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Molar-incisor hypomineralization in relation to its etiological factors

A Project Submitted to the College of Dentistry, University of Baghdad, Department of Pedodontics and Prevention dentistry in partial fulfillment for the requirement to award the degree B.D.S

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Declaration

This is to certify that the organization and the preparation of this thesis had been made by graduate student **Mohammed Fadhel Hammad** under my supervision in the College of Dentistry, University of Baghdad in partial fulfillment of the requirement for the 5th grade.

Signature:
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The supervisor

Dedication

To…

My family who have always stood by my side and gave me the support that I needed to be the man that I am today.

Acknowledgment

Thanks **Allah** for the graces that i was given and giving the chance to be what I am today.

I would like to express grateful thanks to dean of College of Dentistry, University of Baghdad Prof. **Dr. Raghad A. Al-Hashimi.**

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Introduction

Tooth hypoplasia is a defect in quality and quantity of tooth structure due to developmental origin. Molar incisor hypomineralization (MIH) is a specific form of tooth hypoplasia. Defined MIH as a “hypomineralization of one to four permanent first molars frequently associated with affected incisors and is due to systemic origin. Enamel hypoplasia is a quantitative defect of the enamel, and enamel hypomineralization is a qualitative defect of the enamel identified visually as an alteration in the translucency of the enamel, with a clear border, variable in degree, and a white, yellow or brown colour. It has also been termed a demarcated opacity.

The first permanent molars with hypomineralization are often associated with affected permanent upper incisors and, more rarely, lower incisors. Therefore, the name Molar Incisor Hypomineralization (MIH) is currently used (William, et al. 2006, Alaluusua, et al. 2010).

Aim of study

1- To establish the difference between the normal tooth structure and the molar-incisor hypomineralization affected teeth.
2- To identify the different degrees of severity of molar-incisor hypomineralization.
3- to discuss the etiological factors related to (MIH) incidence.

1- Tooth structure

The tooth has three distinct layers; the enamel which covers the crown, the root cementum on the root surface and an inner layer of dentin mola in the crown and the root. The pulp, which contains arteries, veins and nerves, is in the inner part of the tooth. Any change of these structures is incisor likely to cause an alteration in the outward appearance of the tooth caused by changes of its light transmitting and reflecting properties (Jenssen and Tran, 2011).

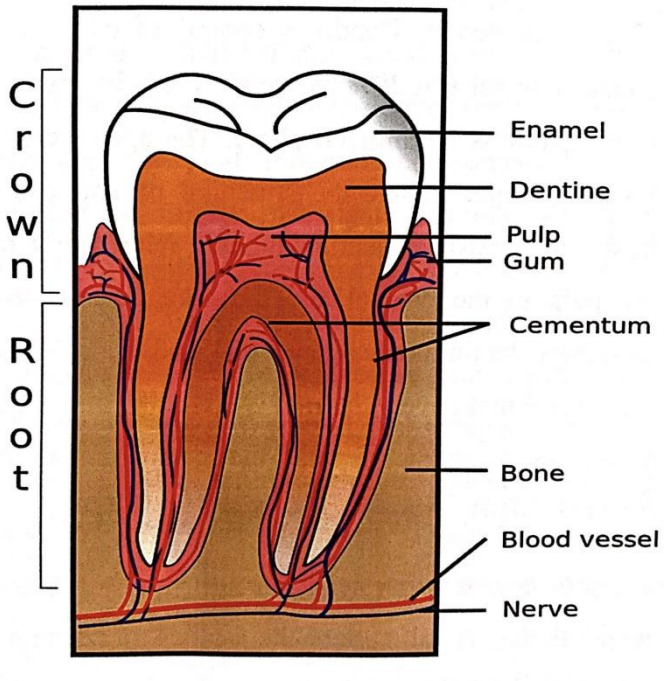


Figure (1). Tooth structure (Jenssen and Tran, 2011).

The structure is layered, with an outer layer consisting of enamel supported by the underlying dentin. Enamel and cementum meet in the cementoenamel junction (CEJ) on the root surface. The anatomic crown is above the CEJ and enamel covers this part of the tooth. The cementum covers the anatomic part of the root and is found below the CEJ. Most of the root composes of dentin. The normal dental pulp consists of pulp chamber filled with soft connective tissue (Fejerskov and Kidd, 2008).

Dental enamel is a highly mineralized acellular tissue in which microscopic calcium phosphate crystals comprise of some 99% of the dry weight. The crystals resemble the mineral hydroxyapatite, Cal0 (PO4)6(OH) 2 in a way that the calcium, phosphate and hydroxyl ions are arranged in a repeating pattern in the crystal lattice structure. Inclusions of carbonate, sodium, fluoride and other ions make it an impure form of the mineral. The space between the crystals is occupied by water (11% by volume) and organic material (2% by volume). The dentinal cells, the odontoblasts, produce dentin. Dentin is comprised of 70% by weight (50% by volume) mineral and 20% by weight (50% by volume) organic matrix. Hydroxyapatite is the mineral phase.

The apatite crystallites are held together by collagen. Thus, the structural backbone of dentin is collagen. The dentin consists of dentinal tubules, microscopic canals, that run from the pulp to the enamel and the odontoblasts have cellular extensions into these dentin tubules (Fejerskov and Kidd, 2008).

The cementum that covers the root is less mineralized than enamel and dentin and as a result of this, the cementum is also softer (Bergenholtz, et al. 2010).

A variety of colors can typically be seen in a tooth and from the gingival margin to the incisal edge of the tooth a gradation of the color occurs (Tredwin, et al, 2005).

Normal enamel is colorless and translucent, and the color of the dentin is mainly responsible for the color of the tooth. The dentin influences more on the tooth color where it consists of thick layers and where the enamel layer is thin (Fejerskov and Kidd, 2008).

The composition of tooth structures will also affect the outward appearance of a tooth. If structures like the enamel, the dentin or the pulp changes, the light transmitting and light reflecting properties of a tooth are likely to be altered (Tredwin, et al. 2005).

2-Tooth Development

Ectodermal organs such as teeth, hair, mammary gland and salivary gland are all developed from epithelial and mesenchymal tissue. Although diverse in their mature forms, these ectodermal organs share the initial developing pattern (Li, 2013). Specifically, tooth development requires sequential and reciprocal signals that are transmitted between ectodermal or endodermal-derived epithelium and the cranial neural crest-derived mesenchyme (Li, 2013).

Morphologically, tooth formation commences with a structure of dental epithelial thickening, the dental lamina, which subsequently proliferates and invaginates into underlying mesenchyme. At the same time, signals from thickened epithelium induce condensation of mesenchymal cells, which is recognized as a tooth germ. The condensed ename mesenchyme then guides further epithelial invagination and convolution to progress the enamel organ through the sequential bud, cap, and bell- shaped stages of tooth morphogenesis. During these stages, distinct anatomical and functional parts of the tooth form, and the basic shape of tooth crown is established. Eventually, the mesenchyme-derived odontoblasts and epithelium-derived ameloblasts differentiate at the epithelial-mesenchymal interface to form dentin and enamel respectively (Figure 2) (Nanci , 2008 and Bei , 2009).

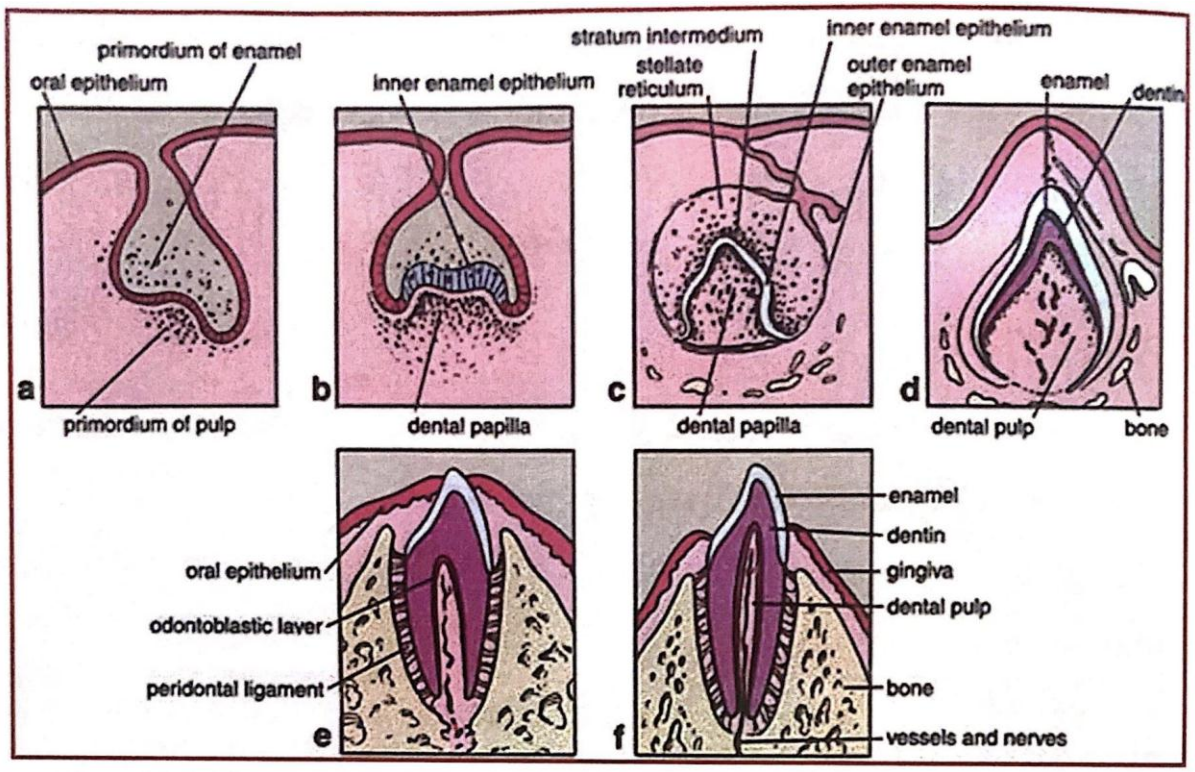


Figure (2). Tooth development. (Nanci , 2008 and Bei , 2009).

Studies identified multiple gene networks that control the formation of teeth (Yaseen, 2005).

Many genes and signaling pathways have been demonstrated to regulate morphological patterning and cell differentiation at specific stages of tooth development. Members of the transforming growth factor B (TGFB) and fibroblast growth factor (FGF), signaling pathways constitute the key pathways that mediate epithelial- mesenchymal interactions during tooth development (Bei, 2009).

Animal studies have shown that aberration of any of these pathways leads to developmental arrest of teeth at early stages of tooth formation. Furthermore, transcription factors mediating these signaling networks are critical for early tooth development as well. In humans, environmental or genetic disturbances during the early process of tooth development also lead to the developmental arrest of tooth germs and cause tooth agenesis (Parkin, et al. 2009).

3- Overview of Enamel Development

Enamel development (amelogenesis) can be broken down into four defined stages: presecretory, secretory, transition and maturation. The stages are defined by the morphology and function of the ameloblasts (Figure 3).

The ameloblasts are a single cell layer that covers the developing enamel and is responsible for enamel composition. Ameloblasts are part of the enamel organ that is composed of an outer epithelial layer, the stellate reticulum, the stratum intermedium, and the inner enamel epithelium (ameloblast layer). The basal (proximal) end of the preameloblast is attached by desmosomes to the stratumintermedium, and the apical (distal) end is attached by hemidesmosomes to a basement membrane (basal lamina) located at the future site of the DEJ (Bartlett, 2013).

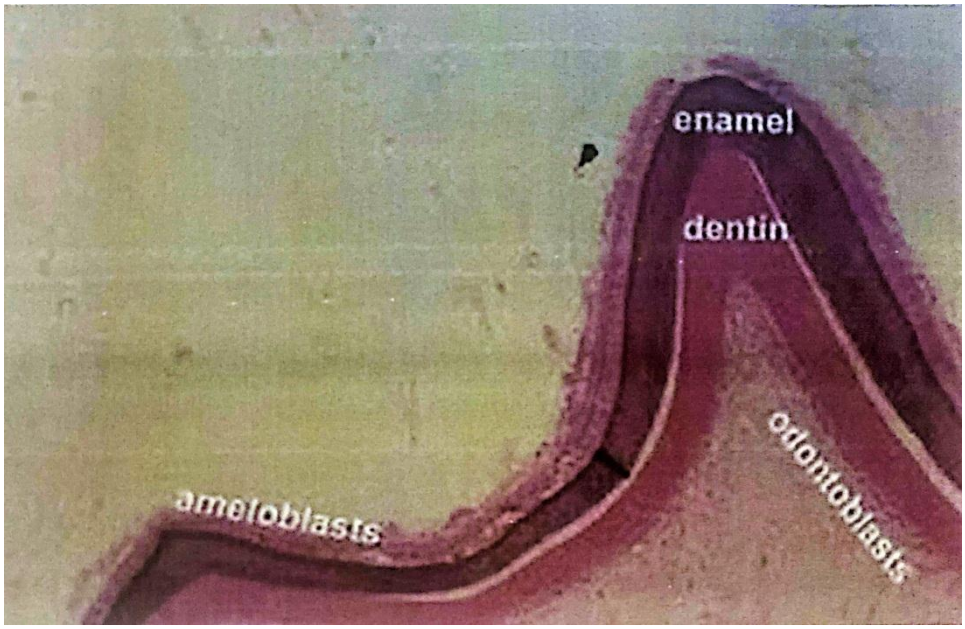


Figure (3). Enamel development. (Bartlett, 2013).

4- Developmental defects of enamel

4.1 Diffuse opacity

Enamel opacity (defect) can be defined as a qualitative defect in enamel and as a visible abnormality in the translucency of enamel. Qualitative defects in enamel imply a disturbance in enamel matrix formation and/ or in its mineralization or maturation during amelogenesis. It is widely accepted that enamel defects may arise from a large variety of causes. More than 100 causes of enamel defects have been described. These may be broadly divided into localized and generalized. (Khan H, 2005).

Generalized causes include predominantly environmental and genetic. Ingestion of inappropriate levels of fluoride during the developmental period of the teeth represents one environmental cause of enamel defects. This type of opacity (defect) has been extensively investigated, and has been described in relation to excessive ingestion of fluoride in water, food and drinks, fluoride supplements and fluoride toothpaste. Some authors suggest that opacities related to excessive fluoride ingestion (fluorosis) can be differentiated on the basis of clinical appearance alone. However, differential diagnosis of enamel defects has proved difficult even when comprehensive medical and dental histories are available. This is particularly true in the case of mild enamel defects in low fluoride areas or of defects arising from more than a single cause. One reason for the difficulty in determining etiology has been the lack of a well-defined and universally acceptable index of measurement. A wide range of indices has been used in the past. These can be divided into specific fluorosis indices and descriptive indices (Khan, 2005).

4.2 Hypoplasia

Enamel hypoplasia (figure 4) is a commonly occurring dental defect that is widely considered to be a good indicator of physiological stress occurring during dental crown development. Dental enamel hypoplasia can be simply defined as a deficiency in the thickness of the dental enamel. Dental enamel hypoplasia have been used in many archaeological, anthropological and zoological studies to reveal aspects of dietary deficiencies and general health in past human and animal populations (Oyamada , et al. 2008).

The clinical study concluded that the defect in the teeth will be a big problems for most of patients as esthetic, functional and psychological problems (Ibraheem, 2013).

While it has been suggested that microscopic analysis of tooth section is the only sure way to accurately identify all enamel hypoplasia, this obviously not practical on a large scale Therefore specific methodologies for the macroscopic recording of enamel hypoplasia have been developed (Kierdorf, et al. 2006).

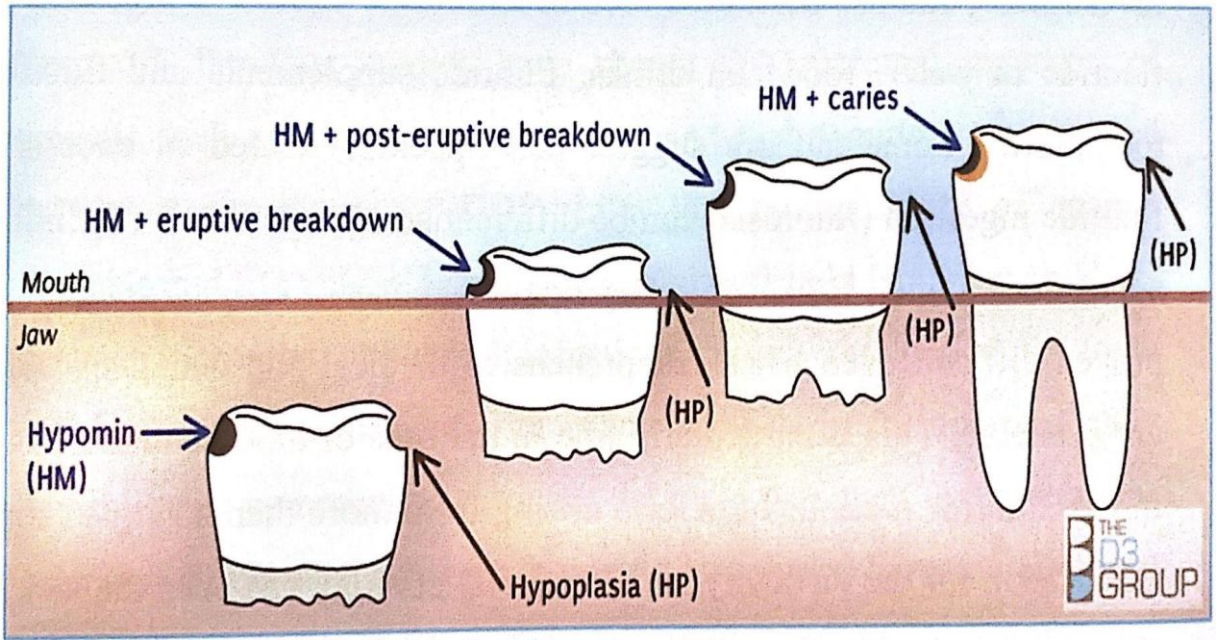


Figure (4). Enamel hypoplasia. (kierdorf, et al. 2006).

5- Molar-incisor hypomineralization (MIH)

Molar–incisor hypomineralization (MIH) is a developmental defect of the human dentition that primarily affects the enamel of the first permanent molars and can involve the incisors. Typically, the second permanent molars and premolars are not involved. This condition has been recognized since around 1970 and has been described using a variety of terms (e.g., cheese molars, idiopathic hypomineralization of enamel) (Weerheijm et al., 2001a).

Once the tooth begins to erupt and come into function, rapid enamel loss can make the crowns appear hypoplastic, but this is typically the result of enamel fracturing, wear, and dental caries. Discoloration of the involved areas is a result of the decreased mineral content and increased protein and water content that change the optical character of the hypomineralized enamel (Fagrell, 2011).

The enamel color changes range from white opaque lesions to a creamy yellow or brown. The more severe the level of hypomineralization, the more likely the tooth is to have early loss of enamel. Early enamel loss is often associated with the development and progression of dental caries that can lead to rapid deterioration of the clinical crown) and pulpal involvement if left untreated.

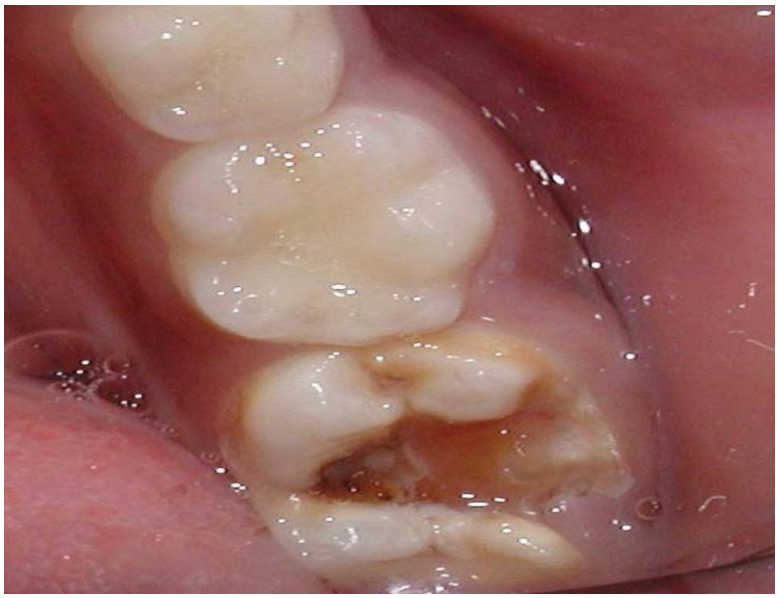


Figure (5). Molar brown discoloration in sever MIH (William et al., 2006).

The degree of hypersensitivity associated with these defects varies but can be quite pronounced and appears to be frequently associated with the severity of hypomineralization and enamel loss. Hypersensitivity and difficulty anesthetizing the affected molars can add to the challenge of treating individuals with MIH (William et al., 2006).

5.1 ETIOLOGY

Investigators have put forward a number of possible causes; the etiologies were divided into five groups: (1) Exposure to environmental contaminants, (2) pre/peri and neonatal problems, (3) exposure to fluoride, (4) common childhood illnesses, and (5) medically compromised children (Gotler M & Ratson T., 2010).

Willmott et al suggested asthma, pneumonia, upper respiratory tract infections, otitis media, antibiotics, dioxins in mother's milk, tonsillitis and tonsillectomy, and exanthamatous fevers of childhood (Willmott et al., 2008).

Crombie et al opined polychlorinated biphenyl/dioxin nutrition, birth and neonatal factors, and acute or chronic childhood illness/treatment, fluoride or breastfeeding. (Crombie et al., 2009).

Alaluusua in his review suggested high fever, hypoxia, hypocalcemia, exposure to antibiotics (amoxicillin), and dioxins as risk factors. Combined effect of several factors should also be taken into account (Alaluusua 2010).

Despite existing knowledge regarding etiological factors, adequate proof to validate the effect of etiological factors is still required. Experimental dose/response studies on molecular mechanism of ameloblasts are essential to deepen our knowledge of presently presumed factors. Also, prospective studies are needed to reveal new factors that might be involved (Willmott et al .,2008).
With regard to the timing of the defect formation, the review of the literature has shown systemic factors act around the time of birth or during the child's first 3 years (corresponds to formative stages of incisors and molars).

5.2 Prevalence

The prevalence of MIH ranges from about 3–40% in the population, making it relatively common and a condition that will challenge clinicians on a regular basis (Jalevik, 2010).

The first permanent molar is the permanent tooth most likely to develop dental caries, and hypomineralized molars are at increased risk for developing dental caries. Caries development can obscure the presence of enamel hypomineralization that likely contributes to the carious involvement in at least some first permanent molars. The first permanent molars begin to form in utero and typically start to mineralize just before or shortly after birth (first permanent molars of girls tend to form earlier than those of boys). The enamel of the permanent first molars and permanent incisors does not fully mineralize until the age of 3–5years. Having enamel hypomineralization in the primary dentition increases the odds that the individual will have MIH in the per- manent dentition (Elfrink et al., 2012).

5.3 Diagnosis of molar-incisor hypomineralization

Clinical evaluation for the presence of MIH ideally involves examining the four first permanent molars and eight permanent incisors and is often best accomplished in an 8-year-old child (Weerheijm et al., 2003).

The examination should be performed when the teeth are clean and moist. They are examined for the presence of demarcated changes in enamel color and translucency (opacities) and areas of enamel loss that most often occur in the affected molars. A severity scale has been developed to classify MIH as mild, moderate, or severe at the tooth level, meaning that one tooth may be mild, and another tooth in the same patient may be severe, and seeing this amount of variability is a common occurrence (Table 1) (Mathu-Muju & Wright, 2006).

Enamel color changes are caused by changes in the enamel composition (amount of mineral and protein) and structure. Enamel that is yellow brown

tends to have less mineral compared to white opacities and is more likely to succumb to enamel loss. These yellow-brown areas tend to lack the shiny reflective surfaces of normal enamel and have a more ground glass and slightly rough appearance indicative of a decreased mineral content. Clinically assessing these characteristics is helpful in determining prognosis for an individual tooth and the likelihood that it will break down over time as a result of enamel loss. These clinical attributes are also helpful in selecting appropriate treatment approaches and optimizing therapeutic success (Mathu-Muju & Wright, 2006).

Table (1): Severity score of teeth affected with MIH

	Mild	Moderate	severe
Crown appearance	Demarcated opacities in non-stress-bearing area of molar	Intact atypical restoration present	post-eruptive enamel break-down present
Enamel loss	Isolated opacities	occlusal/incisal third of teeth without initial post-eruptive enamel break-down	post-eruptive enamel break-down on erupting tooth that can be rapid
caries	No caries associated with affected enamel	Post-eruptive enamel break-down/caries limited to one or two surfaces without cuspal involvement	Often develop widespread caries associated with affected enamel
sensitivity	Normal dental sensitivity	Usually normal dental sensitivity	Usually history of dental sensitivity
esthetics	Usually not an issue	Parents often express concern	Parents typically concerned

5.4 Treatment approaches for molar–incisor hypomineralization

Treatment approaches for MIH will vary substantially depending on the level of severity of the defect. The treatment goals for molars are to prevent the tooth from developing dental caries, to help prevent or reduce enamel loss, to restore form and function when there is enamel loss, and to address esthetic issues (this issue is typically more important in the affected incisors and is discussed) (William et al., 2006; Lygidakis et al., 2010).

For some moderately and most severely affected molars, another goal is to manage the hypersensitivity associated with the hypomineralized and lost enamel. Normally, enamel has excellent insulating properties and protects the tooth from chemical and thermal stresses. When the mineral content is reduced and the protein and water content is increased or the enamel is lost, the tooth often becomes easily stimulated, and the level of sensitivity can be quite severe. Treatment approaches thus tend to be predicated on the severity of the MIH and the presence or absence of dental sensitivity (Table 2).

Table (2): Management of molar-incisor hypomineralization

	Mild	Moderate	severe
Molars	Desensitizing toothpaste fluoride varnish sealant	Fluoride varnish sealant resin restorations	Glass ionomer coverage interm resins Stainless steel crowns Extraction
Incisors	No treatment Resin perfusion	bleach/seal Resin perfusion Microabrasion Resin restoration	bleach/seal Resin perfusion Microabrasion Resin restoration Veneers

5.4.1 Mild degree of MIH

For milder levels of dental sensitivity, the use of toothpaste for sensitive teeth might be of some benefit, although there are no studies to verify their usefulness. Other treatments such as amorphous calcium phosphate – casein phosphopeptide have been suggested, but there is little evidence that they function with any greater effectiveness than fluoride toothpaste and are much more costly. The use of chlorhexidene rinses at the concentration sold in the United States (0.12%) is not effective for controlling caries and can cause significant staining and so are not recommended for caries management (Rethman et al., 2011).

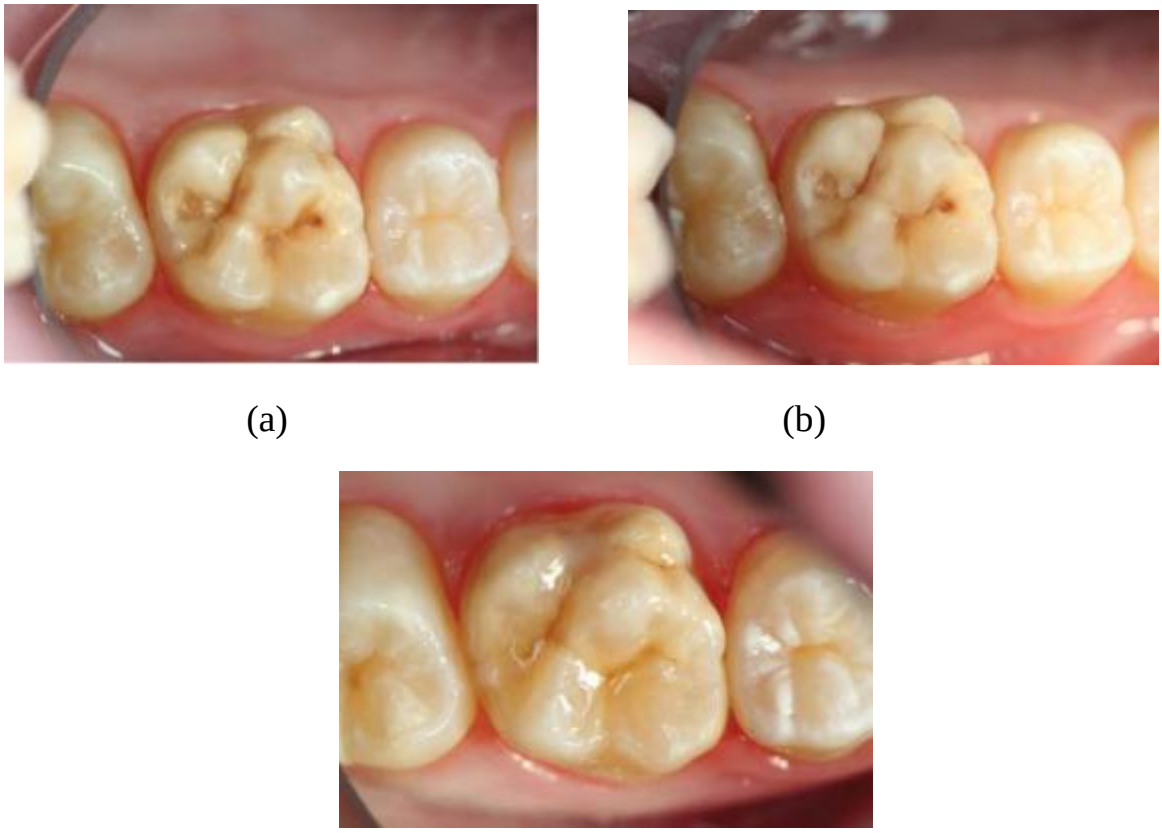
For mild MIH fissure sealants provide a valuable preventive measure. Sealant retention can be an issue in teeth with MIH because the enamel is inherently weakened and has an increased protein and water content. Studies on sealant retention in MIH-affected teeth have been limited in numbers of teeth studied but indicate that sealants can be successful; however, retention varies with the material and technique for placement. A number of approaches have been suggested to enhance the bonding to these defective enamel surfaces (Rethman et al., 2011).

5.4.2 Moderate degree of MIH

Teeth with moderate degrees of MIH will have more extensive areas of affected enamel, and there is a high likelihood that stress-bearing areas will eventually fracture with function over time. Thus, cuspal and marginal ridge enamel fractures are common in these teeth. If there is no enamel loss, then initial treatment should be placement of a resin or GI sealant that can help protect the enamel surface. If enamel fracturing occurs, then a filled resin or GI-type restoration can be placed to restore the normal tooth contours and help prevent caries from forming in uncleansable and exposed areas. Removal of the

severely affected enamel areas to allow for a minimal bulk of material required for fracture resistance and to allow the restoration to extend to minimally or non-affected enamel that is adjacent to the fractured area will improve restoration longevity. The technique for placement of resin restorations is the same as that outlined for resin sealant placement. Amalgam restorations also perform relatively well in the treatment of these teeth (Lygidakis et al., 2010).

If this non-adhesive approach is used, the restoration should not end on severely affected areas of enamel that will be stress bearing, as they will likely fracture away from the amalgam (Lygidakis et al., 2010).



(a) (b)

(c)

Figure 6. This maxillary first permanent molar has moderate MIH with some enamel loss already occurring in both the mesial and distal occlusal areas (6 A). The tooth was cleaned and treated with 5% NaOCl, which shows the improved color change that can occur with this treatment (6 B). The tooth was etched, bonding agent placed and polymerized, and then sealed with a white opaque sealant in the lingual area for improved esthetics and then coated entirely with a highly penetrating clear resin sealant (6 C). (Lygidakis et al., 2010).

5.4.3 Severe degree of MIH

Teeth with severe MIH are the most challenging to treat. The molars may be losing enamel and becoming cariously involved as they first begin to emerge into the oral cavity. Extreme sensitivity can accompany this clinical presentation. These teeth can often be treated using GI materials that can be placed with no tooth preparation or need for anesthetic. The tooth can be blotted dry with cotton, and either a chemical or light-cured or resin-modified GI placed (Figure 7).

If the tooth is early in its course of eruption, then additional material will likely need to be placed at regular intervals until a more definitive restorative approach can be considered. Placement of these materials provides an effective barrier to thermal and chemical stimulation and can provide immediate relief from sensitivity (Lygidakis et al., 2010).

Although these restorations can serve as excellent protective treatments, they typically are not definitive, and when the tooth is fully erupted, additional or other treatment can be considered. In some cases, these types of treatments can last for years, allowing additional dentin formation, root apexification, and increased maturity of the patient.

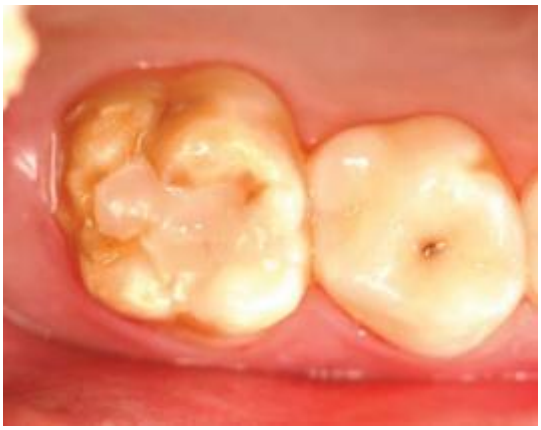


Figure 7. This maxillary first permanent molar caused extreme sensitivity with temperature changes and was treated using a protective resin-modified glass ionomer with no tooth preparation other than pumice prophylaxis and blotting the tooth dry. The margins show some deterioration at this 3-year follow-up, but the restoration remains intact with no develop- ment of caries or problems with sensitivity.

If early treatment is not provided in teeth severely affected with MIH, then extensive destruction and caries development can result in pulp necrosis and teeth requiring more extensive restorations. Many of the severely affected teeth will ultimately need to be restored with crowns. Clinical studies show that stainless steel crowns (SSCs) and cast crowns provide outstanding results with very few failures (Lygidakis et al., 2010).

6. Determinants and associated factors

Tooth development, although genetically controlled, is reported to be sensitive to disturbances from the environment. Because enamel is not remodeled like bone, disturbances acquired during its development leave a permanent record in the tooth. Dental development starts with the formation of the dental lamina from the ectodermal epithelium. Tooth development follows the bud, cap and bell stages, generating the shape of the tooth. The cells from the dental lamina differentiate into, among others, ameloblasts and dentinoblasts. Dentin and enamel formation occur simultaneously along a line that will develop into the dentino-enamel junction. At a certain point, the secretory ameloblasts undergo a transition (transitional stage), and the maturation of the enamel will start.During the maturation stage, the enamel layer hardens. The crystals stop their growth in length and start to grow in width and thickness, which results in a mineralised tissue with more than 95% mineral content. After the maturation stage,the ameloblasts degenerate with the other layers of the enamel-epithelium during tooth eruption (Alalunsua, 2010).

The development of the second primary molars occurs somewhat earlier than the development of the first permanent molars and permanent incisors, but the periods of their development overlap and the maturation of the permanent molar is slower. If a risk factor occurs during this overlapping period, a hypomineralization might occur in the primary and permanent dentition. Because the second primary molars erupt 4 years earlier in life than the first

permanent molars, deciduous molar hypomineralisation might be a clinically useful predictor for MIH (Fagrell, et al, 2011).

The evaluation of the etiological factors for severe demarcated enamel opacities in the first permanent molars from a database that contained approximately 4,000 variables with the purpose to prospectively investigate risk factors for immune mediated diseases, approximately, 17,000 children take part in the study. Medical data information from interviews, questionnaires were collected at delivery, at 1, 2.5 years of age with follow up at 5, at 8-9 and at 12 years (Fagrell, et al, 2011).

All information collected for each child covering somatic growth:

1- **Prenatal**: including mother's diseases, medications and treatments received during pregnancy (Lygidakis, et al. 2008).
2- **Perinatal**: Gestational age, mode of delivery, birth weight, duration of Labor neonatal comorbidities admission neonatal intensive care unit (NICU) (Lygidakis, et al. 2008, Ghanim, et al. 2011)**.**
3- **Postnatal**: Feeding type,medication and vaccinations during first 3 years of child life, socioeconomic factors and nutrition during same period (Lygidakis, et al. 2008, Ghanim, et al. 2011).

A positive association between severe demarcated opacities in permanent first molars with breastfeeding for more than 6 months, late introduction of gruel and late introduction of infant formula. Moreover a combination of these variables increased the risk to develop severe demarcated opacities by five times more .According these results the authors concluded that nutritional conditions during first 6 months of life may influence the risk to develop severe demarcated opacities in first permanent molars (Fagrell, et al, 2011).

7- Body mass index and MIH:

A substantial burden of dental caries is found among minority, lower socioeconomic groups and those who are medically compromised or disabled. Effect of body mass index on molar incisor hypomineralization was the concern of many researchers but much interest was on very low birth weight (VLBW) children that can be susceptible to dental caries due to biological and socioeconomic factors (Hong, et al. 2005 and Nelson, et al 2011).

Biological problems include the increased prevalence of developmental defects of enamel in the primary and permanent dentition as a result of systemic (illnesses/disturbances/deficiencies of prematurity) and local (trauma from endotracheal tubes) causes. Developmental enamel defects are more prevalent in VLBW children who require prolonged oral endotracheal intubation. These defects typically persist at least 10 years into childhood (Hong, et al. 2005, Nelson, et al. 2011).

The common hypothesis for developmental defects of enamel in VLBW the neonatal period is through altered calcium children during homeostasis because of the many systemic illnesses. Additionally, many high-risk VLBW (HR-VLBW) infants experience local factors because of orotracheal intubation and mechanical ventilation that has a traumatic effect on the soft forming enamel resulting in enamel defects. DDE is thought to be the result of abrupt, short-term or long-term ameloblastic insults during the secretory or maturation phase of tooth development which can indicate if the insult occurred in utero or after birth. For the permanent incisors and first molars, the secretory phase begins in utero and the maturation phase starts at birth, and thus inadequate mineralization or trauma during these periods can result in DDE. Demarcated opacities in early erupting permanent teeth (incisors and first molars) are associated with neonatal risk factors while diffuse opacities are associated with infant fluoride and amoxicillin exposure. Demarcated opacities in permanent teeth have been associated with caries in the precursor primary teeth. A few

studies have suggested a relationship between preterm birth, birth weight and primary dental caries. Hypoplasia in the primary dentition of VLBW children. The two studies A significant association was found between caries and enamel conflicting results regarding this association. These permanent dentition in the permanent dentition of low-birth-weight (2,500 g) children have did they look specifically at VLBW children (Hong et al., 2005).

Studies did not measure enamel defects as they relate to dental caries, nor did they look specifically at VLBW children (Hong et al., 2005).

Conclusion

There is still need for further studies to shed light on causative factors and their effect as well as on improving the permanence of restorations.

Although a specific etiology remains to be established, it appears likely that these defects are the result of a combination of genetic predisposition and environmental insults. Managing patients with MIH requires a variety of patient management approaches, materials, and techniques. The dental hypersensitivity these patients experience and associate with their first permanent molars can make them extremely challenging to manage from a behavioral and anesthetic point of view.

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