

Molar incisor hypomineralization: A complex puzzle

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Abstract

Molar incisor hypomineralization (MIH) was first described in 2001 as a developmental enamel defect characterized by hypomineralization, typically affecting incisors along with at least one first permanent molar. According to current systematic reviews and meta-analyses, its global prevalence ranges between 13.1% and 14.2%. Although research on its etiology is ongoing, various prenatal, perinatal, and postnatal factors originating from the mother and child, as well as environmental and genetic/epigenetic factors, are thought to contribute to this condition. The affected teeth exhibit white/creamy and yellow/brown demarcated opacities, post-eruptive enamel breakdown, atypical dental caries and restorations, hypersensitivity, and aesthetic problems. Consequently, the teeth are quickly damaged, and their functions are compromised. This condition also affects the psychosocial development and quality of life of affected individuals. Despite numerous treatment methods being tried on anterior and posterior teeth affected by MIH, research continues to progress with advancements in material science and a better understanding of the disease's pathophysiology. Studies have not only focused on materials but have also explored different caries removal techniques and preparation methods for the affected teeth. While understanding and consensus on certain aspects of MIH have increased since it was first described, many issues remain unresolved. These include the regional and global prevalence of the disease, its etiology, treatment options for affected anterior and posterior teeth, and the short- and long-term success of these treatment materials and methods. This article aimed to comprehensively discuss the "still to solve" issues related to MIH as a complex puzzle and provide up-to-date information on these topics.

Keywords: Developmental defects of enamel, enamel hypomineralization, first permanent molar, molar incisor hypomineralization, prevalence, remineralization, treatment

1. Introduction: Definition, characteristics, and clinical symptoms

Molar incisor hypomineralization (MIH), officially described for the first time in 2001 by Weerheijm et al. (1), is defined as a qualitative developmental enamel defect characterized by reduced inorganic content and calcification rate of the enamel, affecting incisors along with at least one first permanent molar. The condition, previously known by various names such as non-fluoride enamel opacities, non-endemic mottling of enamel, idiopathic enamel hypomineralization, cheese molars, and fever hypoplasia, was named MIH after the pathophysiology of the defects in the affected teeth was determined (2). Although it can often be confused with hypoplasia, as seen in its older definitions, different characteristics are observed in teeth depending on the stage of tooth development at which the damage occurs (3). Hypoplasia is a quantitative defect resulting from damage during the phase of enamel matrix secretion in amelogenesis. In hypoplasia, the enamel's calcification is usually unaffected, but the amount and thickness of the enamel are reduced. In contrast, hypomineralization results from damage during the calcification phase of tooth development, leading to qualitative enamel defects (3).

In MIH-affected teeth, there is often no reduction in the amount of enamel, but a deterioration in inorganic content, hardness, and elasticity is observed. Enamel with MIH-specific findings exhibits distinct microscopic and macroscopic features compared with healthy enamel (4). Due to calcification problems, the mineral density of these teeth decreases by up to 20% (5). This reduction is typically due to a decrease in the amount and ratio of calcium (Ca) and phosphorus (P). The reduced amounts of these two elements, along with an increase in carbon (C) content in the enamel, make the enamel more vulnerable to chewing forces. The defective enamel structure causes it to erode and disintegrate easily under chewing forces over time (6). This condition, specific to MIH, is called post-eruptive enamel breakdown and can occur very soon after the eruption, depending on the severity of the defect (7) (Fig. 1). The decrease in inorganic content leads to a proportional increase in the organic content of these teeth. Proteomic studies have shown that the amount and ratio of various non-enamel-specific proteins are higher in hypomineralized enamel (8, 9). The increased organic content and decreased inorganic content may necessitate different treatment approaches and influence the severity and timing of disease-specific symptoms observed in the teeth. For instance, the increase in organic content results in more resistance to acid etching, altering the etching technique, timing, and pattern in hypomineralized enamel (10).



Figure 1. Post-eruptive enamel breakdown in the distal fossa of the occlusal surface of the upper right first permanent molar tooth affected by MIH and yellow/brown demarcated opacities in the entire tooth (This image has been taken from Dr. Berkant Sezer's patient archive).

Many disease-specific findings can be observed in teeth with MIH, ranging from mild to severe, depending on the defect's severity. The mildest and most common finding in the first permanent molars and incisors is demarcated opacities, which may be white/creamy and/or yellow/brown (Fig. 2). As the color darkens, the enamel's hardness decreases, indicating increased severity of the defect (11, 12). The enamel, with reduced inorganic content, allows factors such as hot/cold stimuli and/or microorganisms and their products to penetrate the underlying layers more easily, influenced by the wide dentinal tubules just below it. Consequently, affected teeth exhibit hypersensitivity of varying severity and atypical caries, which are specific to this rapidly progressing disease (13, 14) (Fig. 3). Additionally, obtaining local anesthesia in teeth affected by MIH is challenging because the dental pulp becomes defensive against stimuli due to chronic inflammation (15). Another diagnostic criterion for affected teeth is the presence of atypical restorations. Although detailed information about restorations for these teeth is provided in the following sections of the article, differences in the appearance of the restoration occur over time as the defective enamel continues to wear away, even after the restoration. Due to bonding issues with defective enamel, restoration fractures, losses, and secondary caries may also be observed. Moreover, tooth loss due to MIH, aesthetic problems from lesions in the anterior teeth, decreased quality of life due to advanced hypersensitivity, and other symptoms are additional diagnostic criteria for the disease (11, 12).

Various diagnostic indices and criteria have been developed to evaluate MIH. The signs and symptoms that may be encountered in patients with teeth and MIH are systematically and comprehensively outlined.

accompanied by post-eruptive enamel breakdown and atypical caries, persistent hypersensitivity that affects function and oral care practices, and significant aesthetic concerns that have a negative socio-psychological impact (12) (Table 1).



Figure 2. Post-eruptive enamel breakdown and yellow/brown demarcated opacities on the buccal surface of the upper left permanent central tooth affected by MIH (This image has been taken from Dr. Berkant Sezer's patient archive).



Figure 3. Atypical caries that progresses rapidly following eruption in the lower right first permanent molar tooth affected by MIH (This image has been taken from Dr. Berkant Sezer's patient archive).

The severity of the affected teeth is divided into two categories: mild and severe. The condition is considered mild if there are white/creamy and/or yellow/brown demarcated opacities without post-eruptive enamel breakdown, decreased hypersensitivity to external stimuli, and minimal aesthetic concerns due to the lesions on the incisors. Conversely, the condition is described as severe when demarcated opacity is

Table 1. Classification of MIH according to severity (12).

Severity level	Signs and symptoms
Mild	Demarcated opacities without enamel breakdown, Decreased hypersensitivity to external stimuli, e.g., air, water, but not brushing Mild aesthetic concerns on the discoloration of the incisors.
Severe	Demarcated enamel opacity with post-eruptive enamel breakdown and caries, Spontaneous and persistent hypersensitivity affecting function, e.g., brushing practices, Strong aesthetic concerns that may have a socio-psychological impact

An Australian working group (16) was the first in 2014 to combine the defect size of individual teeth and the entire dentition with a treatment recommendation by introducing the molar hypomineralization severity index (MHSI). Nevertheless, a distinction was made only between mild, medium, and severe MIH cases. There is still a missing connection between a standardized, simple assessment criterion that represents defect size and hypersensitivity in single teeth and the entire individual's oral health, along with a treatment recommendation (17). Such an index can then be used

for epidemiological studies on data collection as well as for individual patients for the assessment of the condition and treatment planning. The newly developed the MIH Treatment Need Index (MIH-TNI) can be used to investigate larger populations in a standardized way and to use standardized therapy procedures in MIH patients (18). MIH-TNI from 0 to 4, according to a combination of absence/presence of sensitivity, presence/absence of enamel defect, and extension of the defect (18). The different codes and their descriptions of the index are presented in Table 2.

Table 2. MIH-TNI codes and descriptions (18).

Index	Definition
Index 0	No MIH, clinically free of MIH
Index 1	MIH without hypersensitivity, without defect
Index 2	MIH without hypersensitivity, with defect
2a	<1/3 defect extension
2b	>1/3<2/3 defect extension
2c	>2/3 defect extension or/and defect close to the pulp or extraction or atypical restoration
Index 3	MIH with hypersensitivity, without defect
Index 4	MIH with hypersensitivity, with defect
4a	<1/3 defect extension
4b	>1/3<2/3 defect extension
4c	>2/3 defect extension or/and defect close to the pulp or extraction or atypical restoration

2. Differential diagnosis of MIH

The differential diagnosis of MIH includes enamel hypoplasia, amelogenesis imperfecta, dental fluorosis, and white spot lesions. An accurate differential diagnosis is crucial, especially when dental caries is extensive enough to mask disease-specific findings (12).

Enamel hypoplasia is often localized with irregular lesion borders and is characterized by a decrease in the amount and thickness of enamel, making it a quantitative defect (19). In contrast, MIH typically affects multiple teeth and regions within these teeth. MIH lesions have sharp boundaries and are described as demarcated opacities. Despite having normal enamel quantity and thickness, the enamel's calcification is defective, classifying MIH as a qualitative disorder (12).

Amelogenesis imperfecta is a genetic disorder that can present as hypoplastic and/or hypocalcified enamel (20). While MIH primarily affects the permanent dentition, lesions similar to MIH are also observed in primary second molars, known as hypomineralized second primary molars (21). Given the similarity in lesion characteristics and timing of etiological exposure during tooth development, hypomineralized second primary molars are often discussed alongside MIH (21). Both amelogenesis imperfecta and MIH can affect primary and permanent dentition. However, MIH typically affects a specific group of teeth (first permanent molars and often incisors), whereas amelogenesis imperfecta affects all teeth in the dentition. Amelogenesis imperfecta has a clear genetic transmission and family history, whereas the etiology of MIH remains unclear, despite possible genetic factors (12, 20).

Dental fluorosis results from chronic exposure to high fluoride levels during tooth formation and development. These lesions are symmetrical, affect the entire dentition, lack clear borders, and are irregular (22). In contrast, MIH affects only a specific group of teeth, and the lesions need not be symmetrical. The demarcated opacities in MIH-affected teeth have regular

borders. High fluoride exposure is a critical factor in differentiating dental fluorosis from MIH (12).

White spot lesions are the early stage of caries caused by high consumption of fermentable carbohydrates and inadequate oral hygiene (23). The primary difference between MIH lesions and these lesions is their location (12). White spot lesions are often found in the cervical region of the anterior teeth, areas prone to dental plaque accumulation (23). In contrast, MIH-demarcated opacities are more commonly found in the middle and incisal regions. Additionally, MIH lesions can appear yellow or brown, whereas white spot lesions are typically white or cream. White spot lesions also have more irregular borders compared to MIH lesions (12).

3. Oral health-related quality of life (OHRQOL) and MIH

OHRQoL includes a set of indicators that determine the impact of oral and dental conditions on a person's life. These indicators measure oral health, well-being, expectations, and satisfaction with dental care received, and the individual's self-concept. Studies have shown that low income, poor sociocultural status, orthodontic and periodontal problems, and the presence of oral health problems such as dental caries are associated with reduced OHRQoL (24). Although some studies have evaluated the OHRQoL in individuals suffering from MIH, it can be said that the OHRQoL in these individuals is generally lower than their healthy peers (24). The basis of this decrease is problems such as dental caries, enamel breakdown, and pain in the posterior teeth affected by moderate and severe MIH, or aesthetic problems in the anterior teeth. Various studies have also reported that OHRQoL in these individuals increases with the elimination of MIH-related symptoms (25).

To date, numerous scientific studies have been conducted on MIH. These include case reports and/or

series, retrospective and/or prospective cohort studies, in vitro and in vivo studies, randomized clinical trials, and systematic reviews and meta-analyses derived from these studies. All these studies provide a wealth of information that is close to being exact today, as presented in this section. However, many "still to solve" issues regarding MIH remain, and research is ongoing. These unresolved issues, which are addressed in the following sections of the article, include the prevalence and etiology of the disease and the management of affected anterior and posterior teeth.

4. Prevalence of MIH

In the more than 20 years since the generally accepted and official definition of MIH was announced, considerable effort has been devoted to assessing the prevalence of MIH. Meanwhile, different observational studies have been conducted in smaller regions within countries and across continents. Unfortunately, the issue of both regional and global prevalence continues to be up-to-date as a "still to solve" issue.

From a broader perspective, two main prevalence studies have provided fundamental data by evaluating the global prevalence of MIH using advanced statistical methods. The first of these comprehensive studies, published in 2018, analyzed data from 70 different prevalence studies and reported a pooled global prevalence of 14.2% (26). Continent-based subgroup analyses revealed prevalence rates of 18% in South America, 16.3% in Oceania, 14.3% in Europe, 13% in Asia, and 10.9% in Africa, with no prevalence data available for North America. This study also found no statistically significant difference in prevalence between females and males (26). The second study reported a mean global prevalence of 13.1% (27). It reported statistically significant differences in prevalence between super-regions, regions, and countries and estimated the mean number of global incident cases in 2016 to be 17.5 million (27).

A newly published meta-analysis investigating whether the prevalence of MIH has increased over time determined the weighted mean prevalence of MIH to be 12.8% (28). Although the term MIH has a history of approximately 20 years, these types of lesions have been known for many years. Considering this, the lesion rate, which was 3% in 1992, increased to 13% in 2013 (28). Inferring from this subgroup result obtained from some prevalence studies included in the meta-analysis, it can be said that the prevalence of MIH is gradually increasing. However, as the authors particularly emphasize, although the prevalence appears to be increasing, the risk of bias and heterogeneity in the studies add to the uncertainty (28).

Cross-sectional, longitudinal, and retrospective prevalence studies conducted at different times cannot provide clear data on the global prevalence of MIH, and the existing data seem insufficient (28). Many regions, countries, and even continents have not yet conducted

prevalence studies (28). Another reason for the varying results of studies conducted in different years and regions, and even within the same region on similar dates, appears to be the examiners themselves. The level of knowledge of examiners about MIH and the necessity of a detailed examination for diagnosing even the smallest lesions raise doubts about the accuracy and clarity of the prevalence results. Additionally, the populations included in the studies may be from specific cohorts, which might obscure the true prevalence, especially considering the etiological factors of MIH, such as environmental factors. Considering all these factors, as examiners' knowledge about MIH increases and populations are examined with larger sample groups, we can reach more accurate conclusions about the true prevalence of MIH.

5. Etiology of MIH

It has long been a matter of curiosity why a group of lesions that only affect the first permanent molars and often the permanent central and lateral incisors are observed only in these teeth. Many etiological studies have been conducted to understand the cause of the disease, which has become an increasingly important public health problem within the scope of public oral and dental health. Currently, the number of these studies is increasing, and they are gradually evolving from observational and clinical studies to genetic and epigenetic studies.

The enamel development of the first permanent molars and permanent incisors corresponds to approximately the same chronological time. Systemic, environmental, and/or genetic disorders that occur during this period are likely to cause disruptions in amelogenesis and defects in the structure of the enamel. In the scientific literature, various prenatal, perinatal, and postnatal factors, as well as genetic, epigenetic, and environmental determinants related to the mother and child have been emphasized. However, as clearly stated in the latest and current guideline of the European Academy of Paediatric Dentistry (EAPD), the exact etiological factor of MIH remains unclear (12, 29).

The EAPD guideline was prepared as a result of a comprehensive meta-analysis, which concluded that no prenatal disease or condition can definitely cause MIH (30). A strong relationship could not be established between the medications used by the mother during pregnancy and/or alcohol and/or cigarette use and MIH. Mothers of children with MIH symptoms are more likely to have systemic diseases; however, the sample numbers in the studies, heterogeneity, and most importantly, determining the diseases as a result of retrospective interviews create a significant level of bias (12). In contrast, the presence of perinatal factors in the etiology of MIH seems more significant than prenatal factors. Deviations in birth time, low birth weight, cesarean section, and birth complications, but most importantly,

birth hypoxia, have been reported to be significantly higher in children with MIH (approximately 2.76 times for birth hypoxia) (30). Although it does not carry as high a risk as birth hypoxia, it has been observed that prematurity and cesarean section also create a significant risk increase of approximately 1.45 times (30). Although there are differences in these issues in different systematic reviews and meta-analyses, this situation is related to the number and types of studies included in these reviews. It has been reported that medications and diseases in children postnatally, especially during the first three years of life, can be found in the etiology of MIH. Although it has been reported that common diseases in childhood, such as measles, chronic urinary tract infections, bronchitis, otitis, gastrointestinal diseases, high fever, chronic renal failure, pneumonia, and asthma, may cause MIH, the characteristics of the studies included in the reviews should also be questioned (30). Although it has been reported that medications, especially antibiotics, may be involved in the etiology of MIH, it is still unclear whether the disease or the medication causes MIH, as antibiotics are preferred in the treatment of the above-mentioned diseases (12). As studies are often methodologically conducted in the form of retrospective narrative inquiries, there is also a risk of bias. Another issue that has been researched is environmental pollutants, and it has been reported that environmental pollutants, especially in breast milk, may increase the risk of MIH. However, current meta-analyses show that no factor related to the breastfeeding process significantly affects the risk of MIH (30, 31).

In recent years, studies aimed at determining the etiology of MIH have increasingly focused on the genetic and epigenetic factors. Different results have been reported regarding the genetic origin of susceptibility, including studies on various genes, single nucleotide polymorphisms (SNPs), epitopes, alleles, and chromosomes. Notably, the presence of the rs5979395 SNP in the AMELX gene has been reported to significantly increase the risk of MIH by approximately 11.7 times (32). More recent epigenetic studies suggest that gene-environment and/or gene-gene interactions may also play a role in the risk of developing MIH (12, 33). Although these studies seem to support the involvement of genetic and epigenetic factors in the etiology of MIH, it is scientifically more accurate to consider MIH a multifactorial condition. Nevertheless, the precise etiology of MIH remains a "still to solve" issue that continues to elude definitive scientific clarification.

6. Management of posterior teeth affected by MIH

Many treatment approaches have been used for posterior teeth affected by MIH, ranging from minimally invasive techniques to tooth extraction. Although randomized controlled studies frequently evaluate the short- and

long-term success of different materials and techniques, the evidence regarding the ideal material and technique remains insufficient in many respects.

Over the past decades, specific guidelines have been developed to improve knowledge regarding the diagnosis, clinical features, and treatment modalities of MIH-affected teeth. Due to the unsatisfactory data situation on the epidemiology, etiology, and therapy of MIH, the recommendations are partly contradictory, which could influence the dentists' knowledge differently. Simultaneously, compliance with the guidelines and their successful implementation is not yet fully known. Management of patients with MIH is complicated by the penetration of bacteria through the often-exposed dentinal tubules at the level of the hypomineralized lesion. This bacterial infiltration is responsible for subclinical inflammation of the pulp and hypersensitivity, which leads to negligence of oral hygiene by the child and a higher anesthetic threshold. Treatment approaches for posterior teeth affected by MIH can be classified into three groups: non-invasive, micro-invasive, and invasive.

Non-invasive treatment approaches include the use of casein phosphopeptide amorphous calcium phosphate (CPP-ACP) (RECALDENT™; GC Toothmousse®, GC MI Paste®, GC MI Varnish®, GC Europe, Leuven, Belgium) and casein phosphopeptide amorphous calcium fluoride phosphate (CPP-ACFP) (RECALDENT™; GC MI Paste Plus®, GC MI Paste® ONE, GC Europe, Leuven, Belgium) or calcium glycerophosphate-included (CaGP) (R.O.C.S. Medical Minerals Gel®, R.O.C.S. Trading GmbH, Munich, Germany) containing gels.

Micro-invasive approaches include pre-treatment of enamel for 60 seconds with 5% sodium hypochlorite, treatment of surface 6% hypochlorite before and after etching, bleaching with 5% sodium hypochloride for 5-10 minutes, re-etching and covering with fissure sealants, bleaching with carbamide peroxide, microabrasion using abrasive pastes and 18% hydrochloric acid, glass ionomer and resin-based fissure sealants, polishing with pumice and conditioning with 37% orthophosphoric acid for 60 seconds.

Invasive approaches include atraumatic restorative treatment protocol, hybrid glass ionomer restorations, glass ionomer cements, application of glass ionomer restorations with fluoride varnishes, resin-modified glass ionomers, amalgam restorations, composite resin restorations, direct and indirect restorations, prefabricated metal crowns, composite/ceramic/gold/cast metal crowns, CAD/CAM systems, and extractions (34, 35).

Due to the weakened hypomineralized enamel structure, posterior teeth affected by MIH are highly susceptible to dental caries and post-eruptive enamel breakdown (36). Therefore, early diagnosis and implementation of necessary preventive measures are crucial. Individuals may avoid oral hygiene practices due to heightened sensitivity, which can accelerate dental caries progression. To mitigate this, nutritional and oral hygiene recommendations for both children and families can help reduce such risks (12). Additionally, frequent

topical fluoride applications (gels, varnishes, etc.) are recommended to prevent dental caries and alleviate hypersensitivity (37). However, evidence regarding the use of topical fluoride or silver diamine fluoride to halt enamel breakdown post-application is limited, thus precluding strong recommendations (12). Resin-based pit and fissure sealants applied to the posterior teeth, where the occlusal surfaces and fissures are entirely within the mouth, effectively reduce the risk of dental caries and strengthen the teeth (12,38). When applying resin-based pit and fissure sealants on posterior teeth affected by MIH, using an adhesive after etching is a recommended approach supported by high-level evidence, as it enhances retention (12).

In cases where cooperation issues with the child are present, local anesthesia cannot be applied effectively, or access to rotary instruments or polymerization devices is limited, glass ionomer-containing materials can be used to treat these teeth using an atraumatic restorative treatment approach (39). While they can serve as temporary restorative materials until a suitable restoration is placed or until the child reaches an appropriate age for tooth extraction or to relieve hypersensitivity (40-42), some studies have explored their use as permanent restorations following selective caries removal (43). However, due to varying study results and biases, there is no strong evidence supporting the use of glass ionomer-containing materials in posterior teeth affected by MIH (12). Despite their promising properties, such as remineralization and reduction of tooth decay, especially in the context of the growing popularity of minimally invasive dentistry, further research is necessary to better understand their efficacy, specifically in posterior teeth affected by MIH.

Composite resins, typically the preferred material in modern restorative dentistry, can yield successful outcomes in posterior teeth affected by MIH when applied with good isolation. The simplicity of the technique, which is often achievable in a single appointment, presents a significant advantage. However, unlike composite resin restorations in non-hypomineralized teeth, different application techniques are recommended for teeth affected by MIH. Despite advancements in minimally invasive dentistry and adhesive technology promoting minimal tooth structure removal, a noninvasive approach is discouraged for MIH-affected teeth because of the reduced bond strength with hypomineralized enamel (12, 44). While some studies suggest successful outcomes with minimal tooth preparation using glass ionomer and composite resin restorations (43-48), current systematic reviews and meta-analyses based on evidence recommend composite resin restorations in posterior teeth affected by MIH for three primary reasons. First, an invasive approach is advised for teeth with mild to severe defects under optimal isolation conditions. Second, noninvasive composite resin restorations are not recommended for these teeth. Third, deproteinization using self-etch, total-etch, or sodium hypochlorite prior to restoration is recommended (12, 44).

Stainless steel crowns are commonly favored in pediatric dentistry because of their affordability, durability, ease of application, and ability to be completed in a single session. However, their aesthetic properties can sometimes limit their use. They provide successful outcomes by alleviating hypersensitivity and restoring the function of teeth affected by MIH (49, 50). While preparation and application to the affected tooth may be necessary in some cases, they can also be applied using techniques such as the Hall technique (51). The recommendation for stainless steel crowns in posterior teeth affected by MIH is strong because of their demonstrated success. However, the level of evidence supporting their use is moderate, primarily due to the limited number of studies and sample sizes (12, 44).

In recent years, there has been an increase in studies exploring laboratory-produced indirect restorations/crowns for posterior teeth affected by MIH, aligned with the advancements in prosthetic and digital dentistry (52, 53). Nevertheless, the current literature lacks sufficient studies to establish clear success criteria, and existing studies exhibit biases and heterogeneity, further limiting recommendations (12, 44). Moreover, in pediatric dentistry, their use may be restricted because of requirements such as technique-sensitive tooth preparation, higher costs, and the need for laboratory and technician resources. Therefore, the quality of evidence supporting these restorations is moderate, similar to that of other types of restorations, and their recommendation remains conditional (12).

Scheduled extractions may be necessary, particularly for teeth with abscesses, cellulitis, extensive coronal damage unsuitable for restoration, or severely affected pulp structures (12, 44). When the long-term prognosis of repeated treatments is uncertain, this approach can impact the child and family psychologically and pose financial challenges. Factors such as the child's occlusal relationship, degree of hypomineralization, amount of overjet and overbite, presence of crowding and diastema, hypodontia/oligodontia status, presence of third molars and other supernumerary teeth, dental age, cooperation, systemic health, and presence of malocclusion should all be considered when making extraction decisions (12, 44). Owing to the varying study outcomes, offering a definitive recommendation on this matter is challenging.

In posterior teeth affected by MIH, ensuring preservation of the defective tooth, restoration of dental caries and hypomineralized defects, and elimination of hypersensitivity are crucial for maintaining dental arch integrity, occlusal relationships, function, aesthetics, phonation, and supporting the child's sociopsychological development in the short and long term. Despite numerous studies of varying designs evaluating the success of different restorative techniques and materials for these teeth, current systematic reviews and meta-analyses indicate that none achieve a high-quality evidence rating (12, 44). Therefore, the issue of restorative treatment for posterior teeth affected by MIH remain to be solved.

7. Management of anterior teeth affected by MIH

Diagnosing MIH requires the identification of hypomineralized defects in at least one first permanent molar tooth. However, studies have indicated that permanent incisors are frequently affected by MIH because of the close timing of calcification. These lesions on the affected incisors often present as white/creamy and/or yellow/brown demarcated opacities, increasing the risk of enamel fracture when near the incisal edge (12, 44). Apart from aesthetic concerns due to changes in appearance, these issues can impact the child's psychosocial development and quality of life (12, 43, 53). In severe cases, these lesions may also cause hypersensitivity. Despite the known impact of aesthetic changes on psychosocial development, clinical studies specifically focusing on incisors affected by MIH are unfortunately limited (12, 44, 53).

Studies have demonstrated the advantages of various products aimed at strengthening mineral structure, not only in anterior but also in posterior teeth, to alleviate hypersensitivity and enhance enamel strength. Research in this area has explored options such as CPP-ACP, CPP-ACFP, CaGP, tricalcium phosphate, arginine, calcium carbonate, and low-level laser treatment, showing that these agents effectively improve mineral content (55-59). However, these recommendations are conditional due to the limited number of studies, heterogeneity among them, inherent biases, and other methodological issues (12). CPP-ACP and CPP-ACFP can accelerate and increase the potential maturation of enamel affected by MIH and improve the mechanical properties of this enamel after tooth eruption. The transfer of Ca and phosphate to demineralized and hypomineralized tissues through casein binding reduces surface porosity and minimizes the risk of caries and hypersensitivity (56). When CaGP adsorbs onto enamel surfaces with the phosphate it contains, almost all phosphate groups are neutralized by the Ca and hydroxyl groups of hydroxyapatite molecules, thus releasing calcium ions from CaGP onto enamel surfaces. Thus, the hardness of the enamel is increased, the risk of caries is reduced, and remineralization is achieved (56). Although there are differences between the mechanisms of action of different products, the evidence quality for CPP-ACP use is moderate, and the evidence for other products remains low or very low. This highlights the need for increased prospective randomized clinical studies to further validate the beneficial outcomes observed with these materials (12, 44).

The methods commonly preferred for the restorative and aesthetic treatment of anterior teeth affected by MIH include microabrasion, resin infiltration, external bleaching, etch-bleach-seal technique, composite resin restorations, and combinations thereof (60-66). While the number of randomized controlled trials testing these techniques has increased in recent

years, the current evidence remains inadequate. Resin infiltration is gaining popularity because of its simplicity and adherence to minimally invasive dentistry principles. However, most published literature on aesthetic and restorative treatments for anterior teeth affected by MIH consists of case reports or series, limiting both the strength of recommendations and the quality of evidence (12, 44). Nevertheless, clinical studies and case reports/series have generally reported positive outcomes in terms of aesthetic improvement and enhanced quality of life (QoL) (61). According to the latest guidelines from the EAPD, although treatment approaches for MIH-affected incisors are conditionally recommended, the evidence quality from relevant studies is generally low or very low (12, 44). This underscores the urgent need for further research on MIH treatment, given its significant impact as a public health concern affecting nearly one in seven children today (66). Especially in an era marked by increasing peer bullying and rapid regional and global digital aesthetic standards, the impact of MIH lesions on a child's psychosocial development should be carefully considered (66, 67). Managing treatment in anterior teeth remains a pressing challenge, with much yet to be resolved compared with posterior teeth.

8. Conclusion

MIH represents a significant public oral health challenge, with studies indicating an increasing prevalence since its initial identification. Research across various domains, including etiology, treatment, prevalence, and microscopic analysis, is expanding. While there is greater consensus on aspects such as its definition, diagnostic criteria, microscopic and macroscopic features, and the composition of affected enamel (both inorganic and organic), consensus remains lacking on the disease's prevalence, etiology, and optimal management of the affected anterior and posterior teeth. The variability in bias levels, study designs, sample sizes, and heterogeneity among studies underscores the need for more randomized controlled trials and prospective cohort studies with minimal bias, heterogeneity, and methodological flaws. Advances in scientific research hold promise for resolving these "still to solve" issues, paving the way for improved understanding and management of MIH in the future.

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