MOLAR INCISOR HYPOMINERALIZATION IN MONOZYGOTIC TWINS: A CASE REPORT

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ABSTRACT **DOI: 10.25241/stomaeduj.2017.4(3).art.7**

Aim: The aim of this case report is to discuss the features of molar incisor hypomineralization (MIH), a developmental enamel defect, which was noted in a set of monozygotic twin males.
Summary: MIH is the hypomineralization of one to four first permanent molars (FPMs) frequently associated with affected incisors and varies in clinical severity. Early diagnosis and management of the condition is paramount and will avoid the premature loss of the molars.
Key learning points: The clinical characteristics, as well as the short and long term management of this condition are discussed.
Keywords: molar incisor hypomineralization (MIH), prevalence, diagnosis, management.

1. Introduction

Molar incisor hypomineralization (MIH) is a developmental qualitative enamel defect that was first defined by Weerheijm et al. in 2001[1] as "hypomineralization of systemic origin of 1-4 First Permanent Molars (FPMs), frequently associated with affected incisors".

Clinically, the hypomineralized areas appear as well demarcated opacities affecting the FPMs and incisors. These demarcated areas are abnormalities in the translucency of the enamel and may vary in color from white to yellow to brown. They are often associated with post eruptive enamel breakdown in the molars due to the teeth being subjected to masticatory forces and are also more severely involved than the incisors.[2]

The exact etiology of the condition remains unknown, however many factors that affect the stages of ameloblast formation during the prenatal, perinatal and postnatal periods were found to be possibly linked with MIH. Some of these potential risk factors include urinary tract infection during pregnancy, Caesarian section, premature delivery, exposure to environmental toxins, such as dioxin, childhood illnesses, asthma, pneumonia, high fever and antibiotic treatment especially during the first year of life. It is strongly believed that these factors act synergistically since none of them has a definite causative relation to the condition.[3,4,5] The prevalence of MIH has been studied extensively and it varies greatly from 2.4% in Germany and Bulgaria to 40.2% in Brazil.[6] The condition shows no gender predilection and the maxillary teeth, most specifically the molars, are most commonly affected.[7] The FPMs may not be affected to the same degree in an individual and some molars could be relatively unaffected.[8]

Hypomineralized teeth were initially classified into three groups, based on the severity of the enamel defect: mild (white, yellow or brown discoloration of the enamel), moderate (enamel loss only) and severe (loss of enamel in combination with affected dentin).[9] Due to some similarities in the clinical appearance of the moderate and severe cases, Lygidakis et al.[8] suggested the use of only two categories: mild and moderate-severe. The mild cases include demarcated enamel opacities with intact surfaces, occasional sensitivity and mild esthetic concerns. In moderate-severe cases, there is enamel surface breakdown, persistent or spontaneous hypersensitivity with major esthetic concerns.[8]

The aim of this case report is to discuss the features and management of MIH in a set of healthy monozygotic twin males.

2. Case Report

Two healthy monozygotic twin males, MB and SB, 9.6 years of age, presented with their father to the department of pediatric dentistry for a routine checkup with no major complaints.

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2.1. Medical History: The patients were healthy and were born at 38 weeks via an uncomplicated Caesarean section delivery. The mother developed food intolerance to dairy, wheat and fructose during the pregnancy and also took antibiotics during that time. During the first year of life, the twins also developed allergies to lactose and fructose and were given antibiotics for repeated otitis media infections.

2.2. Dental History: A detailed history, extraoral examination, intra oral examination and radiographic evaluation were conducted.

Patient MB presented areas of white and yellowish brown discoloration in the surface enamel on teeth 21 and 46 (FDI Notation System), respectively. The defective enamel appeared to be of normal thickness and had a distinct boundary demarcating it from the unaffected enamel. The other FPMs and maxillary incisors seemed unaffected.

A unilateral, left posterior crossbite was also observed involving teeth 64-26 and 74-36 (Figs. 1-5).

Patient SB presented a similar white opacity on the labial surface of tooth 21 in addition to yellowish brown discoloration of the surface enamel in teeth 16, 26, 36 and 46. SB also had a unilateral, right posterior crossbite involving teeth 16-53 and 46-83 (Figs. 6-10).

In both cases the MIH-affected teeth were not carious and there was no post eruptive breakdown involved. The patients initially reported no pain or sensitivity related to the MIH-affected teeth.

Both MB and SB had good oral hygiene and reported brushing twice daily using fluoridated toothpaste (1450ppm F).

A panoramic and bitewing radiographs were taken at the initial visit for MB and SB and revealed no signs of pathology or caries.

A comprehensive dental treatment plan was formulated and discussed in detail with the parent and informed consent was obtained. The treatment plan was similar for both patients.

The short term dental plan included dental prophylaxis and fluoride varnish (Duraphat® 22,600ppm F, Colgate Oral Care, Colgate-Palmolive (UK) Limited) application on all teeth.

Fissure sealants (Embrace®, PULPDENT Corporation, USA) were placed on all the FPMs as an initial measure in order to prevent caries formation on the areas affected by MIH.[10]

The patients and parents were educated about MIH and instructed to continue brushing two times daily with fluoridated toothpaste and additional brushing at night with MI plus (900 ppm F, GC) toothpaste in order to prevent sensitivity of the MIH-affected teeth.[11] Daily night flossing was also recommended. Dietary advice was given to both the parent and the patients. Three month recall visits were recommended to assess the oral hygiene and any enamel breakdown or sensitivity of the MIH affected teeth.

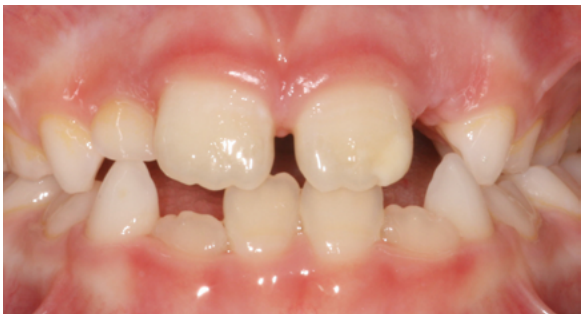

Figure 1. Intraoral front view, MB.

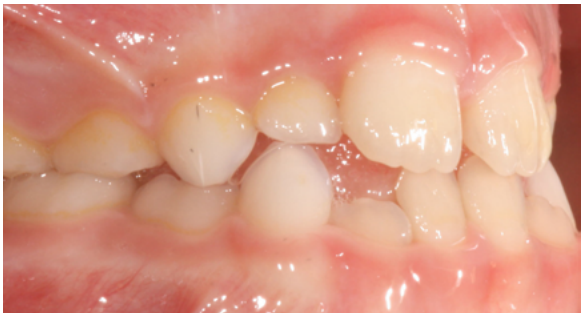

Figure 2. Right buccal view, MB.

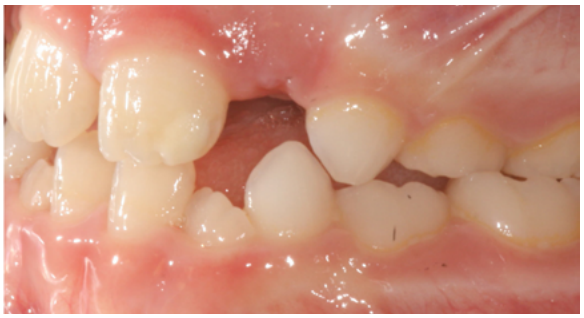

Figure 3. Left buccal view, MB.

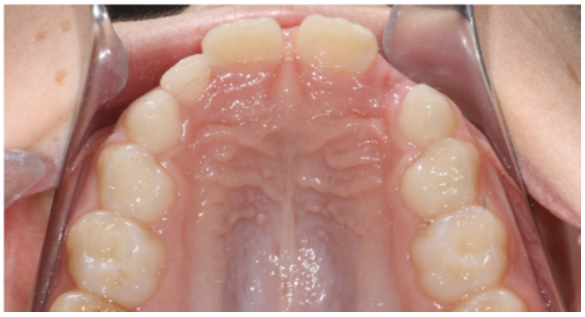

Figure 4. Upper occlusal, MB.

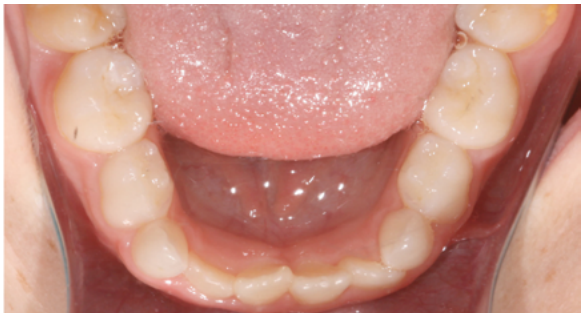

Figure 5. Lower occlusal, MB.

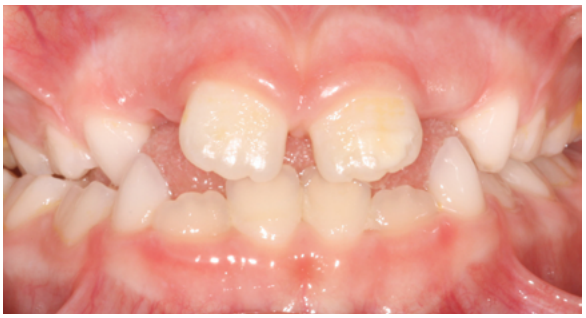

Figure 6. Intraoral front view, SB.

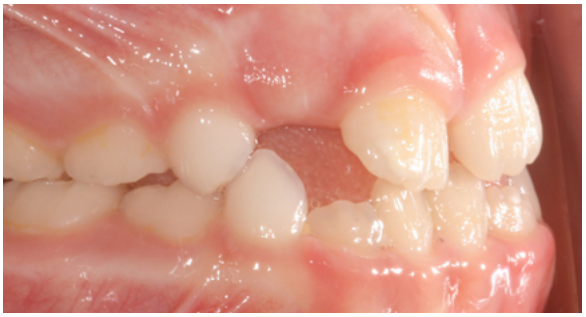

Figure 7. Right buccal view, SB.

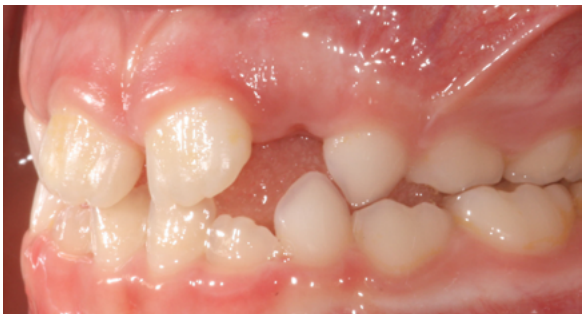

Figure 8. Left buccal view, SB.

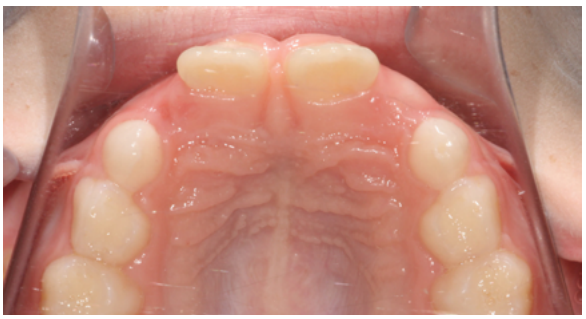

Figure 9. Upper occlusal, SB.

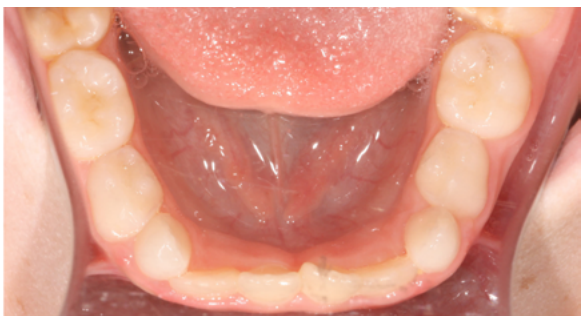

Figure10. Lower occlusal, SB.

An orthodontic consultation was done and recommendations were made to correct the posterior crossbite using a quadhelix expander.[12] The appliance was activated for 8 months which accomplished the correction of the crossbite relation in both MB and SB.

On the subsequent 6 month recall visit, the patients reported pain from one of the MIH-affected teeth, MB from tooth16 and SB from tooth 26. Although all the FPMs had retained the fissure sealants, the teeth causing sensitivity showed further enamel breakdown. Due to that sensitivity and in order to avoid further breakdown of the enamel,[13] prefabricated stainless steel crowns were the treatment of choice.

The patients continued to remain caries free and good oral hygiene was continuously maintained at the subsequent recall visits (Figs. 11-14).

3. Discussion

Molar incisor Hypomineralization is a qualitative developmental defect of the enamel, that affects at least one FPM and is often associated with affected incisors.[4]

There are several etiological factors suggested in the literature related to the development of MIH, but none of them is a clear definitive cause. These factors are complications that might occur during the prenatal, perinatal or postnatal period and disrupt the enamel formation during amelogenesis. They include low birth weight, premature delivery, malnutrition during the last trimester of pregnancy and maternal urinary tract infections. Prolonged childhood illnesses, especially during the first year of life, are also implicated as causative agents of MIH and include otitis media, asthma, pneumonia and prolonged high fever due to infections. Exposure to environmental toxins (dioxins) and diseases like mumps, measles and chicken pox have also been cited as possible causes.[4,5]

Since the early mineralization phase of the FPMs occurs close to birth until the first year of life, the teeth are susceptible to the various etiological factors causing MIH during this critical stage of tooth development.[8]

Lygidakis et al.[5] found that MIH was more common in a study group with perinatal complications of Caesarean section, premature birth, prolonged delivery and twinning.

This case report presented monozygotic twins that were born on term, via Caesarean section delivery with a normal birth weight. The mother did report taking antibiotics on one instance during the pregnancy for an infection that she could not recall. The twins also took antibiotics on two occasions for otitis media during the first year of life.

The diagnosis of MIH was confirmed with the characteristic clinical appearance of the affected molars and incisors in combination with the medical history, which could have been a contributing factor.

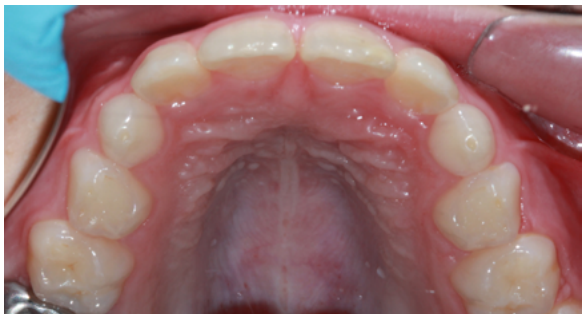

Figure 11. Postop upper occlusal, MB.

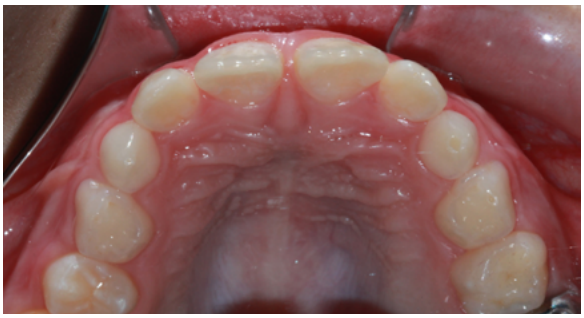

Figure 13. Postop lower occlusal, MB.

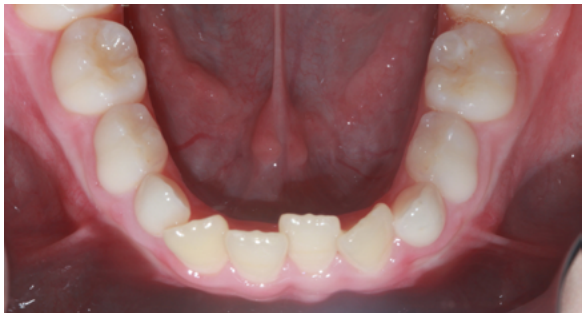

Figure 12. Postop upper occlusal, SB.

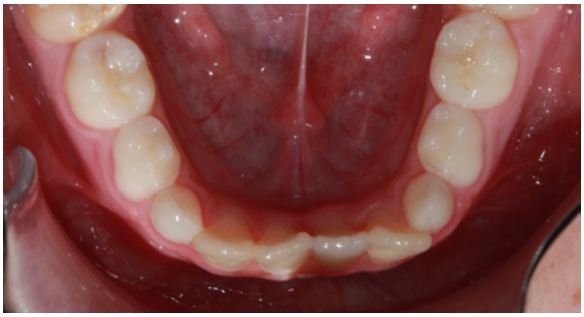

Figure 14. Postop lower occlusal, SB.

This follows the recommendations made by Weerheijm in 2003,[14] that the diagnosis of MIH is confirmed by clinical examination on clean and wet teeth ideally after the age of 8 years when all permanent incisors and first molars will mostly be erupted and that at least one FPM has to be affected.[8,14] Weerheijm et al.[15] also proposed that the clinical appearance of the 4 FPMs and 8 incisors should be recorded for the following features which will aid in the correct diagnosis of the condition: presence or absence of demarcated opacities; post eruptive enamel breakdown; atypical restorations; extraction of a FPM; failure of eruption of a FPM or an incisor.

Teeth affected by MIH, with or without enamel loss, are often associated with hypersensitivity to air and cold stimuli. This could be explained by the findings of Rodd et al.[16] that there is increased neural innervation in the pulp horn and subodontoblastic areas of the hypomineralized teeth. An increase in immune cells and vascularity, resembling an inflammatory response, was noted in hypomineralized teeth with enamel loss. The post eruptive enamel breakdown often leads to dentinal exposure to external oral stimuli, which may further contribute to hypersensitivity.[16] As a result, sometimes the affected teeth might not be adequately anesthetized due to peripheral sensitization, despite effective local anesthesia techniques. This may lead to poor patient cooperation and difficulty in treating these teeth. In addition, the sensitivity may lead to the avoidance of brushing in the area, which can hasten the post eruptive enamel breakdown.[3,8]

The management of MIH is thus very challenging and depends on various factors such as the extent and severity of the lesion, presence of sensitivity, post eruptive enamel breakdown, the patient's age and cooperation level and the child and parental expectations. Clinical approaches may vary accordingly, from simple preventive measures such as resin or glass ionomer sealants to more invasive approaches such as extraction in association with orthodontic management. A multidisciplinary approach is therefore mandatory.[3,8,13]

In a study conducted by Jalevik et al.[17] on Swedish children, it was found that by the age of 9, children with MIH-affected FPMs had undergone dental treatment nearly ten times more frequently than the controls undergone by healthy children, who were the controls, and the affected teeth had each been treated twice, on an average. This underscores the importance of maintaining good oral hygiene and other preventive measures which may help in the prevention of dental caries and post eruptive breakdown. Brushing with a fluoridated toothpaste (1000-1500ppm F) twice a day and good dietary habits must be reinforced. Remineralizing agents such as fluoride varnish (Duraphat, 22600ppm) and Casein Phosphopepetide Amorphous Calcium Phosphate (CPP-ACP) have been shown to reduce the sensitivity of the enamel.[11]

Preventive measures such as fissure sealants are recommended on FPMs with mild MIH with no evident enamel breakdown or sensitivity. These teeth do however require regular follow up to monitor the retention of the sealants.[3] A study by Lygidakis[10] has shown that application of a 5th generation bonding agent prior to sealant placement improves its retention.

Glass ionomer sealants are recommended for partially erupted molars or when adequate

control of moisture is not possible, but are shown to have very poor retention rates.[8,13]

In mild MIH-affected molars with small carious lesions involving 1 to 2 surfaces, composite resins are the restorations of choice and in carious teeth involving 2 or more surfaces with increased or spontaneous sensitivity, full coronal coverage with prefabricated stainless steel crowns is the preferred treatment option.[3,8,13]

Composite restorations bonded with a self etching primer adhesive are shown to be successful in mildly affected MIH lesions.[12]

In some cases of teeth with severe MIH where the teeth are non restorable and have a poor prognosis, extraction may be the only option. In such cases, an orthodontic evaluation is necessary to determine the timing of the extraction as well as future orthodontic intervention.[17]

MIH-affected incisors pose an esthetic concern for the child and their parents and may respond to microabrasion and bleaching based on the color and thickness of the opacity. The yellowish brown lesions are full thickness and respond better to bleaching with carbamide peroxide.[8]

In the present case, the incisors were mildly affected and posed no esthetic concern to either the children or the parents and were closely monitored at every recall visit for caries or enamel breakdown. The MIH-affected molars, in both MB and SB, were initially asymptomatic and treated with pit and fissure sealants to prevent decay formation. The patients were instructed to continue to maintain good oral hygiene practices with the use of fluoridated toothpaste (1450ppm F) and were also prescribed MI paste (900ppm F, GC) to prevent sensitivity.

When teeth 16 and 46, in cases MB and SB respectively, developed sensitivity and showed signs of slight post eruptive breakdown, stainless steel crowns were recommended as the treatment of choice to avoid further sensitivity and protect the enamel from further post eruptive breakdown. The remaining teeth continued to remain asymptomatic with the sealants remaining intact.

The patients continued to remain caries free and all the affected teeth remained asymptomatic at subsequent recall visits.

4. Conclusion

The diagnosis of MIH is largely based on the clinical appearance of the teeth and is often supported by a history of systemic illnesses during the developmental stages of the ameloblasts. Early, accurate diagnosis and long term follow-up is essential in order to avoid sequelae such as post eruptive enamel breakdown, caries formation and sensitivity of these teeth. Management options vary and are based on the extent and severity of the affected enamel.

Author Contributions

Equal contribution to the paper.

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The authors declare no conflict of interest related to this study. There are no conflicts of interest and no financial interests to be disclosed.

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Questions

Which of the following statements best describes MIH?

❑a. Qualitative defect of dentin that affects the permanent molars and incisors;
❑b. Qualitative defect of enamel that affects the permanent molars and incisors;
❑c. Qualitative defect of enamel that affects the primary molars;
❑d. Quantitative defect of enamel that affects the permanent molars and incisors.

In which of the following periods an insult can result in MIH affected teeth?

❑a. Prenatal period;
❑b. Perinatal period;
❑c. Postnatal period;
❑d. All of the above.

What is the etiology of MIH?

❑a. Genetics;
❑b. High fever during the first year of life;
❑c. Premature birth;
❑d. Unknown etiology.

The management of MIH depends on:

❑a. Age of the patient;
❑b. Number of affected teeth;
❑c Severity and extent of the defect;
❑d. All of the above.