are irregular when posteruptive enamel loss had occurred. Figure 6a shows a MIH molar during eruption while in 6b the same molar is shown three months later. Notice the disappearance of the enamel initially present at the disto-buccal cusp. Such enamel opacity, broken secondarily due to trauma from masticatory forces, is often incorrectly referred to as enamel hypoplasia. Histologically the appearance of post eruptive enamel loss differs from that of hypoplasia [Norén et al., 1994]. It seems more appropriate to indicate such defects as Posteruptive Enamel Breakdown (PEB).

In MIH, the lesions in the first permanent molars are often seen together with those in the maxillary and, more rarely, the mandibular incisors (Fig. 7). These findings indicate a systemic upset during the first years of a child’s life, more precisely during the period in which the crowns of permanent first molars and incisors are mineralised. In general, the defects of the incisors are milder than those of the molars. As masticatory forces on the opacities in incisors are absent, the enamel substance does not disintegrate so easily after eruption. When more molars are affected, the relative risk of incisors showing opacities is increased [Koch et al., 1987; Jälevik et al., 2001; Weerheijm et al., 2001a]. For the time being opacities on erupting incisors should be considered as a risk factor for the occurrence of MIH molars.

**TABLE 1 - Outline of possible descriptions for Molar Incisor Hypomineralisation (MIH) in permanent molars.**

<table>
<thead>
<tr>
<th>Description</th>
<th>Description</th>
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<tbody>
<tr>
<td>Hypomineralised permanent first molars</td>
<td>Idiopathic enamel hypomineralisation</td>
</tr>
<tr>
<td>Non-fluoride hypomineralisation</td>
<td>Non-fluoride enamel opacities</td>
</tr>
<tr>
<td>Opaque spots</td>
<td>Idiopathic enamel opacities</td>
</tr>
<tr>
<td>Internal enamel hypoplasia</td>
<td>Non-endemic mottling of enamel</td>
</tr>
<tr>
<td>Enamel opacities</td>
<td>Cheese molars</td>
</tr>
</tbody>
</table>
Clinical implications

Clinically, MIH molars can create discomfort to the child. The affected teeth can be very sensitive to a current of air, cold or warm. Even with enamel that has not disintegrated, mechanical stimuli, for instance tooth brushing, may instigate toothache in these teeth. A dentist has to pay serious attention to this sensitivity. If a child tells us that it hurts, for example during restorative procedures, after proper local analgesia has been provided, the dentist has to improve the comfort for the child. The reasons for the increased sensitivity of MIH molars and sometimes the incisors is as yet known, but primarily it does seem to be a physiological one based on operant conditioning by repeated small painful stimuli. Dentists have to keep in mind that repeated small stimuli (normally not considered as painful) could create serious pain in these children.

Besides the difficulty that dentists can face in obtaining adequate analgesia for a MIH molar there is also the problem that unexpectedly fast caries development in the erupting first permanent molar can be unpleasant. MIH molars are fragile, and caries can develop very easily. This problem is aggravated because the children tend to avoid the sensitive molars when brushing their teeth, leading to increased stagnation of food and plaque. The fast caries progression can clinically mask the reason behind the susceptibility for caries (hypomineralisation of the enamel) in these molars (Fig. 8). Furthermore it is unpredictable which part of the apparently healthy, but hypomineralyzed enamel, will disintegrate in due course of time. It seems that especially during the first year after eruption the opacities are vulnerable. Jälevik and Klingberg [2002] found that, compared with normal molars, MIH molars need ten times more treatment time.

The presence of MIH molars not only requires us as dentists to identify problems at the earliest opportunity, but we also have to explain the problems thoroughly to the parents and child. As only the first permanent 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. The children with MIH molars or opacities on the front teeth should be monitored carefully until all four permanent first molars have erupted.

Prevalence

Today only limited data are available on the prevalence of MIH, as comparable and representative prevalence studies are lacking [Weerheijm et al., 2003]. A recent questionnaire investigation among members of the European Academy of Paediatric Dentistry (EAPD) showed that paediatric dentists in Europe are aware of MIH and that the majority considers it to be a clinical problem [Weerheijm and Mejäre, in press].

Available prevalence data, mostly from Northern Europe, range from 3.6 to 25%. In all cases the areas reported on were low in water fluoride. Koch et al. [1987] looked at Swedish children between 8 and 13 years of age born between 1966 and 1974. They found a range in prevalence of 3.6% to 15.4% depending on the year of birth of the children. A peak value of 15.4% was found in children born in 1970. In those children the maxillary incisors were affected more severely and showed signs of disintegration, while 60% of these children had four affected molars. Jälevik et al. [2001] looked at 8 year old Swedish children born in 1990 and found molars affected with MIH in about 18.4% of the children. Of these children 6.5% had severe, 5% moderate and 7% mild defects on their molars. This study included many

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**Fig. 7** - Photograph showing a yellow opacity at the buccal surface of the second mandibular incisor.

**Fig. 8** - Rapid caries progression in a affected permanent molar. Notice the opaque areas next to the cavity border.
recently erupted molars so probably some of the moderate cases would become severe after more time in the mouth. Children with severe defects also had a larger number of affected teeth, molars as well as incisors in this study.

Leppäniemi et al. [2001] found a comparable prevalence of 19.3% in 7-13 year old Finnish children born between 1983 and 1989. Alaluusua et al. [1996a, 1996b] recorded percentages of 25% and 17% respectively for 12 (born in 1981) and 6-7 year (born in 1987) old Finnish children whose mothers had been encouraged to practice extensive and prolonged breast feeding (longer than 8 months). Compared with these figures Weerheijm et al. [2001a] found a lower percentage of 9.7% in a group of 11 year old Dutch children born in 1988. Of the children where MIH molars were found, 79% had two or more of such molars, suggesting a child related cause.

At the moment there is no conclusive evidence that maxillary molars are more susceptible for MIH than mandibular ones or vice-versa. Leppäniemi et al. [2001] found more MIH molars in the upper jaw compared to the lower jaw. Although not statistically different, Jälevik et al. [2001] found the opposite of this in their study and in that of Weerheijm et al. [2001a] the frequency of MIH molars was evenly divided among the molar type.

### Aetiology

The combination of affected molars together with incisors suggests that in the case of MIH we are dealing with a specific influence on the development of enamel during a limited period of time. In Table 2 the chronological tooth development for first permanent molars and incisors can be found. Enamel formation is a sensitive process, which can be divided into several stages. The secretory phase, where partially mineralised enamel is deposited to the whole enamel thickness, is followed by the maturation phase. In the latter the organic material and water in the enamel are removed to allow an additional influx of mineral. A disturbance occurring during the maturation phase will be clinically visible as an enamel opacity, which suggests that in the case of MIH the ameloblasts are affected in the (early) maturation stage. Jälevik and Norén [2000] found in their study that it seemed that some of the ameloblast cells are irreversibly damaged. Clinically these defects are observable as yellow or yellow brown demarcated opacities. Histologically these opacities show more porosities and are mostly situated through the entire bulk of enamel. Other ameloblasts have probably the potential to recover after the attack. Clinically such defects are observable as creamy yellow or whitish cream demarcated opacities with a bright and hard surface. These opacities are in most cases situated in the inner part of the enamel.

In the literature a number of possible causes for MIH are mentioned, such as environmental changes [Koch et al., 1987; Hölltä et al., 2001]. Others have suggested that exposure to dioxine by prolonged breast-feeding could lead to an increase in the risk of MIH [Alaluusua et al., 1996a, 1996b, 1999]. Also respiratory diseases and oxygen shortage of the ameloblasts have been mentioned [Bronckers, 1984; van Amerongen and Kreulen, 1995; Jälevik and Norén, 2000]. Similarly an oxygen shortage combined with low birth weight [Johnsen, 1984].

<table>
<thead>
<tr>
<th>Tooth</th>
<th>Maxilla</th>
<th>Mandibula</th>
<th>Maxilla</th>
<th>Mandibula</th>
<th>Maxilla</th>
<th>Mandibula</th>
</tr>
</thead>
<tbody>
<tr>
<td>I₁</td>
<td>3 months</td>
<td>3 months</td>
<td>4⁵/₈ year</td>
<td>3³/₈ year</td>
<td>7³/₈ year</td>
<td>6⁵/₈ year</td>
</tr>
<tr>
<td>I₂</td>
<td>11 months</td>
<td>3 months</td>
<td>5³/₈ year</td>
<td>4 year</td>
<td>8 year</td>
<td>7³/₈ year</td>
</tr>
<tr>
<td>M₁</td>
<td>32 weeks in utero</td>
<td>32 weeks in utero</td>
<td>4⁵/₈ year</td>
<td>3³/₈ year</td>
<td>6⁵/₈ year</td>
<td>6 year</td>
</tr>
</tbody>
</table>

I₁ = permanent central incisor; I₂ = permanent lateral incisor; M₁ = first permanent molar

**Table 2 - Chronology of tooth development of permanent molars and incisors as described by Proffit [1993].**
disturbances in the calcium/phosphate metabolism [Jontells and Lindhe, 1986] and more frequent childhood diseases with high fever [Jälevik, 2000; Beentjes, 2002] could all be possible.

Sometimes the use of antibiotics has been mentioned, but as antibiotics are in most cases related to the occurrence of a disease, it is difficult to distinguish whether the association with MIH was caused by the antibiotic use or by the illness itself. Also, vaccines given during early childhood have been suggested as a possible cause, however at the moment no data are available to substantiate this.

Most of the current studies are performed retrospectively. The drawback of retrospective studies is that data are collected a long time after an event took place and rely on the memory of people concerned, which in MIH studies this is mostly the parents. Prospective studies, starting around the date of birth to the time of eruption of the permanent molars, are needed to clarify the factors and mechanisms behind the defects [Beentjes et al., 2002].

**Conclusion**

At present it seems that unknown contributing factors are involved in the aetiology of MIH together with a number of possible causes. For children with repeated illnesses in the first years after birth and children with opacities on erupted molars or incisors it is strongly suggested that it is useful to increase the frequency of dental check-ups during the period of erupting first permanent molars.

**References**


