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Molar-incisor hypomineralisation: narrative review on aetiology, epidemiology, diagnostics, and treatment decision

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Molar-incisor hypomineralisation (MIH)
Hypersensitivity
Post-eruptive enamel breakdown
Differential diagnosis

SUMMARY

Molar-incisor hypomineralisation (MIH) is clinically defined as demarcated structural enamel defects affecting at least one first permanent molar with or without the involvement of incisors. It is foremost a qualitative developmental defect of systemic origin. The prevalence for MIH is estimated at 12.9% with significant differences between countries. Its aetiology and pathogenesis are still not completely understood. Several environmental and medical causes have been suggested to alter enamel maturation. The hypomineralised enamel may collapse shortly after eruption and as a consequence caries lesions seem more likely to develop. Besides cavitation, hypersensitivity and/or pain are the hallmarks of clinical symptoms. Both are associated with increased dental anxiety and fear of children suffering from MIH. Consequently, patients' care and management are challenging and necessitate a large range of non-, micro- and invasive strategies. MIH might be mixed up with three other types of developmental defects in the enamel: fluorosis, enamel hypoplasia, and amelogenesis imperfecta. Careful diagnostic differentiation should be made before starting any dental treatment. A recent published classification system links the severity of the lesion to a treatment need index. This index is based on four values regarding two key symptoms: hypersensitivity and post-eruptive enamel breakdown (PEB). Without PEB, sealing is strongly recommended in order to prevent caries. For hypersensitive teeth as well as those with PEB, use of glass ionomer cement as an intermediate cover is recommended, but mainly composite resins are materials of choice. For improvement of aesthetically compromised MIH incisors, the resin infiltration technique has been proposed.

Introduction

Developmental defects in the enamel (DDE) derive from disturbances in hard tissue matrices formation and/or mineralisation during odontogenesis. These defects can be localised or appear more widespread, affecting single/multiple teeth or groups of teeth (Commission on Oral Health, Research and Epidemiology 1992). Examples of DDE are fluorosis (FS), enamel hypoplasia (EH) and amelogenesis imperfecta (AI). Another entity of DDE is the so-called molar-incisor hypomineralisation (MIH).

Since the early 1970s, dentists have reported a developmental defect primarily located in first molars and incisors in the permanent dentition. Early denominations referred to this clinical condition in a descriptive manner for "which is not":

1. nonendemic stained enamel (Jackson 1961)
2. idiopathic hypomineralisation of the enamel of the first molars (Koch et al. 1987)
3. hypomineralisation of the permanent first molars not caused by fluoride (Leppäniemi et al. 2001)
4. cheese molars (Kreulen et al. 1995)

This reflected a poor understanding of its aetiology that continues to this date. Weerheijm et al. baptised this pathology as MIH in 2001 and it was adopted as the official denomination at the sixth annual conference of the European Academy of Paediatric Dentistry (EAPD) in 2003 (Weerheijm et al. 2001). MIH is defined as developmental (systemic) qualitative enamel defects that are present on one or more first permanent molars (FPMs), each possibly with different degrees of severity. The (central) permanent incisors might be affected additionally. If incisors are affected, at least one first molar must also show enamel hypomineralisation to confirm the diagnosis MIH.

The aim of this review is to summarise the current knowledge on the aetiology, prevalence and diagnostics of MIH as well as to provide guidance on treatment decision and to discuss evidence of non- and microinvasive interventions. A second review by our group will focus on the choice of invasive treatment options.

Prevalence and incidence

Nowadays, MIH is recognised as a global dental problem. In spite of the EAPD criteria cited above, cross-comparisons of the results from various epidemiological studies have been difficult due to the use of various indices and criteria, examination variability, methods of recording and varying age groups (Jälevik 2010). Prevalence is defined by how often a condition is present in a population and incidence by how many new cases occur each year. The reported prevalence of MIH in children and adolescents differs significantly between studies varying between 2 and 40% (Jälevik 2010). A recent systematic review and metaregression analysis (Schwendicke et al. 2018) estimated a global mean (95% CI) prevalence for MIH of 12.9% (11.7–14.3%) with significant differences between countries. The highest numbers of prevalent cases were found in high-income and heavily populated countries. Another metaanalysis (Zhao et al. 2018) estimated the prevalence of MIH around 14% globally with no statistical difference between sexes, but <10-year-olds had a higher prevalence than older individuals (15% vs 12%). It was also noted that MIH was particularly high in some regions such as South America (18%) and Spain (21%). For Switzerland no representative data have been reported so far.

A study on the distribution and severity of MIH affected molars in four areas in Germany revealed that the majority of children with MIH showed more than one affected molar (only one affected molar in 39.2%, two affected molars in 33.5%, three affected molars in 12.0%, and four affected molars in 15.3%) (Petrou 2012). About half showed hypomineralisation in FPMs without additionally affected incisors. 12% of the children with MIH showed hypomineralised defects in at least one of the second primary molars. Furthermore, FPMs were usually more often and more severely affected than permanent incisors. Upper permanent incisors were usually more often affected than lower permanent incisors and, if lower incisors were affected, in most cases hypomineralisation in upper permanent incisors as well as in FPMs were documented (Petrou 2012).

This was corroborated by other studies showing that the FPMs and incisors are often affected, although there can be considerable differences within a dentition. MIH has also been observed in other teeth of the permanent dentition (second molar, second premolar, canine) as well as in second primary molars and also primary canines (Elfrink et al. 2012; Fuchs et al. 2009). MIH is supposed to affect 878 million individuals with a global number of incident cases estimated at 16.0 million people in 2016. It is, therefore, imperative to develop appropriate dental healthcare strategies to treat MIH and to identify its aetiology in order to prevent.

Aetiology and pathogenesis

MIH has been reported to have a negative impact on children's quality of life and sociopsychological status (Dantas-Neta et al. 2016). Affected teeth are in higher need of dental treatment, especially those with post-eruptive enamel breakdown (PEB). As a consequence, caries lesions develop more easily, leading finally to pulpal inflammation along with hypersensitivity or pain. Thus, it is of great importance to identify the aetiology of MIH and to understand its pathogenesis. Despite an augmented interest in MIH and much published research on the subject, its aetiology remains, to this day, not completely understood (Crombie et al. 2009; Alaluusua 2010; Silva et al. 2016).

Environmental causes

MIH is considered to establish due to an impaired calcium and phosphate incorporation during enamel matrix formation and enamel maturation. Ameloblasts are sensitive to insults, both indirect and direct. Even the smallest changes in the environment of the ameloblasts may irreversibly disrupt the formation of the enamel matrix and the maturation of the enamel. Another key factor in understanding the aetiology of MIH is the chronology of tooth mineralisation. The mineralisation of enamel in a first permanent molar starts just before birth and is completed in the first year of life (Reid & Dean 2006). Therefore, it seems logical that any possible cause must have occurred during the period from just before birth until the end of the first year of life (Fig. 1) (Schroeder 2000; Dulla et al. 2018). Research on the aetiology of MIH has compiled a long list of candidates but failed to prove causality due to low numbers of prospective birth-cohort studies. Nonetheless, ongoing studies have elucidated some risk factors (e.g., "LISA" and "GINIplus"; Flexeder et al. 2020). "LISA" (influence of life-style factors on the development of the immune system and allergies in East and West Germany) and "GINIplus" (German infant study on the influence of nutrition intervention PLUS environmental and genetic influences on allergy development) are two populated-based German birth cohorts. They aim to describe the natural course

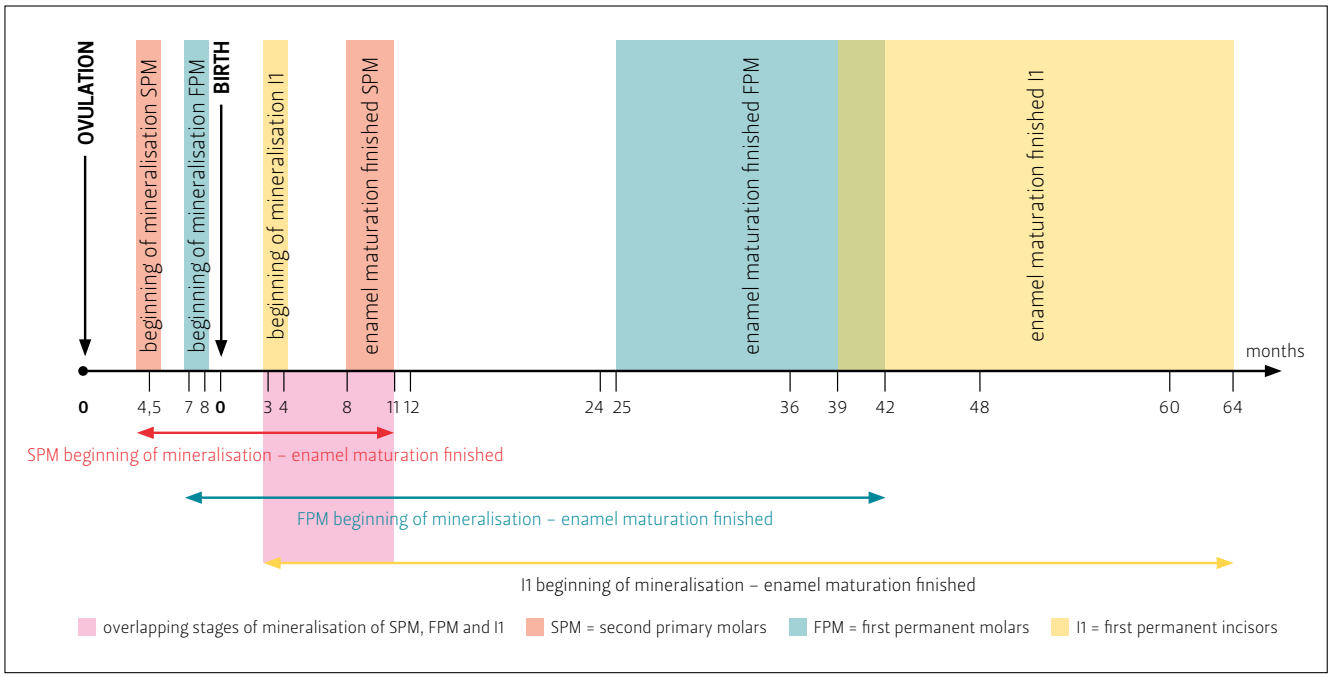


Fig. 1 Sequence of enamel mineralisation from onset to final maturation for first permanent molars (FPM), first permanent incisors (I1) and second primary molars (SPM) (modified from Dulla et al. 2018)

of chronic diseases and intermediate phenotypes in childhood and its determinants and to identify potential genetic effect modifications.

Potential environmental causes can be grouped into either being of prenatal, perinatal or postnatal origin (Tab. I). As indicated in Table I, there was little evidence of an association between the most frequently investigated prenatal factors (maternal smoking, illness, medication) and MIH as well as perinatal factors (prematurity, low birth weight, caesarean delivery, birth complications) and MIH. A consistent finding is that early childhood illness, in particular pyrexia, appears to be associated with MIH.

Genetic influence

The amelogenesis phase has been shown to be modulated by genes (Fincham et al. 1999), and the size, shape, structure, and composition of the enamel seem to be influenced by genetic variations. The potential role of genetics or epigenetics in association with MIH has been discussed (Vieira & Manton 2019; Hočevar et al. 2020). Several genes related to MIH, such as enam, ameloblastin, amelogenin, and bone morphogenetic protein 2, have been investigated (Bussaneli et al. 2019; Jeremias et al. 2013). In addition to these single gene effects, gene-to-gene interactions may also play a role in MIH (Pang et al. 2020). However, data based on twin studies (Teixeira et al. 2018) only showed tendencies of an influence of genetics on MIH prevalence, while higher family income and gestational bleeding were strongly positively associated with MIH. Genetic variability may influence the aetiology, but seems not to act as the primary cause of MIH.

Some intriguing observations

MIH can affect one sibling and not the other(s), and teeth forming at the same period can be affected to varying degrees or not at all. Histological studies reported that MIH lesions extend through the full thickness of enamel, affecting mainly the coronal and not the cervical enamel, and often the buccal surface of the tooth (Crombie et al. 2013; Fagrell et al. 2013; Gambetta-Tessini et al. 2017). Vieira & Manton attempted to address these variables in the clinical presentation of MIH (Vieira & Manton 2019). According to them, the reason why only one side of a bilateral structure is affected likely involves differential gene expression between the left and right despite the dentition on one side being mirrored on the other. As for the disturbances of specific areas and the multiple degrees of the severity, they postulated that this resulting anatomical appearance is a combination of random microenvironment influences (i.e., pressure from surrounding liquid within the connective tissues surrounding the enamel organ) and/or genetic variants (i.e., differential levels of expression at the cellular level and the directionality of molecular signalling).

Diagnosis

General diagnostic criteria and a simple classification system were set by the EAPD to facilitate the diagnosis of MIH (Weerheijm et al. 2003). It can be summarised by the following:

- The visual aspect of the lesion is opaque, clearly demarcated from the healthy enamel, varies in colour (white, yellow, brownish) and size. The darker opacity indicates more hypomineralised (softer) enamel.
- The enamel thickness is normal, but its breakdown appears after the eruption of the affected tooth (PEB).
- When restorations are present, these extend in most cases to the buccal and palatal/lingual surfaces with an opacity at the margin of the restoration.
- For incisors, these restorations are not related to a history of trauma.
- A missing FPM in an otherwise sound dentition can be an indication of a history of MIH.
- In some cases, eruption difficulties of FPMs due to enamel roughness have been proposed (Almuallem & Busuttil-Naudi 2018).

Tab. I Suspected MIH-associated factors (grouped into prenatal, perinatal and postnatal origin) and their quality of evidence (modified from Silva et al. 2016)

	Suspected associated factors	Most notable studies	Type of study	Quality of evidence
Prenatal	Maternal smoking	Kühnisch et al. 2014	Prospective cohort	No significant association
		Souza et al. 2012	Retrospective cohort	
		Arrow 2009	Retrospective cohort	
		Pitiphat et al. 2014	Retrospective cohort	
	Maternal illness	Ghanim et al. 2013	Retrospective cohort	Unadjusted to no significant association
		Sönmez et al. 2013	Retrospective cohort	
		Pitiphat et al. 2014	Retrospective cohort	
		Allazzam et al. 2014	Retrospective cohort	
	Maternal medicine use	Ghanim et al. 2013	Retrospective cohort	No proof of association
		Souza et al. 2012	Retrospective cohort	
		Arrow 2009	Retrospective cohort	
		Allazzam et al. 2014	Retrospective cohort	
Perinatal	Prematurity	Sönmez et al. 2013	Retrospective cohort	Contradictory results (3 studies found positive association, 1 study found reduced risk!)
		Brogårdh-Roth et al. 2011	Retrospective cohort	
		Allazzam et al. 2014	Retrospective cohort	
		Kuscu et al. 2009	Retrospective cohort	
		Pitiphat et al. 2014	Retrospective cohort	
	Low birth weight	Ghanim et al. 2013	Retrospective cohort	No proof of association
		Pitiphat et al. 2014	Retrospective cohort	
		Allazzam et al. 2014	Retrospective cohort	
		Sönmez et al. 2013	Retrospective cohort	
	Birth complications	Allazzam et al. 2014	Retrospective cohort	No proof of association
		Durmus et al. 2013	Case-control	
		Sönmez et al. 2013	Retrospective cohort	
		Pitiphat et al. 2014	Retrospective cohort	
	Caesarean delivery	Jälevik et al. 2001	Retrospective cohort	Unsufficient evidence (only 1 study found positive association)
		Ghanim et al. 2013	Retrospective cohort	
		Pitiphat et al. 2014	Retrospective cohort	
		Souza et al. 2012	Retrospective cohort	
Postnatal	Pneumonia	Sönmez et al. 2013	Retrospective cohort	Unsufficient evidence (only 2 studies found positive association)
		Brogårdh-Roth et al. 2011	Retrospective cohort	
		Allazzam et al. 2014	Retrospective cohort	
		Beentjes et al. 2002	Case-control	
		Pitiphat et al. 2014	Retrospective cohort	
	Asthma	Kühnisch et al. 2014	Prospective cohort	Unsufficient evidence (only 3 studies found positive association)
		Pitiphat et al. 2014	Retrospective cohort	
		Allazzam et al. 2014	Retrospective cohort	
		Sönmez et al. 2013	Retrospective cohort	
	Fever	Allazzam et al. 2014	Retrospective cohort	High probablity (many studies found a positive association with higher odds) Attention: fever is associated with many illnesses (i.e., pneumonia, otitis media)
		Ghanim et al. 2013	Retrospective cohort	
		Sönmez et al. 2013	Retrospective cohort	
		Pitiphat et al. 2014	Retrospective cohort	
		Beentjes et al. 2002	Case-control	
	General childhood illness	Allazzam et al. 2014	Retrospective cohort	Higher probability in case of associated fever
		Ghanim et al. 2013	Retrospective cohort	
		Pitiphat et al. 2014	Retrospective cohort	
		Souza et al. 2012	Retrospective cohort	
	Antibiotic use (early childhood medication)	Allazzam et al. 2014	Retrospective cohort	Unsufficient evidence (few studies found positive association and many did not)
		Pitiphat et al. 2014	Retrospective cohort	
		Whatling & Fearne 2008	Case-control	
		Laisi et al. 2009	Retrospective cohort	
		Arrow 2009	Retrospective cohort	
		Ghanim et al. 2013	Retrospective cohort	
	Antiasthma medication (early childhood medication)	Wogelius et al. 2010	Retrospective cohort	
		Arrow 2009	Retrospective cohort	
	Breastfeeding – dioxine	Laisi et al. 2008	Prospective cohort	No proof of association (except Fagrell et al. 2011)
		Fagrell et al. 2011	Case-control	
		Alaluusua et al. 1996a	Prospective cohort	
		Alaluusua et al. 1996b	Prospective cohort	

MIH – TNI	PEB	
	without	with
Hypersensitivity: without	1	2
Hypersensitivity: with	3	4

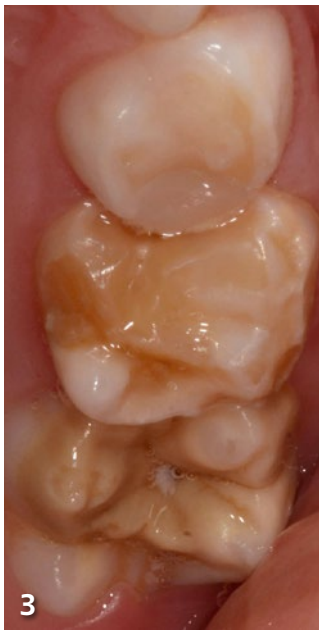
Fig. 2 Index values 1–4 based on the two clinically most important symptoms: hypersensitivity and post-eruptive enamel breakdown (PEB) (modified from Bekes et al. 2016, Steffen et al. 2017)

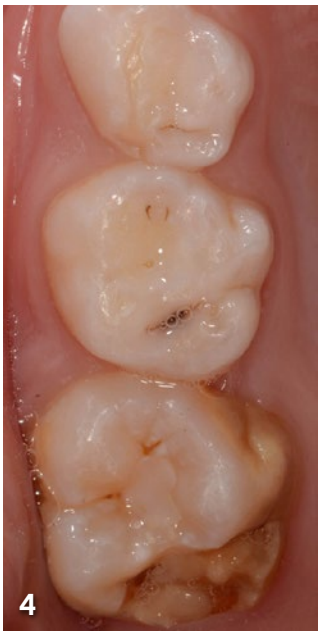
Several descriptive classifications have been proposed; e.g., Leppäniemi et al. (2001) rated the severity of MIH within three categories:

1. mild: opacities without PEB
2. moderate: opacities with PEB limited to enamel
3. severe: PEB with dentin involvement, atypical restorations, and tooth extraction due to MIH

Another simple classification system, also based on the severity of MIH, proposed distinction between mild and severe cases (Lygidakis et al. 2010):

1. In mild cases, the demarcated opacities do not exhibit a PEB, but they can present occasional sensitivity to external stimulus with less aesthetic concerns.
2. In severe cases, the demarcated enamel is associated with PEB, hypersensitivity (HS), and high aesthetic demands.





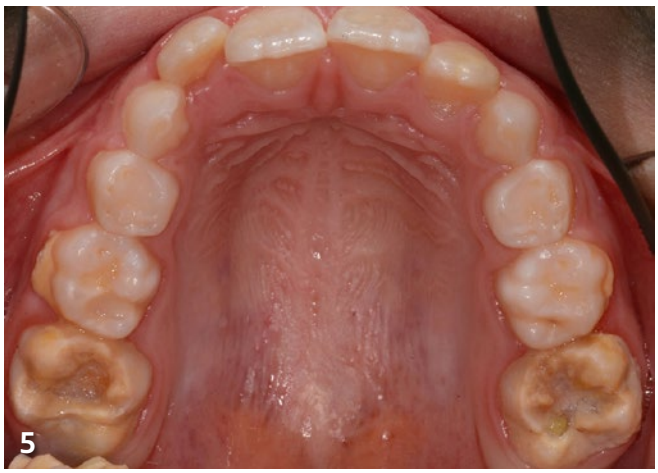


Fig. 3 First permanent upper molar with well-demarcated brown-yellowish opacities and a very small defect of the mesio-palatal cusp. The second primary molar shows opacities and an irregular surface with substance loss.

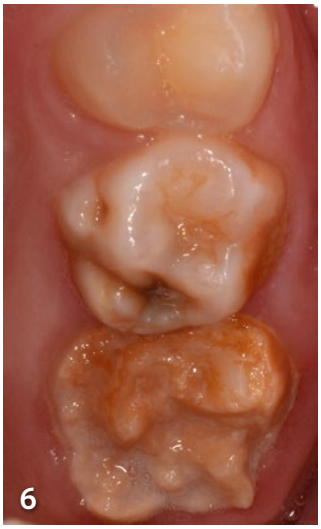
Fig. 4 First permanent upper molar with opacities principally located palatally-distally and with a defect of approximately 1/3 of the surface. The occlusal surface has already been sealed with a temporary filling material.

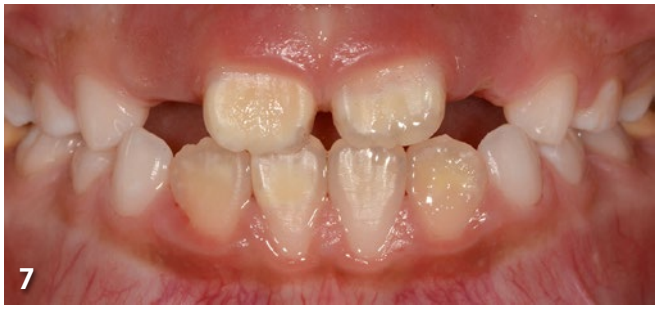
Fig. 5 First permanent upper molars showing brown-yellowish opacities and defects with decay located in the occlusal fissure

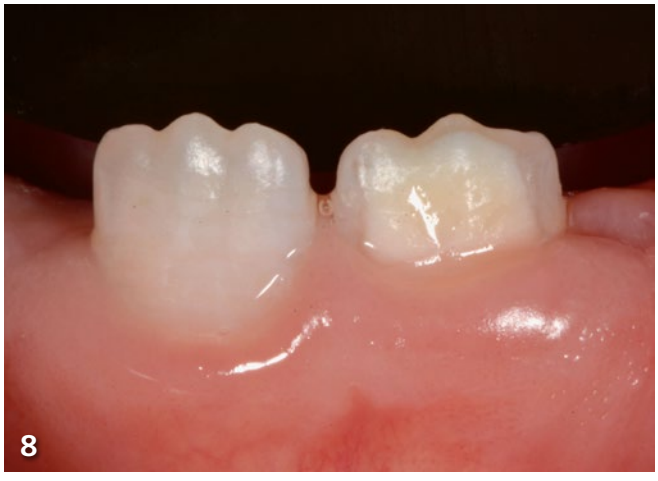
Fig. 6 First permanent upper molar with complete destruction of the surface

Fig. 7 Permanent upper and lower incisors with well-demarcated white-yellowish opacities; first permanent lower molars with yellowish opacities

Fig. 8 Eruption of a permanent lower incisor with a well-demarcated white-yellowish opacity on the labial surface







Tab. II Developmental defects and their typical characteristics

	Molar–incisor hypomineralisation	Amelogenesis imperfecta	Fluorosis	Enamel hypoplasia
Generalised (all primary and permanent teeth affected)		x		
Symmetric		x	x	
Reduced enamel thickness		x		x
Mainly qualitative defects	x	x	x	
Mainly limited to one or more FPM	x			
Often, but not always hypersensitive	x	x		x
History of trauma or periapical inflammation of primary tooth				x

Steffen et al. in an attempt to standardise MIH diagnostic criteria and treatment needs conceived a classification system that links the severity of the lesion to a treatment need index (TNI) (Steffen et al. 2017). This index is based on four values (Fig. 2) regarding two key symptoms that are considered the most important ones with respect to MIH: HS and PEB. The highest value is recorded for each sextant by the use of good light and drying with an air syringe.

Teeth affected by MIH show various characteristics (Figs. 3–8). HS is a common symptom in MIH teeth that might impair oral hygiene, limit dietary habits, cause chronic pain and trigger dental anxiety. Its intensity depends on the severity of the lesion. A recent study (Raposo et al. 2019) concluded that mild cases of MIH are associated with HS of a low intensity, while severe cases showed more frequently higher degrees of HS. Severely hypomineralised teeth are at higher risk of developping caries, which could increase HS considerably. The cause of this HS appears to be chronic pulp inflammation due to repeated triggers, whether thermal, mechanical, or bacterial (Fagrell et al. 2008). PEB occurs due to the severe porosity of the hypomineralised opaque areas that fracture when subjected to masticatory forces, resulting in unprotected dentin being more prone to external triggers (Garg et al. 2012). Interestingly, whitish discoloured MIH teeth seem to be at a ca. 33% lower risk for PEB than yellowish/brownish ones (Cabral et al. 2016).

Clinical examination

With the complete eruption of all first molars and incisors at ca. 8 years of age hypomineralised enamel of relevant teeth can be preferably detected and MIH be diagnosed (Lygidakis et al. 2010). Nonetheless, MIH might be diagnosed during the eruption of the FPM. An early diagnosis may limit the degree and size of PEB and the high risk of subsequent HS and dental caries (Garg et al. 2012).

FPMs are first screened for MIH and then for caries. Teeth should be cleaned with a toothbrush and fluoride toothpaste and examined in wet condition using a mirror and probe. The tooth surface can also be gently cleaned with a cotton roll, but it should preferably not be dried. Intraoral photography is a great tool to better examine and assess the damaged tooth on a computer monitor (Chen et al. 2013). Subsequently, the tooth is dried using air jet or, if not possible, with cotton rolls, to examine for caries and to evaluate possible HS.

Differential diagnosis

MIH might be mixed up with three different other types of DDE: FS, EH, and AI. It can be helpful that MIH is the most prevalent type. It is a well-demarcated qualitative "chalky" defect (contrary to EH), nonsymmetrical (contrary to FS, AI), limited to one or more FPM (contrary to AI, FS), with or without central permanent incisors implication (contrary to FS, AI), caries prone (contrary to FS), generally, but not always hypersensitive (contrary to FS), and with no history of trauma on the affected tooth (Weerheijm 2004; Commission on Oral Health, Research and Epidemiology 1992; Elcock et al. 2006; Crawford et al. 2007) (Tab. II). Furthermore, traumatic injury as well as prolonged periapical inflammation process of a primary tooth may affect the development or maturation of the permanent successor and lead to a so-called Turner's tooth/Turner's hypoplastic tooth, which is one example for EH.

Pain control and therapy

For the affected and suffering quite young child, visiting a dentist is often accompanied with a great portion of anxiety and reluctance. In order to rebuild the bridge of trust between the child and the dentist, a painless first examination is absolutely essential. Thermal stimuli, such as air syringe, cold water, or cold instruments, should be avoided. Any subsequent dental treatment should be performed under a very effective pain control protocol (Jälevik & Klingberg 2002).

Pain is a subjective experience, especially when it overlaps with anxiety and fear. The fear of having pain can be overwhelming for a child, especially in case of previous traumatic dental experiences. A prerequisite for any pain control protocol to be effective is to ensure a proper emotional management of the suffering child; otherwise, the protocol is doomed to failure. This behavioural and emotional management is the standard in pediatric dentistry (McNeil et al. 2006). A combination of three techniques (i.e., analgesic, anesthetic, and sedation) is often necessary in pain management of MIH teeth.

Systemic analgesic premedication

The use of an analgesic premedication can be helpful in the treatment of hypersensitive MIH molars. The choice of the drug can be paracetamol or nonsteroidal anti-inflammatory drugs NSAID (ibuprofen or metamizole). Steffen & van Waes described a treatment protocol based on patients suffering from chronic back pain. The protocol recommends the ingestion of a very high but short-term dosage (24–48 h). The anti-inflammatory effect of NSAIDs is desirable, especially in chronically inflamed MIH teeth. In order to influence chronic pain, medication should be taken >24 hours before the dental treatment. The four doses are distributed as follows: >24 h, 12 h, 6–8 h before dental treatment and the last dose shortly before the procedure (Steffen & van Waes 2011).

Anaesthesia

Treatment of MIH-affected teeth in young patients is challenging due to chronic pulpal inflammation caused by porous enamel and exposed dentin. Failure in achieving profound and sufficient anesthesia may lead to behaviour problems and affect the quality of restorations. The clinician might reach the maximum anaesthetic dose (limited by weight and age), and the tooth remains hypersensitive. Type and dose of the anaesthetic are not as important as the accessory techniques employed to achieve anaesthesia (Discepolo & Baker 2011). For lower FPM, inferior alveolar nerve block adjunct to buccal infiltration is commonly used. Periodontal ligament injections can help in the establishment of anaesthesia, although its safety is constantly debated. Intraosseous injection technique used in endodontics is also proven to be effective and provides profound anaesthesia of long duration (60 minutes or longer) (Nusstein et al. 2005). Crestal intraosseous local anaesthesia by the use of a computer-assisted injection system is an effective and safe technique to achieve profound anaesthesia in MIH-affected hypersensitive teeth. This technique could be beneficial when treating an MIH tooth with a pulpitis (Dixit & Joshi 2018; Discepolo & Baker 2011; Fouad & Levin 2006).

Sedation

While anaesthesia helps to reduce pain, sedation helps tremendously in removing the emotional context of fear and anxiety accompanying the dental treatment. Drug sedation can be used, especially in very young children. However, whenever the child maintains nasal breathing, inhalation sedation (nitrous oxide/oxygen mixture) is the medication of choice, especially in treating MIH cases (Esch 2009; Steffen & Langerweger 2018; Steffen 2018). If the child shows no cooperation at all, general anaesthesia remains the only option.

Treatment decision

Fortunately, not all MIH-affected teeth need immediate treatment. In any case, an intensive prophylactic protocol should be administered as early as MIH is diagnosed. The purpose is to delay enamel breakdown and prevent the development of dental caries. Based on the MIH TNI, Bekes et al. have developed two therapy schemes for patients with either low or high caries risk. Prophylaxis is based on a self-applied "home" and a professional "in office" part. The former comprises mainly the home use of fluoride toothpaste, but also other preventive measures (e.g., CPP-ACP mousse) have been proposed, but no conclusive evidence of increased beneficial effects exists. It should be combined with regular professional application of fluoride varnishes (Bekes et al. 2016; Fütterer et al. 2020). Nonetheless, all of these recommendations are mainly based on clinical studies regarding caries prevention, since no distinct noninvasive intervention has yet been proven to be more efficacious than another with respect to PEB prevention and/or HS reduction in teeth affected by MIH.

Indication and choice of treatment are driven by local factors (HS with or without PEB) and general factors (mainly patient age). For a FPM affected by MIH without PEB with or without HS, sealing therapy with a resin-based fissure sealant is the method of choice in addition to intensive home-based preventive measures. In a high-caries-risk patient with partially erupted FPM, the application of a flowable glass ionomer cement (GIC) is recommended as an intermediate protection. In order to stabilise the porous structure of hypomineralised molars, resin infiltration seems to prevent from enamel breakdown to a greater extent compared with fluoride varnish (Nogueira et al. 2020). If HS persists after the application of a sealant, a direct or indirect restoration should be chosen.

The presence of PEB (with or without HS) will result in an invasive, mostly restorative treatment. Its extent determines whether the tooth is restorable or not. The treatment of a FPM with PEB but without HS is determined by the localisation and the size of the defect. If the loss of substance is not in the fissure and includes less than 1/3 of the tooth surface, the application of a sealant is recommended. However, if there is substance loss located in the fissure or the defect size is more than 1/3 of the tooth or the defect is close to the pulp, short-term temporary restoration using GIC with or without an orthodontic band should be the therapy of choice. After the tooth has matured, the temporary filling can be replaced by a definitive restoration. Alternatively, a temporary long-term restoration in form of a steel crown can also be an option. If a FPM shows substance loss and HS, the treatment follows the same stages as in MIH TNI 2 (Bekes et al. 2016). It should be noted that restorative recommendations are only based on a low scientific evidence level (see the second paper regarding MIH of our group; Weber et al. 2021). If future extraction is indicated, orthodontic treatment should follow. The aim is to allow the second permanent molar to erupt naturally as a substitute of the lost FPM. Thus, the timing of the extraction (ca. 8–11 years of age depending on the status of tooth development) is of great importance.

Therapy of MIH-affected incisors

For improvement of aesthetically compromised MIH incisors, the resin infiltration technique, originally developed to arrest and mask caries lesions, has been proposed. However, in contrast to caries lesions and fluorotic teeth, MIH is not always completely masked by resin infiltration only. A higher degree of surface removal (etching) prior to the application of the infiltrating resin has been proposed for better aesthetic results. Still, in non-satisfying cases as well as when hypoplastic areas are apparent from the beginning, composite on top of deeply infiltrated areas are necessary to achieve optimal results (Meyer-Lückel et al. 2017, 2020).

Conclusion

- The reported prevalence of MIH in children and adolescents differs significantly between studies varying between 2.4 to 40.2% (Jälevik 2010). Globally, a mean (95% CI) prevalence for MIH of 12.9% (11.7–14.3%), with significant differences between countries, has been estimated (Schwendicke et al. 2018).
- FPMs are usually more often and more severely affected than permanent incisors. Upper permanent incisors are more often affected than lower permanent incisors (Petrou 2012).
- Despite an augmented interest in MIH and much published research on the subject, its aetiology is still not completely understood (Crombie et al. 2009; Alaluusua 2010; Silva et al. 2016). The current evidence suggests that multiple, most presumably environmental and medical risk factors trigger MIH during enamel formation and maturation.
- MIH might be mixed up with three different other types of DDE: FS, EH, and AI. Careful diagnostic differentiation should be made before starting any dental treatment.
- In an attempt to standardise MIH diagnostics and treatment, a new classification system that links the severity of the

lesion to a TNI has been proposed (Steffen et al. 2017). The index is based on the two most important key symptoms with respect to MIH: HS and PEB.
- Without PEB, sealing is strongly recommended in order to prevent caries. For hypersensitive teeth as well as those with PEB use of GIC as an intermediate cover is recommended, but mainly composite resins are materials of choice. Resin infiltration might be a suitable additional option, in particular for aesthetic improvement in visible teeth. It should be noted that restorative recommendations are only based on a low scientific evidence level (Weber et al. 2021).

Conflicts of interest

JAD declares no conflicts of interest, real or perceived, financial or nonfinancial. HML is appointed as inventor for patents of an infiltration technique for dental caries lesions, held by Charité-Universitätsmedizin Berlin, and receives royalties from DMG, the manufacturer of Icon.

Zusammenfassung

Ziel dieser Übersichtsarbeit war es, die Literatur nach dem aktuellsten Wissensstand über die Ätiologie, Prävalenz und Diagnose von MIH sowie über den Therapieentscheid hinsichtlich der Optionen non-, mikro- und invasiver Behandlungen zu durchsuchen.

Der Begriff Molaren-Inzisiven-Hypomineralisation (MIH) wurde im Jahr 2001 von Weerheijm et al. eingeführt und beschreibt einen zumeist qualitativen Schmelzdefekt an einem oder mehreren ersten Molaren, häufig mit Beteiligung der permanenten Inzisiven und hier vor allem im Oberkiefer. MIH ist eine entwicklungsbedingte Schmelzbildungsstörung aufgrund einer fehlerhaften Kalzium- und Phosphateinlagerung während der Schmelzmatrixbildung und Schmelzreifung. Die weltweite Prävalenz von MIH bei Kindern und Jugendlichen wird auf 12,9% geschätzt; ein ähnliches Vorkommen wird auch für die Schweiz vermutet. Die Ätiologie ist bis heute nicht vollständig geklärt. In der Literatur werden perinatale Ursachen (z.B. Hypoxie, Hypokalzämie, Geburtskomplikationen, Frühgeburt, Dioxine, polychlorierte Biphenyle), neonatale Ursachen (z.B. Krankheiten im Kleinkindesalter, Medikation, Fieber) sowie auch die Beeinflussung durch genetische Polymorphismen diskutiert.

Klinisch weisen hypomineralisierte Zähne weissliche, gelbliche bis bräunliche, gut abgrenzbare Opazitäten auf. Je dunkler die Opazität, desto poröser der Schmelz. Erste permanente Molaren sind in der Regel häufiger und stärker betroffen als permanente Inzisiven. Obere permanente Inzisiven sind in der Regel häufiger betroffen als untere permanente Inzisiven. Wenn untere permanente Inzisiven betroffen sind, ist in den meisten Fällen eine Hypomineralisation der oberen permanenten Inzisiven sowie der ersten permanenten Molaren feststellbar.

Die Hypersensibilität ist ein häufiges Symptom bei MIH-Zähnen, wodurch die Mundhygiene und die Ernährungsgewohnheiten beeinträchtigt werden. Der chronische Schmerz kann in der Folge eine gesteigerte Angst vor dem Zahnarzt verursachen. Eine frühzeitige Diagnose ist entscheidend, um einen posteruptiven Schmelzverlust zu verzögern oder am besten zu verhindern.

Kürzlich wurde ein neues Klassifizierungssystem publiziert (MIH treatment need index; MIH TNI), das im Besonderen das Ausmass an Destruktion der Zahnhartsubstanz in Kombination mit den bei MIH auftretenden Hypersensibilitäten berücksichtigt (Steffen et al. 2017). Anästhesieversager bei hypersensiblen MIH-Molaren sind häufig. Die krestale intraossäre Lokalanästhesie unter Verwendung eines computergestützten Injektionssystems kann hilfreich sein, um eine ausreichende Anästhesie bei hypersensiblen MIH-Molaren zu erreichen. Gelingt keine suffiziente Schmerzausschaltung, so können die Einnahme einer analgetischen Prämedikation sowie auch die Lachgassedation hilfreich sein. Während die Anästhesie hilft, Schmerzen zu lindern oder zu beseitigen, hilft die Sedierung, den emotionalen Kontext von Angst und Furcht, der mit der Zahnbehandlung verbunden ist, zu beseitigen. Oftmals ist eine Kombination von analgetischer Prämedikation, Anästhesie und Sedierung in der Behandlung hypersensibler MIH-Molaren notwendig. Zeigt das Kind keine Kooperation, bleibt die Vollnarkose die einzige Option.

Die Indikation für eine non-, mikro- oder invasive Therapie von MIH-Molaren hängt von lokalen (Hypersensibilität und posteruptivem Schmelzverlust) und allgemeinen Faktoren (Patientenalter und Kariesrisiko) ab. Hierbei steht eine optimale Mundhygiene, unterstützt durch die professionelle Applikation von Fluoridlacken, im Vordergrund. Generell liegt keine gesicherte klinische Evidenz zur Überlegenheit einer speziellen prophylaktischen (non-invasiven) Methode bei MIH vor, sodass man sich am besten an etablierte Methoden zur Verhinderung von Karies hält.

Zur Verbesserung ästhetisch beeinträchtigter MIH-Inzisiven kann die kunststoffbasierte Infiltrationstechnik angewendet werden, die ursprünglich entwickelt wurde, um Kariesläsionen zu stoppen und zu maskieren. Zu den invasiven Therapien wird unsere Arbeitsgruppe in einer zweiten Veröffentlichung Empfehlungen geben (Weber et al. 2021).

Résumé

Le but de cette revue était de rechercher dans la littérature les connaissances les plus récentes sur l'étiologie, la prévalence et le diagnostic de la MIH, ainsi que sur les options de traitement non-, micro- et invasif.

Le terme d'« hypominéralisation molaires incisives » (MIH) a été développé en 2001 par Weerheijm et coll. et décrit un défaut d'émail essentiellement qualitatif sur une ou plusieurs premières molaires, souvent avec atteinte des incisives permanentes et ici surtout de la mâchoire supérieure. MIH est un trouble du développement de la formation de l'émail dû à un stockage défectueux du calcium et du phosphate pendant la formation de la matrice de l'émail et la maturation de l'émail. La prévalence mondiale de MIH chez les enfants et les adolescents est estimée à 12,9 % ; une prévalence similaire est supposée pour la Suisse. L'étiologie n'est pas encore entièrement connue. Dans la littérature, les causes périnatales (p. ex. l'hypoxie, l'hypocalcémie, des complications à la naissance, la naissance prématurée, les dioxines, les polychlorobiphényles), les causes néonatales (p. ex. les maladies infantiles, des médicaments, la fièvre) et l'influence des polymorphismes génétiques sont discutées.

Cliniquement, les dents hypominéralisées présentent des opacités blanchâtres, jaunâtres à brunâtres qui peuvent être clairement définies. Plus l'opacité est foncée, plus l'émail est poreux. Les premières molaires permanentes sont généralement plus fréquemment et plus sévèrement atteintes que les incisives permanentes. Les incisives permanentes supérieures sont généralement plus souvent atteintes que les incisives permanentes inférieures. Si les incisives permanentes inférieures sont at-

teintes, une hypominéralisation des incisives permanentes supérieures et des premières molaires permanentes peut être identifiée dans la plupart des cas.

L'hypersensibilité est un symptôme courant dans les dents MIH, qui affecte l'hygiène bucco-dentaire et les habitudes alimentaires. Par la suite, la douleur chronique peut provoquer une angoisse accrue du dentiste. Un diagnostic précoce est crucial afin de retarder la perte d'émail postéruptive ou, si possible, de la prévenir.

Un nouveau système de classification a été récemment publié (MIH-Treatment Need Index ; MIH-TNI), qui prend notamment en compte le degré de destruction du tissu dentaire en combinaison avec les hypersensibilités qui apparaissent avec MIH (Steffen et coll. 2017). Les échecs d'anesthésie des molaires hypersensibles sont fréquents. L'anesthésie locale intraosseuse utilisant un système d'injection informatisé peut être utile pour obtenir une anesthésie adéquate dans les molaires hypersensibles. Si la douleur ne peut pas être éliminée de manière adéquate, l'utilisation d'une prémédication analgésique et d'une sédation au protoxyde d'azote peut être utile. Pendant que l'anesthésie aide à soulager ou à éliminer la douleur, la sédation aide à éliminer le contexte émotionnel de l'anxiété et de la peur associées au traitement dentaire. Une combinaison de prémédication analgésique, d'anesthésie locale et de sédation est souvent nécessaire dans le traitement des molaires hypersensibles. Si l'enfant ne coopère pas, l'anesthésie générale reste la seule option.

L'indication d'un traitement non, micro ou invasif des molaires MIH dépend de facteurs locaux (hypersensibilité et perte d'émail postéruptive) et généraux (âge du patient et risque de carie). L'accent est mis ici sur une hygiène bucco-dentaire optimale, soutenue par l'application professionnelle de vernis fluorés. En général, il n'y a pas de preuve clinique fiable de la supériorité d'une méthode prophylactique spéciale (non invasive) dans la MIH, il est donc préférable de s'en tenir aux méthodes établies de prévention des caries dentaires.

Pour améliorer l'esthétique des incisives atteintes par MIH, la technique d'infiltration à base de composite, qui a été initialement développée pour arrêter et masquer les lésions carieuses, peut être utilisée. Notre groupe de travail montre des recommandations sur les thérapies invasives dans une deuxième publication (Weber et coll. 2021).

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