EDITORIAL

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Molar-Incisor Hypomineralisation – Etiology, Prevalence, Clinical Picture and Treatment – Review

Hipomineralizacja trzonowcowo-siekaczowa – etiologia, częstość występowania, obraz kliniczny i leczenie – przegląd piśmiennictwa

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Abstract

Molar-incisor-hypomineralization (MIH) is a clinical entity manifested by a developmental disturbance of the enamel of a systemic origin. The abnomality can affect one or more first permanent molars and, usually less frequently, one or more incisors. The aim of the paper was to describe the prevalence, etiological factors, clinical manifestation and the treatment of MIH in children based on the papers published in the last 10 years. Moreover, the diagnostic criteria and the severity of MIH accepted by EAPD were described. It was found that MIH prevalence greatly varies in the population of different countries ranging from 2.8 to 40.2%. MIH being a quatitative defect of enamel manifests as localized demarcated opacities whitish-yellow or yellowish-brown colour. Post-eruptive enamel breakdown can occur soon after the tooth eruption, revealing irregular decay which is prone to caries development. The condition is caused by various prenatal, perinatal and postnatal environmental factors disturbing the mineralization process of the enamel. The hypomineralized enamel in MIH teeth reveals a high degree of porosity extending from enamel-dentine junction to the normal cervical enamel and lower hardness and some differences in chemical composition. The treatment options of the abnomality are related to the severity of the enamel defect and range from prevention and reconstruction to extraction **(Dent. Med. Probl. 2014, 51, 2, 165–171)**.

Key words: molar-incisor, hypomineralization, prevalence, diagnosis, etiology.

Streszczenie

Hipomineralizacja trzonowcowo-siekaczowa (MIH) jest oddzielną jednostką chorobową objawiającą się rozwojowym zaburzeniem szkliwa pochodzenia systemowego. Nieprawidłowością może być objęty jeden lub więcej stałych zębów pierwszych trzonowych i zwykle rzadziej jeden lub więcej zębów siecznych. Celem pracy jest przedstawienie częstości występowania, czynników etiologicznych, obrazu klinicznego i leczenia MIH u dzieci na podstawie przeglądu prac opublikowanych w ostatnich 10 latach. Podano ponadto kryteria diagnostyki i ciężkości MIH zaakceptowane przez EAPD. Wykazano, że częstość występowania MIH wykazuje znaczne zróżnicowanie w populacjach różnych krajów i wynosi 2,8–40,2%. MIH, będąc jakościowym defektem szkliwa, manifestuje się jako odgraniczona nieprzezierność koloru białawożółtego lub żółtawobrązowego. Poerupcyjne odłamanie szkliwa może wystąpić wkrótce po wyrznięciu zęba, ujawniając nieregularny ubytek, który jest podatny na rozwój próchnicy. Zaburzenie jest spowodowane różnymi prenatalnymi, perinatalnymi i postnatalnymi czynnikami środowiskowymi, zaburzającymi proces mineralizacji szkliwa. Hipozmineralizowane szkliwo w zębach dotkniętych MIH wykazuje dużą porowatość szerzącą się od połączenia szkliwno-zębinowego do prawidłowego szkliwa w rejonie przyszyjkowym, mniejszą twardość i różnice w składzie chemicznym. Opcje leczenia tej nieprawidłowości zależą od ciężkości zmiany i wahają się od zapobiegania i odbudowy do ekstrakcji **(Dent. Med. Probl. 2014, 51, 2, 165–171)**.

Słowa kluczowe: hipomineralizacja trzonowcowo-siekaczowa, frekwencja, diagnoza, etiologia.

The occurrence of permanent first molars affected by white, yellow or brown opacities and enamel breakdown had been described since the late 1970s. The abnormality was named in a different way, such as cheese molars, idiopathic enamel opacities, idiopathic enamel hypomineralization, opaque spots, non-fluoride enamel opacities, internal enamel hypoplasia, non-endemic mottling of enamel, dysmineralized or hypomineralized first permanent molars [1, 2]. The term "molar-incisor-hypomineralization" (MIH) was proposed by Weerhejim et al. [2] in order to describe specific developmental defects of enamel involving permanent first molars and permanent incisor.

Definition and Diagnosis of Molar-Incisor Hypomineralization

The terminology "molar-incisor-hypomineralization" (MIH) is used to describe hypomineralization of a systemic origin of 1 to 4 permanent first molars, often associated with affected incisors and accepted by EAPD [2]. Therefore, MIH is defined as a separate clinical entity of developmental disturbances of hard dental tissue. During the seminar of the European Academy of Paediatric Dentistry (EAPD) on molar-incisor hypomineralization that was held in Athens 2003, diagnostic criteria were established [3]. For MIH diagnosis at least one permanent first molar must be affected with or without the involvement of the incisors. The defects can also occur in primary second molars as well as involve tips of canines. The more first molars and incisors are affected, the more severe the defect is. The following conditions should be fulfilled for MIH study: the examination of 12 index teeth in 8-year-olds, i.e. permanent first molars and incisors, wet teeth after cleaning (to distinguish from incipient carious lesions), each index tooth recorded for absence or the presence of demarcated opacity, post-eruptive enamel breakdown, atypical restoration, extraction or failure of eruption first permanent molar or incisor due to MIH. When hypomineralization involves only 1 or 4 first molars the term molar hypomineralization (MH) can be used to distinguish children without affected incisors [4]. However, when opacities involved the incisors only, the condition is not MIH [3, 5].

Diagnostic and severity criteria of MIH recommended by EAPD are presented in Tables 1 and 2.

Prevalence

Molar-incisor hypomineralization oocurs in many populations throughout the world. Before the formulation of EAPD diagnostic criteria for MIH, the prevalence of those enamel defects was estimated by the modified developmental defects of enamel index (m-DDE index) or other modifications. After the establishment of MIH diagnostic criteria, many studies on prevalence were carried out in various countries and large variations in the prevalence rates were found, ranging from 2.8–40.2% [6–28]. This broad range could be linked with different age of the subjects or caries level in the studied population masking the real prevalence of MIH. The prevalence of MIH diagnosed with the use of EAPD criteria is presented in Table 3.

Table 1. Diagnostic criteria of molar-incisor hypomineralization according to EAPD [3, 5]

Tabela 1. Kryteria diagnostyczne hipomineralizacji wg EAPD [3, 5]

Criteria	Clinical appearance
Demarcated opacities	clearly visible in various colors (white, creamy, yellow or brownish) and size demarcated opacities located on occlusal and buccal tooth surface, small or large involving the major part of the crown; the affected enamel has normal thickness and a smooth surface; defects less than 1 mm are not recommended to register
Enamel disintegration	the varied degree of porosity of the hypomineralized areas, severe hypomineralized enamel subjected to masticatory forces breaks down soon after the tooth eruption exposing dentin (post-eruptive enamel breakdown – PEB) and leading to rapid caries development, a demarcated opacity may have preexisted
Atypical restoration	molars – size and shape of the restoration are not confirmed typical carious decay, the restoration involves buccal or palatal/lingual surface and at the borders of a demarcated opacity may be seen
Tooth sensitivity	the affected teeth may be sensitive to external stimuli ranging from a mild to spontaneous response; the teeth are usually difficult to anaesthetize
Extracted teeth	extracted tooth due to MIH can be defined only if the other index teeth reveal demarcated opacities, otherwise it not possible to diagnose MIH
Unerupted teeth	index teeth have not yet erupted

Table 2. Severity of MIH according to EAPD [5]

Tabela 2. Zaawansowanie MIH wg EAPD [5]

Severity grade of MIH	Clinical appearance
Mild	demarcated enamel opacities without enamel breakdown occasional sensitivity to external stimuli, e.g., air/water but not brushing mild aesthetic concerns on discoloration of the incisors
Severe	demarcated enamel opacities with breakdown caries persistent/spontaneous hypersensitivity affecting function e.g., during brushing strong aesthetic concerns that may have socio-psychological impact

Table 3. Prevalence of MIH

Tabela 3. Częstość występowania MIH

Frequency	Age	Number of subjects	Country	Reference
10.2	5.5–12	3518	Greece	Lygidakis et al. 2008 [6]
13.73	6–8	3591	Kenya	Kemoli et al. 2008 [7]
37.5	6–8	647	Denmark	Wogelius et al. 2008 [8]
19.8	6–12	918	Brasil	da Costa-Silva et al. 2010 [9]
5.9	6–12	1022	Germany	Preusser et al. 2007 [10]
12.3	6–12	1157	Brasil	Jeremias et al. 2012 [11]
14.6	7	307	Great Britain	Zagdwon et al. 2002 [12]
18.6	7–9	823	Iraqi	Ghanim et al. 2012 [13]
17.6	7–9	570	Jordania	Zawaideh et al. 2011 [14]
14.9	7–9	147	Turkey	Kuscu et al. 2008 [15]
14.9	7–9	1277	Lithuania	Jasulaityte et al. 2007 [16]
10.1	7–10	2395	Germany	Petrou et al. 2013 [17]
40.2	7–13	249	Brasil	Soviero et al. 2009 [18]
19.3	7–13	488	Finland	Leppaniemi et al. 2001 [19]
3.58	7–14	2960	Bulgaria	Kukleva et al. 2008 [20]
18.4	7.6–8.8	516	Sweden	Jalevik et al. 2001 [21]
21.8	8	840	Spain	Garcia-Margit et al. 2013 [22]
9.2	8–12	1366	India	Parikh et al. 2012 [23]
18.8	8.2	235	USA	Mahoney et al. 2011 [24]
14.3	9	422	The Netherlands	Jasulaityte et al. 2008 [25]
36.5	10	693	Germany	Heitmüller et al. 2013 [26]
12.3	12	560	Bosnia and Herzegovina	Muratbegovic et al. 2007 [27]
2.8	12	2635	China	Cho et al. 2008 [28]

Clinical Manifestation

MIH is the qualitative defect of enamel exhibiting localized demarcated opacities whitish-yellow or yellowish-brown color. Occlusal loading of the affected permanent first molars may lead to a post-eruptive enamel breakdown, soon after eruption, presenting decay with irregular and sharp boundaries. Instead, the affected incisors seldom reveal enamel fracture [3, 4]. The degree of staining of the opacity can be linked to the degree of demineralization and prone to post-eruptive enamel breakdown [29, 30]. Farah et al. [29] studied the MIH extracted teeth regarding the color of opacity, mineral density and laser fluorescence. They found a strong correlation between mineral density, laser fluorescence reading and color of opacity concluding that clinically used laser fluorescence reading can reflect the severity of the defect. Da Costa-Silva et al. [30] analyzed in a prospective clinical study white, yellow and brown enamel opacities and found that the brown and yellow opacities revealed a higher risk of post-eruptive enamel breakdown than those of white ones. The severity of MIH can vary not only between subjects but also within the mouth of a single individual in relation to the advancement of each defect

(color, size, post-eruptive enamel breakdown and sequel) as well as the number of affected teeth. Therefore, not all permanent first molars are affected to the same degree; indeed some molars may be untouched [1, 3, 4]. The mean number of affected teeth differs in the studies ranged from 2.4 to 5.7 [6, 8, 13, 20, 27, 26]. The mean number of involved permanent first molars varied from 1.5 to 3.16 and incisors was 2.2 [4, 6, 8, 26]. However, in general, the condition involves more first molars than incisors [6, 10]. The studies also showed more frequently affected maxillary teeth than mandibular ones [6, 18, 30]. Chawla et al. [4], analyzing the distribution of molar-incisor hypomineralization, suggested that MIH is a more severe form of hypomineralization condition than molars hypomineralization (MH). A similar opinion was expressed by Zawaideh et al. [14], who also found no difference between the right and left side of the mouth. Hypomineralization can also involve primary second molars due to sharing the same risk factors for hypomineralization as permanent first molars [31, 32]. Elfrik et al. [31] based on the obtained results (Odds Ration for MIH based on the presence of primary molars hypomineralization was 4.4 95%, CI 3.1–6.4) suggested that the presence of hypomineralization of primary second molars could be a predictor for MIH.

MIH defects with post-eruptive enamel breakdown should not be mistaken for enamel hypoplasia, being a quantitative developmental defect of enamel that manifests a reduction of enamel thickness. The difference is in the margin of the defect; in the case of MIH, the border of the deficient enamel is irregular and sharp contrary to hypoplasia, where the border of normal enamel is smooth [1–3]. Teeth with post-eruptive breakdown can be hypersensitive and susceptible to rapid caries development and progression. MIH can also be mistaken with dental fluorosis. However, in contrast to demarcated margins of MIH, fluorotic enamel opacities are diffused and caries resistant. In comparison to amelogenesis imperfect, in the case of MIH not all teeth are affected and defects are asymmetrically distributed [3].

Some studies showed equal MIH distribution between gender [4, 22, 23, 31], but others more in females [7, 14, 27]. The prevalence of MIH seems to be related to higher caries experience in the permanent dentition than the general population of similar age [9, 17, 22, 24]; however, that coincidence is not confirmed by all the studies [26].

Microstructure Changes

The hypomineralized enamel in MIH teeth had a high degree of porosity extending from enamel-dentine junction to the normal cervical enamel as well as lower hardness and some differences in the chemical composition [33, 34]. It contained higher protein, 3- to 15-fold than normal enamel, but a near normal level of residual amelogenins which distinguished from other defects with high residual amelogenins (amelogenesis imperfecta and fluorosis). Moreover, hypomineralized enamel in MIH revealed the accumulation of various proteins from oral fluid and blood depending on integrity of the enamel surface [35]. Brown enamel opacity showed 15–21-fold, yellow and white 8-fold higher protein content in comparison to normal enamel [36]. Oral bacteria may penetrate through hypomineralized enamel into the dentinal tubules and cause inflammatory reactions in the pulp [33].

Pathogenesis

Enamel is the most mineralized tissue of the body produced by ameloblasts of ectodermal origin. After completing its development, the enamel is not remodeled during the life span and the formed disturbances can be recorded when the crown is formed. The development of enamel is sensitive to various environmental injures. Amelogenesis is genetically a controlled process and divided into 4 stages. In the first stage – presecretory the preameloblasts differentiation occurs. During the second stage – secretory stage the full thickness of enamel is deposited starting from the cusp tips or incisal margin. Disturbances in the secretory stage cause a reduced enamel thickness – the hypoplastic defect. During the third stage – transition stage – the enamel matrix degradation along with massive mineralization is performed

Table 4. Development of teeth affected by MIH

Tabela 4. Rozwój zębów objętych MIH

Tooth	Begin of calcification	Completed crown	Eruption
Primary second molars*	18 weeks in utero	8–11 months	23–33 months
Permanent first molars**	32 weeks in utero	3¼–4½ years	6–6¼ years
Central incisors**	3 months	3½–4½ years	6¼–7¼ years
Lateral incisors**	3–11 months	4–5½ years	7½–8 years

* – according to Schroeder [37]; ** – according to Weerrhejim et al. [3].

and during the last one, named maturation stage, the final mineralization occurrs. Abnormalities in those stages cause hypomaturation or hypomineralization of enamel [37, 38]. Fagrell et al. [39] estimating the onset and timing of the MIH found that the ameloblasts producing the hypomineralized enamel are able to form an enamel of normal thickness but reveal a significantly reduced capacity for enamel maturation. Moreover, they estimated that the time for the disturbance occurrence was the first 6–7 months of age.

The mineralization periods of permanent first molars, permanent incisors and primary second molars overlaps (Table 4). Hence, common shared causes of the hypomineralization for these teeth are suggested [31].

Etiologic Factors

The etiology of molar-incisor hypomineralization is unclear probably because the abnormality is not caused by one specific factor. Many environmental conditions may have a harmful effect, leading to an increase of MIH development or they can act together additively or synergistically. Numerous retrospective studies have analyzed systemic conditions and environmental factors influencing prenatal, perinatal and postnatal development of teeth. The prenatal period is critical for enamel defects in primary teeth and permanent first molars. Putative causative factors are presumed to be prolonged maternal diseases (viral infections, hypertension, diabetes, renal insufficiency, vomiting, and malnutrition) and medication. The common conditions occurring in perinatal period as caesarian section, complicated delivery, premature birth, low birth weight, and twinning can disturb ameloblast function [5, 6, 40]. Postnatal medical problems during the first years of life as prolonged childhood diseases (respiratory problems, asthma, bronchitis, otitis), high fever due to infections and medication (amoxicillin, erythomycin) as well as exposure to environmental contamination via mother's milk (dioxins, polychlorinated biphenyls) may be associated with the development of MIH defects [5, 6, 26, 38]. However, some studies suggested a genetic factor responsible for MIH development. Kühnisch et al. [32] have identified a possible locus linked to MIH.

Treatment

The treatment options are related to the severity of enamel defects and they range from prevention, reconstruction to extraction. Preventive measures involve remineralization with the use of fluoridated toothpaste and topical application of fluorides (fluoride varnish), pit and fissure sealants, oral home care program as well as the reduction of diet cariogenicity and erosivity. Application of specimens containing casein phosphopeptide – amorphous calcium phosphate without (CPP-ACP) or with fluoride (CPP-ACPF) can help in mineralization of the hypocalcified areas and reduce sensitivity. Resin-based fissure sealants are suitable for erupted molars with adequate moisture control but for unerupted teeth or with poor moisture control as interim option is used glass ionomer sealant that will be replaced later by resin-based sealant. The goals of the preventive measures are to reinforce hypermineralized enamel, prevent post-eruptive breakdown and caries development. Topical application of fluoride is helpful in diminishing the sensitivity of the affected teeth as well.

The aesthetic appearance of MIH incisors can be improved by bleaching and/or acidic microabrasion procedures as well as by direct and indirect composite veneers replaced by ceramic as the child ages.

After post-eruptive enamel breakdown for rebuilding the lost hard dental tissues, glass ionomer cement restorations, composite resin restorations, performed metal crowns and cast restorations can be used. Glass ionomers are recommended in conditions of poor moisture control but not in stress loading areas due to their weak mechanical properties. Therefore, they can only be used as a temporary restoration or as a base material for resin composite restoration. The margins of fillings should be monitored for further post-eruptive enamel breakdown. The performed stainless steel crowns are advocated for teeth with large loss of tooth structure.

In the case of unrestorable decay in the crown or poor long-term prognosis, the tooth extraction is the last treatment option. However, the decision should be preceded by taking into consideration the possible orthodontic complications [5, 40].

Conclusions

The prevalence of molar-incisor hypomineralization seems to be an increasing problem in paediatric population. The dentists should be able to diagnose this condition and monitor with greater care, so that the early post-eruptive loss of dental structure as well as the early carious involvement can be prevented.

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