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## Understanding MIH: definition, epidemiology, differential diagnosis and new treatment guidelines

### ABSTRACT

**Aim** Molar-Incisor Hypomineralisation (MIH) is a congenital disease which increases in prevalence. It affects permanent first molars and, often to a lesser degree, permanent incisors with variable severity. The aetiology is unknown, but different hypotheses have been advanced. Differential diagnosis is mandatory not to confound MIH with other diseases. Treatment consists in a minimally invasive approach by reinforcing and protecting the existing dental structure. In more severe cases, restorative treatment may be indicated.

**Keywords** Diagnosis; Enamel disorders; Glass ionomer cement; Molar-Incisor Hypomineralisation.

### Introduction

Described for the first time in the 1970s, Molar-Incisor Hypomineralisation, more commonly known as MIH, is not always diagnosed by general practitioners. It requires a multifactorial treatment concept, in order to deal with the different aspects of the disease (hypersensitive teeth in case of temperature changes, fast decay development, unsatisfying local anaesthesia and even recurrent eating disorders), all of this resulting in behavioural problems in young patients.

Of all tooth enamel disorders, MIH appears to be

the most common. From an epidemiological point of view, it progresses constantly. It engenders significant loss of tissue if it is not accurately diagnosed and correctly treated. Molar Incisor Hypomineralisation is a deficit in the mineralisation process of permanent first molars and, less frequently, incisors, resulting from a lack of calcium and phosphate fixing on the matrix formed by the ameloblasts. The resulting enamel is not completely mineralised, causing yellowish, hypersensitive enamel, with little resistance, affecting the dentin's surrounding tissue. The disorder may be limited to a single ridge, though it may extend over the whole smooth surface of the teeth, reaching the occlusal surfaces [Koch et al., 1987].

Several terms are used to define the pathology.

- › Non-endemic stained enamel [Jackson, 1961].
- › Idiopathic hypomineralisation of the enamel of the first molars [Koch et al., 1987].
- › Cheese molars [Van Amerongen and Kreulen]
- › Hypomineralisation of the permanent first molars not caused by fluoride [Leppaniemi et al., 2001].
- › Molar-Incisor Hypomineralisation MIH [Weerheijm et al., 2001].

In 2003, at the 6th annual conference of the European Academy of Paediatric Dentistry (EAPD), it was agreed on that the latest terminology would be used.

It is important to note that the amelogenesis of the permanent incisors lasts from the age of 3 months until the age of 5, and that the one of the first molars begins around the 8th month of pregnancy, ending about the age of 4. So, the mineralisation loss appears during these two development periods, especially during the first 10 months of life and from the age of 2 and 6 months until the age of 5. At that early stage, metabolic disorders will interfere with odontogenesis. The term "idiopathic" hypomineralisation is therefore applied, given the diversity of the possible etiological factors. Hypomineralisation of primary dentition remains an unknown phenomenon, but its epidemiological impact on the population increases constantly. MIH origins are subject to controversy in scientific literature and its exact pathogenesis is still unknown. The most common hypothesis is that it might result from a local malfunction during enamel formation, aligned with general medicine pathologies, taking place at the embryonic stage and during young childhood [Weerheijm, 2003]. Diseases are frequently feverish infections, pathology of the airways, malfunctions in gas exchange, and even low weight at birth. The influence of environmental pollution or excess of dioxins in the mother's milk might also interfere [Kellerhoff, 2004]. At present, the most likely theory is that there is a synergy between systemic and external factors. The aetiology of MIH is therefore multifactorial, but for the moment, this is based on assumptions (Tab. 1).

THE MOST LIKELY CAUSES OF MIH
High dioxin or polychlorinated biphenyl (PCB) content in the mother's milk and over 9 months breastfeeding [Alaluusua et al., 1993, Jan & Vrbic 2000, Weerheijm et al., 2001].
Child premature birth and deprived of oxygen at birth or later [Jackson, 1961, Leppäniemi et al., 2001].
Respiratory disorders in young childhood (relapsing bronchitis, chronic obstructive pulmonary disorder and bronchial asthma) [Van Amerongen and Kreulen, 1995, Jalevik et al., 2001].
Infectious diseases such as diphtheria, scarlet fever, mumps or measles in the first three years [Jackson, 1961].
Mineralisation deficit: hypoparathyroidism, malnutrition, malabsorption, coeliac disease, hypovitaminosis D [Sarnat and Schour, 1942, Follis et al., 1952].

TABLE 1

### Epidemiology

Prevalence of MIH is now between 2.8% and 25% and appears to vary among country, region, and age bracket considered [Gotler and Ratson, 2010]. However, its incidence is rising continuously, worldwide.

### Diagnosis

To compare prevalence studies, criteria for accurate diagnosis of MIH have been proposed [Weerheijm et al. in 2003]. They are based on clinical observation of dry, clean tissues, revealing the presence of clearly marked enamel opacities. The appearance of the tooth is opaque and white-chalky/creamy or yellow-brown, of normal thickness, with smooth surface. While the surface enamel appears hard, the sub-surface is soft and porous. At first, the enamel will quickly crumble in some places, and then everywhere, under the strain of chewing forces after the moment the teeth have erupted completely. In severe cases of MIH, the loss might cause breakup of the enamel or dentine part, or even cause absence of hard tissue, to a greater or lesser extent [Koch et al., 1987]. From a histological point of view, hypomineralised teeth have a reduced mineralisation degree from the amelocemental junction to occlusal surface, and increases again around the canines.

MIH diagnosis might be difficult to pinpoint when the permanent first molars are already very damaged and decayed and have been extensively restored, have unusual occlusal topography or have already been extracted. Chewing and decay easily damage these fragile tissues. Where this happens, observing undamaged enamel on the other permanent first molars might be used to orientate the diagnosis towards MIH. It is essential to distinguish between MIH and other abnormalities in the dental structures. A patient's history is mandatory for seeking acquired, environmental or genetic aetiologies. Accurate differential diagnosis from these pathologies [Ostertag,

2009] should be noticed.

- a) *Amelogenesis imperfecta*: hereditary abnormality, or genetic dysplasia, which affects the permanent dentition. Accordingly, the enamel will have abnormal makeup in terms of chemistry, quantity and/or structure, but the structure of the dentine will be normal [Schroeder, 1991]. Only severe forms of MIH give molar abnormalities, which can be confused with those caused by *amelogenesis imperfecta*. In most cases, MIH produces asymmetrical disorders in the permanent first molars and incisors, unlike *amelogenesis imperfecta* in which all teeth are affected.
- b) *Enamel hypoplasia* is a disorder concerning the quantity of enamel due to damage during the secretory phase of amelogenesis. The reduction in the enamel thickness is localised. Following the fast damage to the surface of the enamel of molars affected by MIH during the post-eruptive phase, the lesions might resemble enamel hypoplasia. However, hypoplastic teeth do have regular borders around healthy enamel, whereas these borders are irregular around molars affected by MIH, whose enamel breaks up after eruption. Possible example: Turner's hypoplastic tooth.
- c) *Dental fluorosis* results from excessive fluoride absorption during tooth mineralisation. Stigmata are diffuse and the disorder is symmetrical. In fluorosis, the tissues are decay-resistant, not like in hypomineralised teeth.
- d) *Tooth decay* is generally found as soon as it appears in the most vulnerable areas. Decay may also develop as a result of accumulation of plaque in a hypoplastic area and make diagnosis difficult.
- e) *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. With calcium, tetracycline forms a chelate complex. This complex becomes irreversibly fixed to the enamel and dentine during formation of tissues in the tooth [Schroeder, 1991].

Clinical studies and patient history allow us to clearly distinguish changes in biological structure due to tetracycline or fluoride from Molar-Incisor Hypomineralisation: Tetracycline or a fluoride overdose exert systemic action and the resulting mineralisation deficit is thus symmetrical, reaching all parts of teeth whose dentine and enamel were being formed.

There is a multitude of clinical difficulties specific to MIH. In particular, practitioners will have to consider:

- a) *Tooth hypersensitivity*, often present from the outset. This especially manifests itself through changes in temperature or contact. It must be treated fast as it will determine whether any other problems will arise. In fact, as affected molars are sensitive to brushing, they will be avoided by children, thus

increasing the risk of decay [Rodd et al., 2007].

b) *Rapidly developing decay*: the physiological immaturity of hard tissues, as well as the fragile nature of enamel, associated with a bucco-dental hygiene problem as evoked previously, will cause serious and fast, irreversible damage to the coronal structures. Moreover, these hypoplastic areas form a reservoir for plaque and food residues.

c) *Anaesthetic difficulties*: Hypersensitivity and rapid damage to the tissues will cause chronic pulpal inflammation, preventing effective local anaesthesia. Loco-regional techniques (Spix), osteo-central techniques or even electronically-assisted local anaesthesia, such as Quick Sleeper or Wand, may be useful.

Quick Sleeper enables optimum performance of all anaesthesia, including osteo-central. Equipment specifications are as follows:

- Exclusive needle rotation system. Crossing the cortical bone is quick and easy and makes for easy access to spongy bone.
- Electronically-managed discontinuous rotation. The rate at which the needle enters the bone is controlled, without heating up.
- Needle anti-obstruction system. The electronics manages the injection during the crossing of the cortical bone and prevents needle obstruction.
- Automatic injection speed and pressure for painless diffusion of fluids into the soft tissues.

Another device is the Wand Plus, which has the following specifications.

- Anaesthesia without loss of sensitivity in the tongue, lips and cheek.
- Constant anaesthetic diffusion rate (below the pain threshold).
- No needle deflection because of the rotating injection technique.
- Use of a fine needle and transparent handpiece.

These 2 devices also offer the benefit that they are less frightening, as they are in the form of a pen with a small nib.

d) *Problem of restoration*: adhesion of the restorative material is low on soft, hypomineralised enamel, so the risk of early loss of restoration and development of secondary decay is higher.

Clinical handling, treatment (short-, medium- and long-term) and progress are early MIH diagnosis (cleaning, magnifying glasses, microscope) and prevention of decay (fissure sealing) and loss of substance due to rapid erosion caused by chewing forces.

Treatment at the School of Dental Medicine of the University of Geneva includes the following:

- › At-home application of Tooth Mousse.
- › 1×/day, every evening before sleeping for 6 weeks. After brushing the teeth, rinse well and swallow saliva before applying. Apply Tooth Mousse using

a cotton bud, the finger or an inter-dental brush in the presence of a parent or guardian. Leave for 3-5 minutes. With their tongue, the patients may then spread the product around their mouth for more than 2 minutes (formation of mousse). The patient must then spit out and not eat or drink for 60 minutes.

- › Application of a high-fluoride, highly adherent GIC for surface protection: Fuji Triage (GC). Protect the gum by placing cotton wool in the vestibule on the side of the tooth to be treated. Dry the occlusal surface. If possible, go over all occlusal surfaces with Prophy-Jet without touching the gum (any bleeding would adversely affect product adhesion). Rinse well and dry gently. Apply the GIC rapidly, and check that there are no bubbles and no gaps; let the patient close and reopen the mouth, let the product set. Polish out any excesses using brownies and finish with Occlubrush or Depurdent on a brush. Follow up the patient regularly and apply fluoride varnish during each session to recharge the GIC with fluoride.
- › At-home application of Elmex gel 2×/week (once only to affected teeth, the second time to all teeth).

## Materials

1. GC Fuji Triage (GC, in capsules) is a protection material. This is a GIC (Glass Ionomer Cement) with high fluoride content. The release of fluoride is 7 to 10 times higher than that of a reinforced conventional GIC. It works by releasing a high amount of fluorides in the first few days after application and the release falls rapidly in the following weeks. This fluoride release is highly influenced by the pH in the area around the restoration: the more acid the pH is, the faster the release of fluoride ions is and so the less time it takes. The permeability of GICs means that they permanently recharge themselves with fluoride ions when they are in an oral environment in which fluoride input is regular (fluoride toothpaste, fluoride varnish, fluoride gel) [Lasfargues et al., 2004] GC Fuji Triage may be applied when salivary control is not possible (tolerates humidity) and when treating erupting molars. There are several benefits: chemical adhesion (neither phosphoric acid etching, nor using an adhesive) to the dental structure, pink colour facilitates the control of the protective layer in recalls, low viscosity, ease of application and very high fluoride liberation. It is indicated for protection from decay, reducing tooth hypersensitivity and stabilising enamel through fluoride release [Reich, 2005].
2. Tooth Mousse or MI Paste (GC) is a water-based, sugar-free cream for topical use, enriched in CPP ACP complexes (Recaldent) [Reeves and Latour,

1958]. CPP is a casein phosphopeptide and ACP is an Amorphous Calcium Phosphate. CPP has the capacity to transport phosphate and calcium ions, bonded to its complex in the form of Amorphous Calcium Phosphate, ACP. Calcium phosphate is normally insoluble, forming a crystalline structure with a neutral pH. When Recaldent is applied to the oral cavity, the "bonding" CPP part of the CPP-ACP complex easily attaches to the enamel, the pellicle, the dental plaque and the soft tissues, delivering calcium and phosphate to exactly the right places. The calcium and phosphate ions available then leave the CPP, penetrate the enamel prisms and turn into apatite crystals [Cross et al., 2005].

Applying Tooth Mousse is indicated:

- *in very small children* at high risk of decay, enabling decay to be stabilised
- treatment of white spots, especially in the young, as well as for reinforcing against tooth erosion
- reducing tooth sensitivity and reinforcing tissues affected by MIH by restoring the balance between remineralisation and demineralisation.

However, it is contraindicated to apply the product to patients who are allergic to milk proteins. The strengths of Tooth Mousse are diversity of flavours, a broad spectrum of effects, almost unrestricted use in young children until they reach maturity, ease-of-use, and a favorable cost-benefit ratio. MI Paste Plus or ToothMousse Plus has the same indications as ToothMousse and is recommended from the age of 6 as its formula also contains Sodium fluoride 0.2 % W/W (900 ppm); not to be used in very small children as, if swallowed, it may cause digestive disorders.

3. Elmex gel is a drug registered with the OICM for intensive anti-decay prophylaxis, and is especially indicated in the event of high levels of decay activity, sensitive tooth necks, and in the event of enamel demineralisation.

The effects of this prophylaxis are stimulation of remineralisation (amine fluorides form a stable fluoride deposit on the surface of the teeth), reduction in solubility in an acidic medium (amine fluorides considerably increase the enamel's acid resistance), homogeneous action across all dental surfaces (because of their softening properties, amine fluorides spread fast and uniformly over all dental surfaces) and inhibition of plaque formation obtained through anti-microbial action.

Elmex gel hardens exposed dentine and increases its resistance to mechanical abrasion. The product is made up of amine fluoride (0.25%, 2,500 ppm), sodium fluoride (1%, 1.000 ppm), excipients, water and a humectant.

c) Restorative treatments: sealants, composite, onlay, inlay. The spectrum of restorative tools extends from fissure sealant for a completely healthy crown,

to almost complete rebuilding of clinical crowns with overlays, always by using adhesive techniques.

d) Special points about the treatment of MIH:

- be aware of sensitivity (fluoride application, MI Paste at the office and at home);
- difficulty not to confound MIH with active decay.
- rapid advance of decay.

e) Lack of child co-operation because the teeth are hypersensitive and require "management" with parents. Application of MI Paste or fluoride (Elmex gel) at home and arrange several treatment and follow-up sessions (prophylactic cleaning, Duraphat) at the office.

f) Tooth extractions if the damage to the tooth structures is irreparable, and co-operation with orthodontists to manage craniofacial development.

## Case report

### *Insurance coverage*

In Switzerland, MIH is not considered a congenital handicap and is thus not covered by Swiss Medical Disability Insurance (AI). Depending on the insurer and under private insurance, conservative and prophylactic treatment may sometimes be covered, also for the treatment of MIH.

### *Description of the issue and legal facts*

DL, born on 29th March 2002, is a child born prematurely, at 33 weeks.

In accordance with the communication from the Disability Insurance Office in the canton of Geneva (OAI), as soon as she was born, she benefited from medical insurance covering treatment of congenital diseases. Cover for treatment of congenital disease, including medical checks and physical therapy from April to May 2003, was confirmed through a ruling passed on April 8, 2004.

The policy holder, her parents, made a claim for medical coverage in November 2009, seeking coverage for prevention and then final treatment of the permanent malformation to the tooth enamel. Since August 2009, she has been treated for dental dysplasia. As certified on November 11, 2009, enamel hypoplasia (MIH) was diagnosed, treated since August 2009, pointing out that this was not brought about by a congenital disease as defined in the Order on Congenital Diseases. It was stated that the final anterior dentition is affected and that there is anterior occlusal open bite in teeth 12 – 22 and 32 – 42. It should also be noted that teeth 11, 32, 16, 26, 36 and especially 46 are affected by dental dysplasia.

In a draft ruling dated November 25, 2009, the OAI rejected the claim, stating that treatment for dental hypoplasia is not covered by the Medical Disability Insurance.

## Conclusion

There are many possible aetiological factors for Molar-Incisor Hypomineralization (MIH). A few of the factors responsible for amelogenesis disorders have been presented in this work: deprivation of oxygen at birth or during the post-natal period, Chronic Obstructive Pulmonary Disorder; presence of dioxins or polychlorinated biphenyl (PCB) in the mother's milk; infectious diseases of young childhood and lack of mineral fixing.

A single set of teeth often presents a significant disorder of clinically variable appearance. In the same patient, one may find a molar whose edge only has diffuse opacity, whilst other molars show extensive hypoplasia on several surfaces. The surface's porous structure does encourage accumulation of dental plaque, encouraging deep decay and extensive conservative treatment, despite the young age of the patients.

It is also very important to begin intensive individual prophylaxis. Because of the hypersensitivity of the teeth, adequate oral hygiene is often lacking in the patients concerned. Many therapy options, ranging from occlusal sealant to tooth extraction, not to mention restoration, exist [Fitzpatrick and O'Connell, 2007]. It is therefore necessary to make an objective choice when it comes to the most suitable treatment. In view of the specific requirements of patients with MIH, we should not hesitate to call upon other specialists, such as orthodontists, for multidisciplinary treatment.

## Disclosure

None of the authors is a consultant or receives or has

received financial support from any of the companies cited.

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