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Molar-incisor hypomineralisation (MIH) – aetiology, clinical picture, treatment

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KEYWORDS

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SUMMARY

Introduction. Molar-incisor hypomineralisation is a disorder of dental enamel of a systemic origin. It may affect one or all four first permanent molars, and often involves permanent incisors.

Aim. The aim of this study was to present views on aetiology, clinical picture and treatment of molar-incisor hypomineralisation.

Material and methods. PubMed database was reviewed for years 2003-2017, the search criteria were: molar incisor, hypomineralisation, permanent teeth.

Results. The aetiology of MIH is multifactorial and not fully explained. Authors agree that the general health during the first 3-4 years of life has a major impact on this disorder. The clinical picture includes demarcated white, yellow or brown tissue spots, and increased porosity of the enamel. Enamel damage and defects can also occur. Therapeutic management depends on the severity of the disease and includes intensive prevention, hard tissue reconstruction, and extractions.

Conclusions. Due to the increasing prevalence of this disorder, special attention should be paid to children whose health status is or was bad in the first 3-4 years of life. They are at a higher risk for this condition.

INTRODUCTION

Molar-incisor hypomineralisation was described as a clinical entity by Weerheijm in 2001 (1). It is defined as a disturbance of the enamel of a systemic origin. One or more first permanent molars are usually effected; however, incisors are also often involved (1-4). It may vary in severity, and first incisors are usually less involved than molars (2). The global incidence varies, ranging between 2.4 and 40.2% (3, 5-7). The increasing prevalence of this disorder is confirmed by the fact that first permanent molar hypomineralisation is a more common problem than caries of chewing surfaces in Denmark (4). No significant sex-related differences in the prevalence of this pathology have been observed (5, 8). Clinical manifestations of MIH include demarcated, white, yellow or brown lesions. The loss of porous enamel, which may break off upon normal occlusal forces, is also common. Children often avoid brushing hypomineralised teeth due to increased sensitivity, and the increased accumulation

of biofilm leads to rapid caries development (7, 9). MIH-related defects may affect the child's quality of life, and the treatment poses a major challenge for patients, parents and doctors (6).

AIM

The aim of this study was to present views on aetiology, diagnosis and preventive/therapeutic management of molar-incisor hypomineralisation based on the available literature.

MATERIAL AND METHODS

PubMed database was reviewed for years 2003-2017. The keywords included molar incisor, hypomineralisation, and permanent teeth. Non-English articles, animal studies and orthodontic papers were excluded from the study. Literature available in text databases of the library of the Medical University of Białystok was used. A total of 29 positions were selected.

AETIOLOGY

The aetiology of MIH is not fully explained. Studies suggest its complexity. The impact of idiopathic systemic and genetic factors that interfere with amelogenesis is emphasised (2, 10, 11).

Enamel development can be broken down into three stages: secretory, transition and maturation (12). As pointed out by Kühnisch et al., genetically determined susceptibility may play a role in the development of MIH. Based on 10-year clinical research, the authors identified a gene locus, which may be associated with this disorder. The gene is SCUBE1 located on chromosome 22 (13).

Among other causative factors, the role of environmental and systemic disorders is emphasised. It is suggested that in the case of molar-incisor hypomineralisation, ameloblast dysfunction occurs at the early stage of enamel maturation or late stage of enamel secretion (12, 13). The process of mineralisation of first permanent molars begins during foetal life, and the first 3-4 years of life are a critical period (1, 14).

The effects of damaging factors may occur during pre-, peri- and postnatal life (2, 10, 15-17). In the prenatal period, attention is paid to maternal viral infections, multiple episodes of high fever, hypertension, diabetes, malnutrition and pharmacotherapy (10, 16, 18).

Particular role in the development of MIH in the perinatal period is attributed to factors that induce oxygen deficit during amelogenesis, as well as hypokalaemia, twin pregnancy, prematurity, low birth weight, complications during delivery and caesarean section (10, 16, 18-20).

In the postnatal period, environmental factors, such as exposure to pollution (dioxins), which may penetrate into the child's body with breast milk, are important (21). Diseases experienced by the child during the first years of life, which often involve high fever and require the use of antibiotics, are also considered. Varicella, measles, respiratory diseases (asthma, bronchitis, pneumonia), otitis media, tonsils, and gastrointestinal diseases are also mentioned (9, 18-23).

Andrade et al. showed in their study in children and adolescents aged between 7 and 15 years that MIH was significantly more common in HIV-positive patients infected by vertical transmission compared to healthy individuals. According to the authors, this is associated with the use of protease inhibitors by these patients (24).

Kühnisch et al. showed that the child's vitamin D levels may be also involved. Vitamin D plays a key role in the formation of mineralised tissues, including enamel and dentin. Based on a 10-year observation period, the authors demonstrated that lower serum 25(OH)D levels are associated with an increased risk of MIH. Additionally, higher vitamin D levels correlate with a lower number of carious permanent teeth (25). The authors pointed out that children with poor general health during first 3-4 years of life are more likely to develop MIH than healthy children (9, 18, 19, 21-23).

CLINICAL PICTURE

Clinical examination should be performed on clean moist teeth (2, 4, 8, 26). The presence or absence of demarcated enamel opacities, post-eruptive breakdown, atypical fillings, and tooth extractions due to MIH is investigated (4).

The clinical picture of MIH includes delineated white, yellow or brown spots, increased enamel porosity, enamel damage and defects. Some authors point to a link between the stage of tissue hypomineralisation and susceptibility to post-eruptive enamel breakdown (10). Da Silva Costa et al. demonstrated in their study that teeth with yellow and brown spots show significantly increased susceptibility to post-eruptive loss of tissue compared to white teeth (10).

EAPD diagnostic criteria for MIH were published during the congress in Athens in 2003 (tab. 1) (4).

Ogdell et al. distinguished 3 stages of molar-incisor hypomineralisation: mild (< 30% of dental tissue damage), moderate (30-50%), and severe (> 50%) (27). A new 4-step MIH Treatment Need Index (MIH-TNI), which, in addition to the degree of tissue damage, considers hypersensitivity, was proposed during the conference of the German Society of Paediatric Dentistry in 2016 in Würzburg (27).

It should be noted that the diagnosis of these lesions may be difficult due to the risk of post-eruptive enamel breakdown induced by occlusal forces. Furthermore, MIH-related damage may be masked by extensive carious lesions or fillings (2). The differential diagnosis should include enamel hypoplasia, fluorosis, amelogenesis imperfecta, white carious lesions, discolouration after tetracycline therapy, and erosions (4) (fig. 1-3).

TREATMENT

There are many difficulties in the treatment of teeth affected by MIH. This is associated with increased dental sensitivity, rapid caries progression, and repeated filling damage (6, 14). The authors point to an increased level of anxiety associated with dental treatment among children with MIH compared to healthy peers (6, 12). This may be due to the difficulty achieving full anaesthesia, which is most likely related to the subclinical inflammatory process in pulpal cells due to enamel porosity and easy bacterial penetration (1, 2, 12, 14).

The management depends on the severity of molar-incisor hypomineralisation (3, 10). The affected children should report for regular check-up visits every three months. Cooperation between the dentist and parents is very important. Jälevik et al. showed in their study that children with MIH required treatment 10 times more often compared to healthy peers (12).

In the case of teeth without post-eruptive enamel breakdown, intensive preventive measures are recommended. These include topical application of fluoride gels and varnishes, as well as fissure sealants. Fluoride toothpastes and soft toothbrushes are recommended for home care.

Tab. 1. EAPD diagnostic criteria for MIH developed in 2003 during the Congress in Athens (4)

Demarcated enamel opacity	The defect involves changes in the translucency of enamel of varying degree. The enamel has normal thickness with smooth surface; it may be white, yellow or brown. The lesions are demarcated from healthy tissues
Post-eruptive enamel breakdown (PEB)	Enamel defect on the surface of erupted teeth may be due to mechanical trauma during chewing and attrition
Atypical restoration	Atypical size and shape of filling. In most cases, the filling also involves buccal and palatal/glossal smooth dental surfaces. The enamel on the border of fillings may show altered translucency
Extraction due to MIH	The absence of the first permanent molar in full dentition may raise a suspicion of MIH, especially if changes typical of this disorder are observed in the remaining teeth

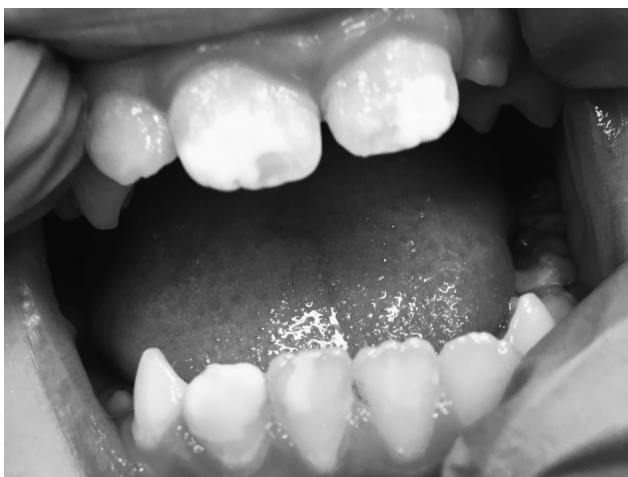


Fig. 1. Hypomineralisation of incisors in a 9-year-old boy



Fig. 2. Molar-incisor hypomineralisation in an 8-year-old boy

Furthermore, the use of phosphopeptide-amorphous calcium phosphate (CPP-ACP) preparations, which enhance remineralisation and relieve hypersensitivity, is also recommended (10, 16, 21, 28).

There is no unambiguous opinion on the adhesion of composite fissure sealants. Fragelli et al. demonstrated that there is no significant difference in maintaining fissure sealants between hypomineralised vs. healthy

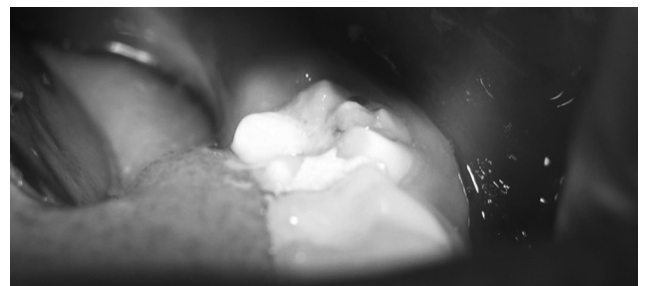


Fig. 3. Molar-incisor hypomineralisation in an 8-year-old boy

teeth (29). Other authors have a different view on this subject. They believe that poor adhesion of sealants to the affected tissues may be the problem. Some authors recommend application of 5% sodium hypochlorite for 60 seconds to remove proteins from the enamel surface before fissure sealing. They believe that this enhances the digestive effects of 35% phosphoric acid (16, 28). Lygadiakis et al. suggest the use of fifth generation bonding systems to increase sealant adhesion, and fissure sealing with glass-ionomer cements for partially erupted teeth (28).

Hard dental tissue restoration is necessary in more severe cases of MIH with post-eruptive enamel breakdown and caries. The use of amalgam is contraindicated due to the poor maintenance of these fillings, which is caused by an atypical shape of fillings in teeth affected by molar-incisor hypomineralisation (16, 28). Adhesive materials, such as conventional glass-ionomer cements, resin-modified glass-ionomer cements, polyacid-modified composites, and conventional composite materials are materials of choice. Although glass-ionomer cements bond to enamel and dentin and release fluoride, they should be used as temporary fillings, base materials for composite fillings and, as already mentioned, fissure sealants for partially erupted teeth, due to their poor mechanical strength (10, 16, 28) (fig. 4). The best long-term outcomes are achieved by reconstructing hard dental tissue with composites. According to Kotsanos et al., maintaining these fillings in a 4-year period oscillates



Fig. 4. A temporarily restored tooth 36 using glass-ionomer cement in an 8-year-old boy with molar-incisor hypomineralisation

at the level of 74% (16). Some authors also recommend the use of standard steel crowns (16, 28).

In cases of unfavourable long-term prognosis, extraction of first permanent molars should be considered following orthodontic consultation. Optimal age for these procedures

is between 8.5 and 9.5 years, when mineralisation of root bifurcation in second molars begins (16).

A 6-step therapeutic management protocol proposed by Werheijm et al. (2007), which involves risk assessment, early diagnosis, remineralisation and elimination of hypersensitivity, prevention of caries and post-eruptive enamel breakdown, restoration or extraction, as well as maintaining therapeutic effects and regular follow-up visits, is very useful (2, 21).

CONCLUSIONS

In the light of the increasing prevalence of molar-incisor hypomineralisation, particular attention should be paid to children whose general health condition is poor or was poor during the first 3-4 years of life. They are at an increased risk of MIH and should be carefully monitored during permanent molar eruption. Also, MIH-related problems, such as hypersensitivity, increased risk of caries, difficulty achieving effective anaesthesia and repeated filling damage, should be taken into account. It is very important to establish the diagnosis as early as possible, take preventive measures and ensure regular monitoring of children affected by this abnormality.

CONFLICT OF INTEREST

None

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