Molar Incisor Hypomineralization (MIH) - a review

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Abstract

MIH was defined by Weerheijm (2001) as "hypomineralisation of systemic origin of 1-4 permanent first molars, frequently associated with affected incisors". The prevalence of MIH varies between 2.8% and 25%, dependent upon the study. At their sixth congress in 2003, The European Association of pediatric dentistry defined criteria for diagnosis of the phenomena. It included the presence of demarcated opacity, posteruptive enamel breakdown, atypical restoration, extracted molar due to MIH and unerupted teeth. According to the teeth involved and to the time of the crown formation, researches focused on environmental and systemic conditions as possible reasons for MIH.Theetiologies were divided into three groups: pre/peri and neonatal problems. The clinical implications include highly sensitive teeth, difficulty to achieve adequate anesthesia, behavioral problems and anxiety, rapid progression of caries and the esthetic implications. A six step approach to management was described suggested: risk identification, early diagnosis, remineralization and desensitization, prevention of caries and posteruption breakdown, restorations and extractions and finally maintenance.

Key words: Hypomineralization, Hypoplasia, Demarcated opacities

Introduction

Dental development and mineralization in humans start before birth and continues to adolescence when the permanent molars complete their mineralization. The first sign of tooth mineralization is seen in the primary lower incisors in the beginning of the second trimester of pregnancy and completed around three months post partum. The first permanent molar is the first tooth in the permanent dentition to mineralize, a process that starts around birth and is completed at approximately three years of age[1].

Tooth enamel is a tissue of epithelial origin, incapable of being regenerated after its formation. As a result, injuries that occur during this period are permanently recorded on its surface, characterizing the Developmental Defects of Enamel (DDE)[2,3].

Tooth development may be influenced by various factors (such as febrile illness, antibiotic use and excessive fluoride intake) during, before or after birth[4]. Depending on the timing and duration of these factors, teeth may undergo various pathological conditions. Factors that disturb the ameloblasts during the secretory stage cause restriction of crystal elongation and result in pathologically thin orhypoplastic enamel. Disturbance during the transitional and maturation stage of amelogenes results in pathologically soft (hypomatured, hypomineralised) enamel of normal thickness[3].

Enamel hypomineralisation of systemic origin of First Permanent Molars(FPMs)and frequently also of incisors is known as Molar-Incisor Hypomineralisation (MIH)[5]. While the enamel is affected to an extent ranging from mild to severe, changes in dentine seem to be mild. In a study by Heijs et al(2007)[6], no morphological changes in the dentine were found by polarized light microscopy except for the presence of interglobular dentine under the affected enamel.

The purpose of this review is to provide comprehensive knowledge on MIH necessary for the dental professional to deal with the clinical and psychological needs of each patient, in order to provide a more satisfactory quality care.

Definition

Weerheijm et al. [2001] suggested the terminology of MIH to describe the clinical picture of a hypomineralisation of systemic origin of one or more of the four permanent first molars, as well as any associated and affected incisors[5].

Nomenclature

At the European Academy of Pediatric Dentistry Congress in 2000 there were six presentations that described the same type of developmental defects of dental enamel affecting the First Permanent Molars (FPM). These reports called the condition, Non-endemic stained enamel (Jakson 1961)[7], Idiopathic hypomineralization of the enamel of the first molars (Koch et al 1987)[8], Hypomineralized FPM (Beentjes 2000)[9], idiopathic enamel hypomineralization in FPM (Jalevik, Noren JG 2000)[10], Non fluoride hypomineralization in FPM (Leppaniemi A et al 2001)[11] and cheese molars (Weerheijm et al 2001)[12] because clinically the lesion resembles the colour and consistency of Dutch cheese. The congress highlighted the fact that multiple names for the same condition could lead to confusions. Weerheijm et al (2001)[12] stated that when discussing these developmental defects of dental enamel, it would be described to use one name, one that made no reference to any possible etiology and thus suggested 'Molar Incisor Hypomineralization (MIH) as a name for this condition.

Etiology

A variety of environmental factors including those of systemic nature occurring in the pre, peri or post-natal period have been postulated as contributing to or causing MIH. A relationship, however, has not been established between MIH and any particular causative fac-

tor(s) even with the availability of dental and medical history records[13,14].

Prenatal period (From 0 to 36-38 weeks in utero)

There was some evidence that medical problems during pregnancy were associated with MIH. In a study by Freden H and Gronvik M (1980)[15] a specific illness, urinary infection during the last trimester was associated with MIH-like lesions. In other studies by Freden H and Gronvik M (1980)[15], specific diseases were not associated with MIH but however Whatling R, Fearne JM (2008)[16] and Lygidakis NA et al (2008)[17] reported that medical problems were more common in mothers of MIH children than in those mothers whose children did not have MIH.

It was suggested that maternal disorders such as cardiologic diseases, infections of the urinary tract, A and D vitamin deficits, anemia, toxicity, diabetes mellitus and rubella embryopathy during pregnancy might result in developmental enamel defects in the child[18].

Perinatal period (From birth to 28 days after birth):

In the perinatal period different medical conditions alone or in combination may affect the welfare of a child. In a study by Lygidakis NA et al(2008)[17] showed that the most common perinatal problems/ conditions were Caesarian section, prolonged delivery, premature birth and twinning. MIH was more frequently seen in the study group than in the control group children. On the other hand, in studies of Whatling R and FearneJM (2008)[16] and Diedrich G (2003)[19] perinatal problems could not be linked with MIH.

Postnatal period (From 29 days to 4 years of age)

In a cohort study, Jalevik B et al (2001)[20] showed a correlation between diseases at 0-1 year of age and MIH was only found in boys. A case control study by Beentjes VE (2002)[21] concluded children with MIH had illnesses during the first 4 years of life than children without. However, a study by Tapias-Ledesma MA et al (2003)[22] found that frequent paediatric care in each of the first 4 years showed a strong correlation with dental enamel defects in the FPMs. Furthermore, in a study by Lygidakis NA (2008)[17]postnatal problems during the first year were clearly more common in children with MIH than in those without[9]. In a study by Tapias-Ledesma MA et al (2003)[22], children with MIH had a disease history from the first 3 years of life more often than children without MIH.

Postnatal factors include childhood illness, antibiotics like amoxicillin and erythromycin, different environmental factors, breast feeding and fluorides.

Prevalence

The prevalence of MIH in European countries range from 3.58-21.8 %[17,20, 23,24]. As far as Asia is concerned the prevalence rate was estimated to be the least (2.8%)[25] in Hong Kong and highest (18.6%) in Iraq[26]. A study by Da costa-silva et al (2010)[27] from South America shows a prevalence of 19.8%.

The prevalence of MIH in Northern India has been reported as 6.31% (Mittal NP et al 2003)[28] and 9.46% (Bhaskar SA et al 2014)[20], 5.6 % (Mishra A et al 2016)[30] and study from western part of India by (Parikh DR et al 2012)[31] shows a prevalence of 9.2%. Prevalence of MIH from Southern part of India ranges from 0.48%-9.7%.[32,33,34,35,36]. Studies from Africa shows a prevalence of 2.9 % (Fteita D et al 2016)[37] and 17.7 % (Oyedele TA et al 2015)[38].

The difference in prevalence amongst various studies may be attributed to disparities in sample size, diagnostic criteria, population group examined and use of different indices (Alaluusua et al (1996a)[30], Aine et al (2000)40, Arrow (2008)41, Weerheijm and Mejare (2003)[42].

Clinical presentations

MIH is a hypomineralized defect of the first permanent molars, frequently associated with affected incisors. The number of affected first permanent molars per patient varies from one to four and expression of the defects may vary from molar to molar[39].

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. 1). 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.

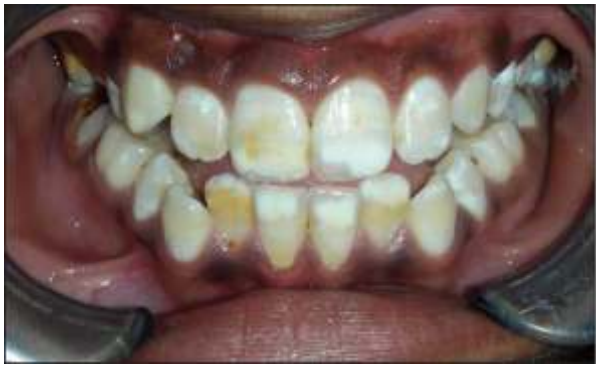


Figure 1

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[10,9,39,43]. For the time being opacities on erupting incisors should be considered as a risk factor for the occurrence of MIH molars.

Clinically, the hypomineralized enamel can be soft, porous and look like discoloured chalk or old Dutch cheese. The enamel defects can vary from white to yellow or brownish but they always show a sharp demarcation between the affected and sound enamel (Figure 2).

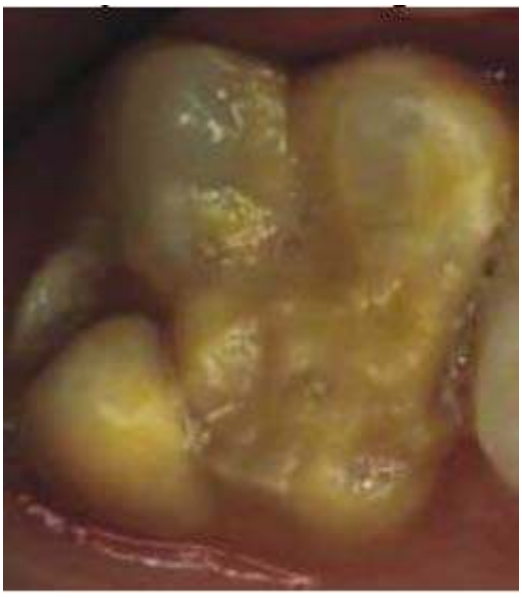


Figure 2

The porous, brittle enamel can easily chip off under the masticatory forces, leading to unprotected dentine and also to an unexpectedly rapid caries development. Sometimes, the loss of enamel (Posteruptive Enamel Breakdown) can occur so rapidly after eruption that it seems as if the enamel was not formed initially.

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[44].

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. 3).

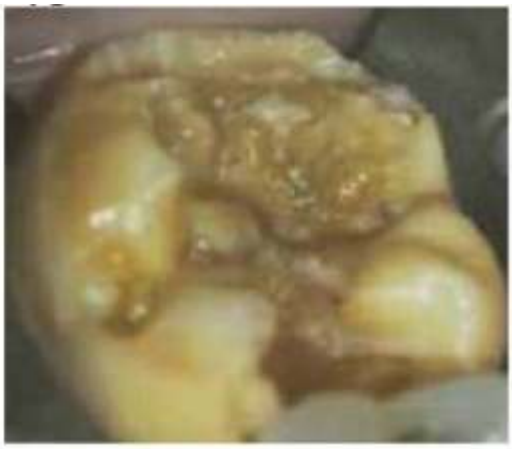


Figure 3

Differential Diagnosis

Hypoplasia- In hypoplasia, the borders to the normal enamel are mostly regular and smooth, whereas in MIH-associated-enamel substance loss, the enamel edges are sharp and irregular where the enamel has chipped off[45].

Amelogenesis imperfect (AI)-In MIH, the appearance of the defects is more asymmetrical, whilst in AI, all permanent and primary teeth tend to be affected (i.e. generalized involvement). Moreover, family history and history of systemic disorders/illnesses are still crucial discriminative factors[46,47]

Administration of tetracycline during pregnancy and to children under 6 causes changes to grey and yellowish color of temporary and permanent teeth. If the dose is high, hypoplastic changes occur in the enamel. Clinical studies and patient history allow us to clearly distinguish changes in biological structure due to tetracycline or fluoride from Molar-Incisor Hypomineralisation[49].

Diagnosis

The diagnostic criteria proposed by Weerheijm (2003)[42] is as follows:

Permanent first molars and incisors. One to all four permanent first molars (FPM) shows hypomineralisation of the enamel. Simultaneously, the permanent incisors can be affected. To diagnose MIH, at least one FPM has to be affected. The defects can also be seen in second primary molars, incisors and the tip of the canines. Where there are more molars and incisors affected the more severe is the defect.

Demarcated opacities. The affected teeth show clearly demarcated opacities at the occlusal and buccal part of the crown. The defects vary in colour and size. The colour can be white, creamy or yellow to brownish. The defect can be negligible or comprise the major part of the crown. It is recommended that defects less than 1 mm are not to be reported.

Enamel disintegration. The degree of porosity of the hypomineralised opaque areas varies. Severely affected enamel subjected to masticatory forces soon breaks down, leading to unprotected dentine and rapid caries development.

Atypical restorations. FPM and incisors with restorations revealing similar extensions as MIH are recommended to be judged as affected.

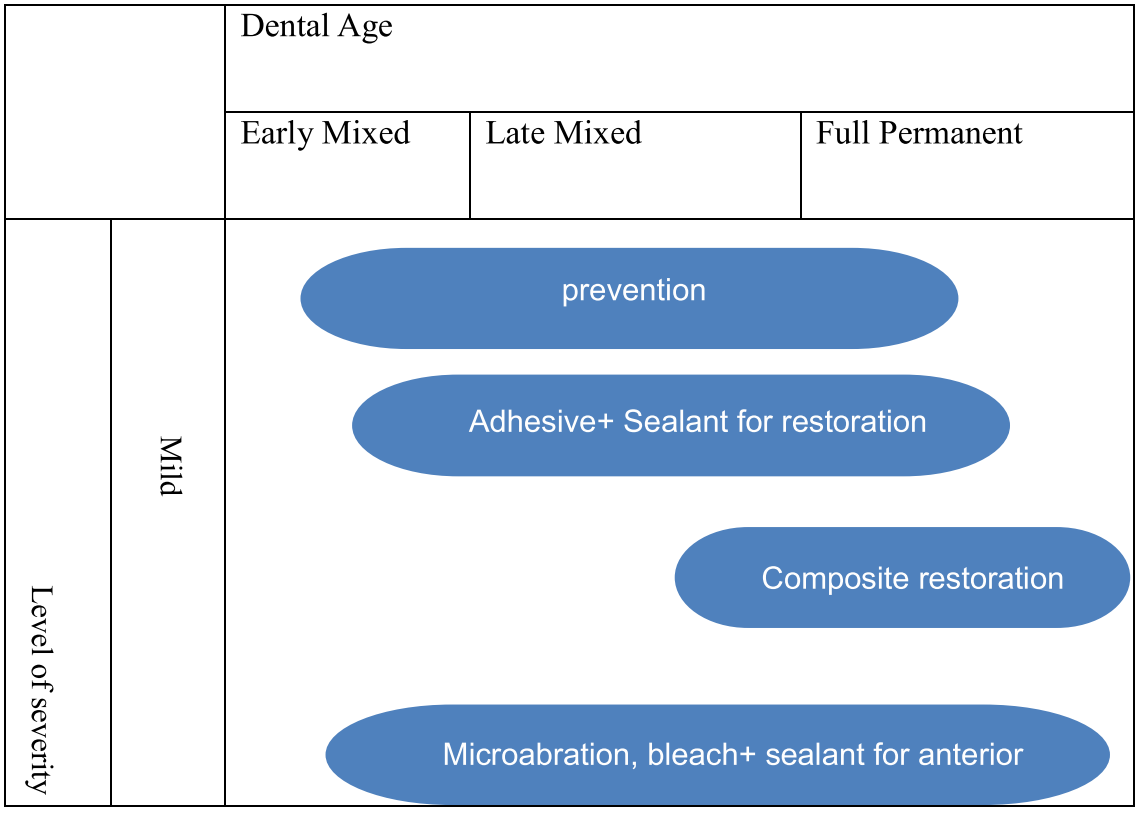
Tooth sensitivity. The affected teeth may be reported by frequent as sensitive, ranging from a mild response to external stimuli to spontaneous hypersensitivity; these teeth are usually difficult to anaesthetize.

Extracted teeth. Extracted teeth can be defined as having MIH only in cases where there are notes in the records or demarcated opacities on the other FPM. Otherwise it is not possible to diagnose MIH.

Management

A diagrammatic summary of possible factors interacting for each treatment modality according to the severity of the condition at a particular dental age, is shown in Figure 4. For example, prevention is very important at an early developmental age because the defective tooth is more likely to have caries and post-eruptive breakdown due to its increased porosity. However, in later development stages, although it is still important, the tooth becomes more mature and if prevention

works in an earlier phase of development and the tooth remains intact, the relative importance of prevention becomes less comparative to the necessity of restorative treatment. As the growth of a child is continuous, the dental age in Figure 1 is in reality a continuum rather than in discrete stages of development. The relative amplitude indicates the appropriateness of the particular treatment modality as the treatment of choice. Finally, this guide should be used in conjunction with other factors such as a child is behavioral management and presence of other anomalies[50].



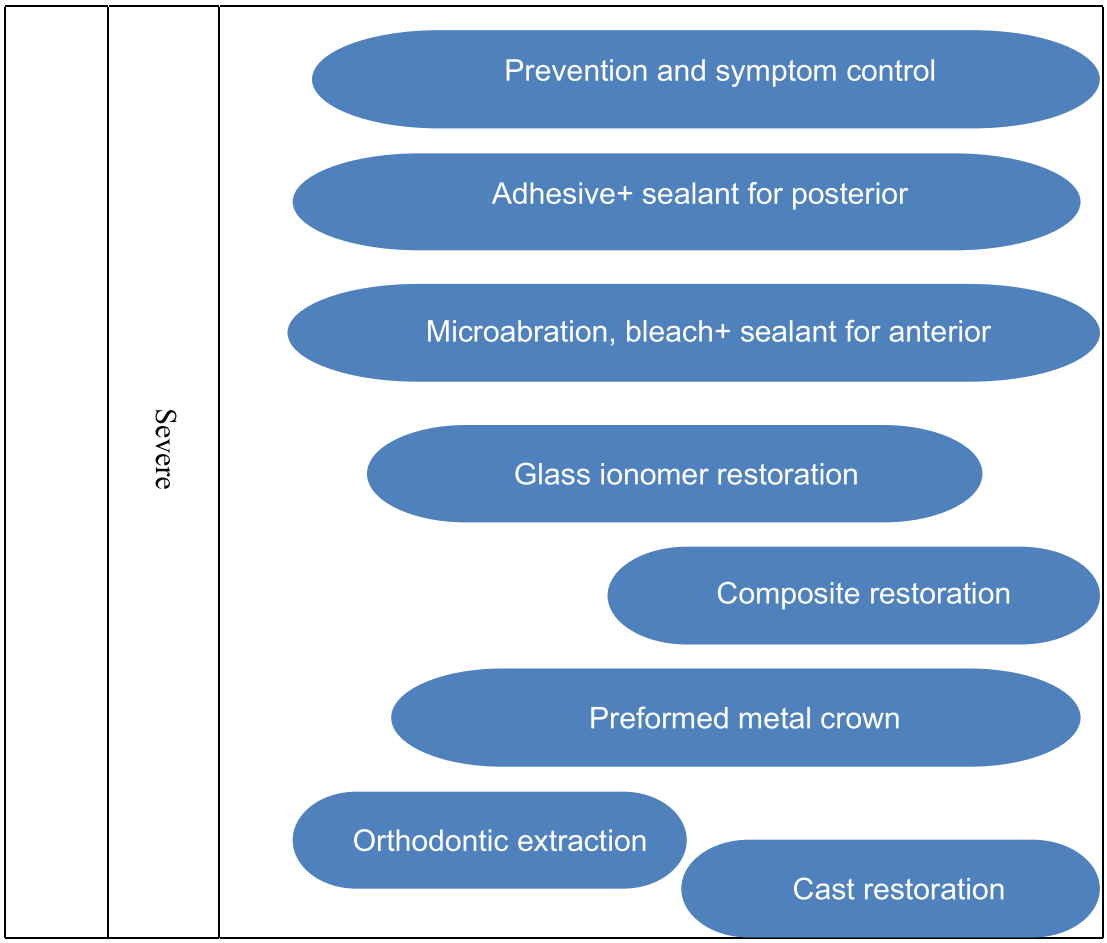


Figure4

A 6-step management approach proposed by William et al (2006)51also have great importance.

Steps	Recommended procedures
Risk identification	Assess medical history for putative etiological factors
Early diagnosis	Examine at-risk molars on radiographs if available
	Monitor these teeth during eruption
Remineralization and desensitization	Apply localized topical fluoride
Prevention of dental caries and post-eruption breakdown (PEB)	Institute thorough oral hygiene home care program
	Reduce cariogenicity and erosivity of diet
	Place pit and fissure sealants
Restorations or extractions	Place intracoronal (resin composite) bonded with a self-etching primer adhesive or extracoronal restorations (stainless steel crowns)
	Consider orthodontic outcomes post-extraction
Maintenance	Monitor margins of restorations for PEB
	Consider full coronal coverage restorations in the long term

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