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Case Report

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Clinical Case Report of Molar-Incisor Hypomineralization in a Pediatric Patient With Affected First Permanent Molars in Dental Practice: Discussion of Etiology and Management

Champilomati Athanasia[1], Kosidou Georgia[2]

[1] Postgraduate Student, MSc "Research in Dentistry," Faculty of Dentistry, International University of Catalonia, Barcelona, Spain

[3] MSc Endodontics, Graduate of School of Health Sciences, Aristotle University of Thessaloniki, Thessaloniki, Greece

Abstract

Molar-incisor hypomineralization (MIH) is a qualitative enamel defect compromising tooth structure, often leading to complications such as caries, hypersensitivity, and extensive restorative treatment needs. This case report discusses a 15-year-old patient with MIH affecting all four first permanent molars, presenting varying degrees of enamel damage. Tooth 36 exhibited pulp necrosis, necessitating endodontic treatment and prosthetic restoration, while conservative approaches were implemented for the other affected molars. The case emphasizes the critical importance of early diagnosis, preventive care, and individualized management strategies to minimize MIH-related complications. A brief literature review explores the etiology, clinical presentation, and management strategies of MIH. The systemic origins and multifactorial nature of MIH, combined with its significant impact on oral health, highlight the necessity for interdisciplinary collaboration in patient care. Further research is required to clarify the condition's etiology, optimize treatment approaches, and improve clinical outcomes for MIH-affected individuals.

Keywords: Molar-Incisor Hypomineralization (MIH), Dental Hypomineralization, Enamel Defects, Pulpal Necrosis, MIH Etiology, MIH Management.

INTRODUCTION

Molar-incisor hypomineralization (MIH) describes a qualitative defect in the enamel of both primary and permanent teeth. Due to inadequate mineralization, teeth affected with MIH are vulnerable to breakdown of tooth structure, caries, sensitivity, tooth loss. and subsequent orthodontic problems [1]. The term molar-incisor hypomineralization (MIH) was first introduced in 2001 to describe a clinical form of enamel hypomineralization of systemic origin, affecting one or more permanent first molars (PFMs) and permanent maxillary incisors [2]. MIH may involve anywhere from one to all four PFMs[2]. The likelihood of MIH affecting the permanent maxillary incisors increases as the number of PFMs affected rises [3]. Additionally, reports have indicated that second primary molars, second permanent molars, and the tips of permanent canines can also be affected [4].

MIH is a common dental condition, impacting up to 25% of children globally [4]. Its precise etiology remains uncertain [2]. Enamel hypomineralization, in general, results from disruptions in ameloblast cell function during any stage of amelogenesis. However, MIH is believed to result from multifactorial systemic factors contributing to enamel hypomineralization [5]. Studies indicate that children born prematurely or experiencing poor general health early in life are at a higher risk of developing MIH [4,6]. The use of antibiotics during early life has also been associated with MIH, though it remains unclear whether the condition is due to the antibiotic itself or the underlying illness being treated [6]. Clinically, molar-incisor hypomineralization (MIH) typically presents as discrete, opaque lesions that range in color from white to yellow-brown, which are distinct from the diffuse linear opacities characteristic of fluorosis. MIH is often accompanied by post-eruptive enamel loss, which can make it challenging to differentiate from enamel hypoplasia [7].

Teeth affected by MIH may exhibit heightened sensitivity to temperature changes and tooth brushing, even when the enamel appears clinically intact [8]. Additionally, MIH is frequently associated with various dental complications, including the rapid progression of carious lesions and a recurring need for dental treatment

***Corresponding author:**
Dr. Champilomati Athanasia
Postgraduate Student, MSc "Research in Dentistry," Faculty of Dentistry, International University of Catalonia, Barcelona, Spain
Email:
drnancychamp@gmail.com

CASE REPORT

A fifteen years old girl was referred to the dental practice for endodontic treatment of tooth 36 which was diagnosed with pulp necrosis. During dental examination molar incisor hypomineralization was noted in all four first molars with different extent to each one of those. According to EAPD scoring criteria for MIH.

16: code 2a (yellow/brown demarcated opacities with post eruptive enamel breakdown) , 26: code 2a , 36: code 3 (atypical restoration), 46: code 3 [11].The minimum size of lesions included in the diagnosis of MIH is also inconsistent between studies. While some studies included only defects larger than 2 mm, others have included all hypomineralized lesions regardless of their size. The exclusion of smaller defects might have thus contributed to lower prevalence rates in some reports [12].

Medical history was obtained from the girl's mother in the form of a questionnaire where the following were noted: During mother's pregnancy there were no general health problems (e.g. infections, hypertension, diabetes, fever). The mother didn't take any medications during the pregnancy and she was neither exposed to toxic factors (e.g. diphenol A) or radiation. She also reports that she gave birth naturally without complications at 38 weeks and 4 days and the birthweight of her daughter was 2.6 kg. Breastfeeding lasted four months and the introduction to solid foods started at 6 months of age. Child's general health during development was normal without fever episodes or repetitive fever episodes. The mother also adds that her daughter received vitamin D3 supplements. The patient doesn't have any past dental trauma history but she has lower limb's hypertonia and glucose 6 phosphate dehydrogenase deficiency. No family history of other or similar dental defects. Patient's oral hygiene is insufficient, while she did not report hypersensitivity during brushing the hypomineralized molar, neither thermal changes are bothering her. She is undergoing orthodontic treatment and her orthodontist reports that she firstly came to his dental practice with severe class II (Angle). No orthodontic rings or brackets were placed on the first molars due to their severe hypomineralization.

The patient was reported to the dental practice, from her general dentist, for an endodontic treatment of tooth 36 which was diagnosed with pulp necrosis. Endodontic procedure was completed after several sessions, where in between those the pus-productive tooth was intraradicularly irrigated, cleaned temporary and sealed with temporary filling (Coltosol).

After endodontic treatment was completed, she was referred for prosthetic restoration of tooth 36 and conservative restoration of teeth 16, 26 and 46. The previously mentioned molars were subjected to cold testing and reported vital without pulp complications and hypersensitivity. After endodontic treatment was completed, she was referred for further restoration of molars. Specifically, complete coverage with crowns in all 4 of them, due to extensive loss of hard tissues, multiple resin restorations and caries in several surfaces of those teeth.

DISCUSSION

The exact causative mechanism of molar-incisor hypomineralization (MIH) remains uncertain, but the localized and asymmetrical nature of the lesions suggests a systemic origin. Disruption in the amelogenesis process likely occurs during the early maturation stage or possibly earlier in the late secretory phase. MIH is generally considered a multifactorial condition, with systemic factors such as acute or chronic illnesses or exposure to environmental pollutants during the last trimester of pregnancy and the first three years of life being implicated as potential causes or contributing factors [13].

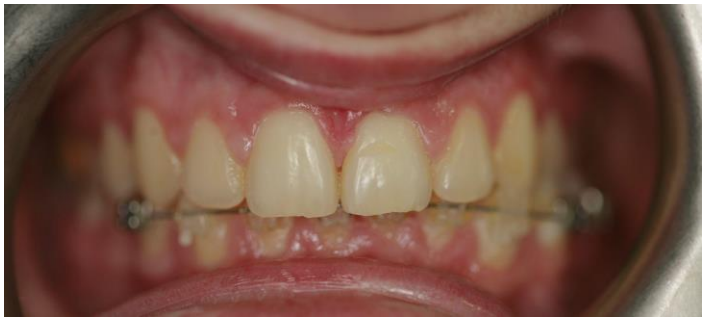

Figure 1: Clinical photograph of tooth 16 (upper right first molar) showing yellow-brown demarcated opacities and post-eruptive enamel breakdown, consistent with EAPD MIH code 2a

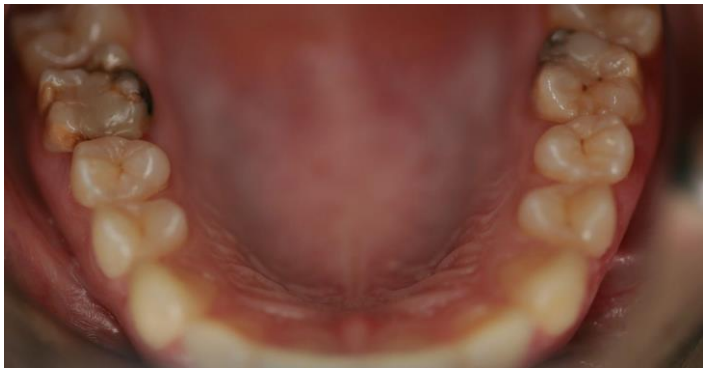

Figure 2: Clinical view of tooth 26 (upper left first molar) with similar enamel defects to tooth 16, classified as MIH code 2a, prior to conservative restorative treatment

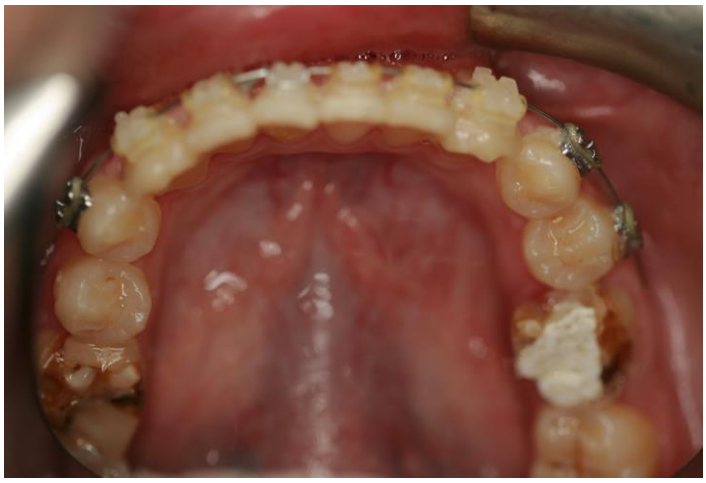

Figure 3: Intraoral image of tooth 36 (lower left first molar), demonstrating atypical restoration due to previous pulp necrosis and endodontic treatment. Classified as MIH code 3

The number of teeth affected appears to correlate with the timing of the systemic disturbance, with prenatal, perinatal, and postnatal issues associated with an increasing number of affected teeth. Various potential causes have been proposed in the literature, including respiratory tract infections, perinatal complications, exposure to dioxins, oxygen deprivation, low birth weight, calcium and phosphate metabolic disorders, frequent childhood illnesses, antibiotic use, and prolonged breastfeeding. Furthermore, some studies suggest a possible genetic component in the etiology of MIH, indicating that genetic variations may interact with systemic factors to contribute to its development [13].

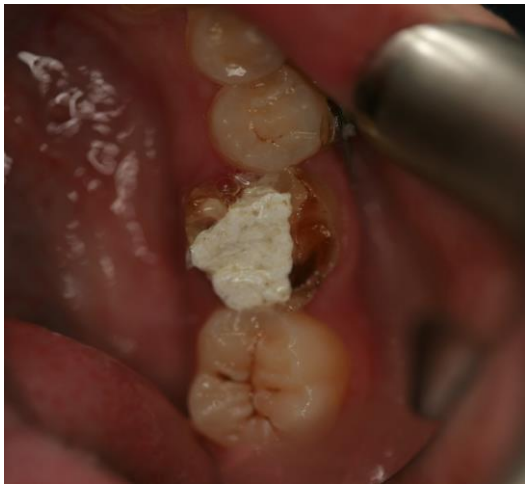

Figure 4: Tooth 46 (lower right first molar) showing multiple resin restorations and enamel loss across occlusal and proximal surfaces, consistent with MIH code 3

Erythrocytes depend exclusively on glucose for energy and utilize the pentose phosphate pathway for metabolism. The enzyme glucose-6-phosphate dehydrogenase (G6PD) plays a critical role as the rate-limiting enzyme in this pathway, catalyzing the conversion of glucose-6-phosphate into 6-phosphoglucono-δ-lactone. This reaction generates NADPH, which is essential for maintaining reduced glutathione levels. Reduced glutathione is vital for neutralizing hydrogen peroxide and other reactive oxygen species produced in the cell during oxidative stress. In individuals with G6PD deficiency, oxidative stress can lead to dangerously elevated levels of hydrogen peroxide, causing protein damage and ultimately cell death [14]. Ameloblasts, the cells responsible for enamel formation, are highly sensitive to oxidative stress and disruptions in their metabolic environment. These cells are particularly vulnerable during the secretion and maturation stages of amelogenesis, where an optimal balance of oxygen and metabolic byproducts is required [15].

G6PD is a critical enzyme in the pentose phosphate pathway, which generates NADPH. NADPH is crucial for maintaining reduced glutathione levels, a key antioxidant that neutralizes reactive oxygen species (ROS) such as hydrogen peroxide. In G6PD-deficient individuals, reduced glutathione levels may be insufficient, leading to increased oxidative damage during periods of stress [16]. The hypothesis that glucose-6-phosphate dehydrogenase (G6PD) deficiency may be linked to molar-incisor hypomineralization (MIH) is plausible. G6PD deficiency leads to increased oxidative stress due to reduced NADPH production, which is essential for maintaining cellular redox balance. Elevated oxidative stress can disrupt ameloblast function during enamel formation, potentially resulting in hypomineralized enamel. Research has shown that G6PD-deficient mice experience heightened oxidative stress, as evidenced by decreased NADPH and glutathione levels, along with increased markers of lipid peroxidation [16]. These findings suggest that the oxidative imbalance caused by G6PD deficiency could adversely affect ameloblasts, thereby contributing to enamel hypomineralization. Future investigation of the connection between G6PD and MIH would be enlightening.

Early identification of teeth affected by molar-incisor hypomineralization (MIH) is crucial for effective management. Since these teeth are at a higher risk of developing caries, early preventive measures are essential. Hypersensitivity associated with MIH may lead children to avoid oral hygiene practices. Therefore, it is important to provide enhanced oral hygiene instruction and dietary advice, which should be regularly reinforced for both children and their caregivers. The application of topical fluoride varnish is also recommended, primarily to prevent caries in permanent teeth [17].

The treatment of MIH is often challenging due to the variability of clinical manifestations and the potential sensitivity of the affected teeth. The primary aim of treatment is to reduce dental sensitivity, protect compromised enamel, and enhance aesthetics. In mild cases, desensitizing agents and fluoride can be used to alleviate sensitivity and prevent caries. For moderate cases, adhesive fillings and sealants might be applied to shield the enamel and provide a functional chewing surface. In instances where MIH is severe, restorative techniques such as dental crowns may be necessary. The therapeutic approach should be individualized based on the severity of MIH, patient needs, and available treatment options. Prevention and early diagnosis are paramount to ensuring effective treatment and minimizing complications associated with MIH [18].

Despite the well-known advantages of glass-ionomer-based restorative materials (GIC) such as fluoride release and chemical bonding, Resin composite (RC) is still the material of choice for one-surface restorations with MIH affected molars. GIC-based restorations can be used as dentin replacement or as an interim restoration until RC can be placed [19]. It must be considered that MIH enamel may have compromised bonding for sealants and composites.

Adhesive restorations cavity preparations should extend into sound tooth hard tissue for better adhesion. Amalgam restorations show high failure rates in atypically shaped molar MIH preparations. The need for retentive cavity preparations might aggravate existing tooth substance defects. For mild cases of MIH affecting incisors, conservative approaches may be employed. These include etching, sealing affected areas, and bleaching to improve aesthetics. For more severe cases, composite veneers or micro-abrasion may be considered to enhance aesthetics and restore affected areas [20].

As for severe cases of MIH in molars, full coverage crowns may be necessary for proper maintenance and function. Additional local anesthetic procedures may be required to manage hypersensitivity during restorative treatments. Tooth extractions of the first permanent molars may be considered in severe MIH cases when more than one molar is affected. This should take into account the patient's dental age (preferably 8–9 years old), the status of the patient’s occlusion, and the eruption of the second permanent molars. Frequent recalls should be established for these patients to monitor the high rate of restoration failure and to avoid secondary caries [20].

In patients showing loss of tooth structure, more extensive restorative treatment may be necessary. The use of bonding systems combined with composite resin on occlusal surfaces may be a good option in these cases. However, due to the poor adhesion of restorative material to hypomineralized tissue, glass ionomer cement should be the material of choice. Glass ionomer cements can also be placed on the occlusal surfaces of hypomineralized molars not completely erupted [21].

More severe cases require more invasive treatments. Semi-permanent restorations using stainless steel crowns or adhesive-retained metal castings are options to be considered. Extraction of affected molars, combined with orthodontic treatment, is another option, especially when a poor long-term prognosis is expected. Preformed metal crowns (PMC) are an inexpensive option with reported high success rates. They have the additional advantage of maintaining the structural integrity of the tooth whilst alleviating symptoms of hypersensitivity, maintaining the occlusal contact and can be placed in one visit making them ideal for use in teeth where multiple surfaces are involved [21].

CONCLUSION

Molar-incisor hypomineralization (MIH) is a complex dental condition characterized by qualitative enamel defects with systemic origins, leading to increased vulnerability to caries, tooth sensitivity, and significant restorative challenges. This case report highlights the

importance of early diagnosis, preventive care, and appropriate treatment strategies for managing MIH. Comprehensive management should involve a combination of preventive measures, such as enhanced oral hygiene instruction, dietary counseling, and the application of topical fluoride, along with restorative interventions tailored to the severity of the condition.

For mild cases, restorative approaches using glass ionomer cements and resin composites can preserve tooth structure and reduce hypersensitivity. However, in more severe cases, preformed metal crowns or extraction combined with orthodontic treatment may be required to ensure long-term oral health and functionality. Multidisciplinary collaboration between dentists, orthodontists, and other healthcare professionals is essential for the effective management of MIH, as illustrated by this patient's care pathway.

Further research is needed to better understand the multifactorial etiology and genetic contributions to MIH, enabling more targeted prevention and treatment strategies. By advancing knowledge and refining clinical practices, the prognosis for patients with MIH can be significantly improved, reducing the associated oral health burden.

Conflicts of Interest

The author reports no conflicts of interest.

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