ACTA SCIENTIFIC DENTAL SCIENCES (ISSN: 2581-4893)

Volume 4 Issue 4 April 2020

Review Article

Molar Incisor Hypomineralization

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Received: January 07, 2020

Published: March 17, 2020

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Abstract

Molar Incisor Hypomineralisation (MIH) is a type of enamel defect which affects first molars and incisors in the permanent dentition. MIH is considered a worldwide problem and it occurs in children under 10 years of age. This developmental condition is caused due to lack of mineralisation of enamel during maturation phase and due to interruption to the function of ameloblasts. Many other factors such as genetics and medical problems during pregnancy are also been associated with MIH. It appears as discoloration on one to four affected permanent molars and the associated incisors. The enamel of the affected teeth appears yellow, brown, cream or white and thus sometimes referred to as 'cheese molars'.

Keywords: Hypomineralization; Childhood; Cheese Molars; Prevalence

Introduction

Tooth development influenced by factors like febrile illness, antibiotic use and excessive fluoride intake during, before or after birth [1]. Depending on the timing and duration of these factors, teeth may undergo various pathological conditions [2].

Developmental enamel defects are classified as

- Permanent hypomineralized first molars (FMs).
- Idiopathic enamel hypomineralization of the FMs.
- Cheese molars.
- Hypomineralization irrespective of dental fluorosis.
- Demineralized First Molars.

During the developmental stage teeth appears normal during eruption but sometimes corrupt mineralization may result in "hypomineralization" while interrupted in formation but normal mineralization might lead to "hypoplasia" [3,4].

During European Academy of Pediatric Dentistry (EAPD) in 2001, researchers concluded that the condition was described as the "Molar Incisor Hypomineralization (MIH) in limited and qualitative defects of enamel origin affecting one of multiple molars with or without incisor retention.

Etiology

Prenatal Period

- Infections of urinary tract
- A and D Vitamin defects
- Cardiologic diseases
- Maternal nausea and vomiting

- Rubella Embryopathy
- Diabetes Mellitus
- Anemia.

Perinatal period

- Twin deliveries
- Prolonged delivery
- Preterm Delivery
- Hypocalcemia
- Hypoxia
- Cesarean sections.

Postnatal Period

- Medical problems like pneumonia, asthma, urinary tract infections, chicken pox etc.
- Medication like antibiotics
- Systemic etiological factors during dental development.
- Use of Fluorides.

Other causes for Molar Incisor Hypomineralizaton are genetic factors, environmental conditions, respiratory tract problems, oxygen starvation dioxins, of the child combined with a low birth weight, calcium and frequent childhood diseases.

Prevalence [3-6]

Study and year of publication	Study sample	First Permanent Molar(FPM) Prevalence reported
Alaluusua., *et al.* (1996)	97 Finnish children 12 years old.	25% children had FPM hypomineralization
Jalevik., *et al.* (2001)	516 Swedish children of 7-8 years old	18% children had MIH; affected children averaged 3.2 hypomineralized teeth, of which 2.4 were FPMs
Leppaniemi., *et al.* (2001)	488 Finnish children 7-13 years old	19% of children had "nonfluoride hypomineralizations" of FPMs
Weerheijm., *et al.* (2001)	497 Dutch children 11 years old and born in 1988	10% of children had "cheese molars (idiopathic enamel disturbances)"; 79% of affected children had 2 or more affected FPMs
Dietrich., *et al.* (2003	2,408 German children 10-17 years old and born 1985-1992	6% of children had MIH; affected children averaged 4.8 hypomineralized teeth, of which 2.2 were FPMs
Lygidakis., *et al.* (2004)	2,640 Greek children attending a children's community health center	6% children had MIH
Kirthiga M., *et al.* (2015)	2000 school children were reviewed	179 cases were identified with a prevalence rate of 8.9% with MIH. percentage of girls and boys affected were 9.3 and 8.7
Parikh DR., *et al.* (2015)	1366 school children were carried out	Prevalence was 9.2 of MIH. Both males and females were equally affected.

Table 1

Clinical features

Clinically, Molar Incisor Hypomineralizaton has large demarcated opacities of altered enamel translucency. This defective enamel is whitish-cream or yellow-brown in color. The opacities are limited to the incisal or cuspal one third of the crown and it rarely involves the cervical third.

The intact enamel surface is hard, smooth and often hyper mineralized following post eruptive maturation; subsurface enamel is soft and porous. There is a subclinical pulpal inflammation due to enamel porosity which often leads to hypersensitivity experienced by some individuals.

In the comparative study of pulps of non-carious hypomineralized FPM to apparently sound FPM from MIH affected individuals, it was concluded that the changes were indicative of inflammatory changes.

Levels of molar Incisor hypomineralization and its symptoms

Mild molar incisor hypomineralization

- Demarcated opacities are in non-stress-bearing areas of First Permanent Molars.
- Isolated opacities.
- No enamel loss from fracturing is present in opaque areas.
- No history of dental hypersensitivity.
- Incisor involvement is usually mild if present.
- No caries associated with the affected enamel.

Moderate molar incisor hypomineralization

- Intact atypical restorations.
- Demarcated opacities are present on occlusal/ incisal third of teeth without post eruptive enamel breakdown.
- Post eruptive enamel breakdown/ caries are limited to 1 or 2 surfaces without copal involvement.
- Dental sensitivity is generally reported as normal esthetic concerns.

Severe molar incisor hypomineralization

- Post eruptive enamel breakdown
- History of dental sensitivity
- Widespread caries is associated with the affected enamel.
- Crown destruction can readily advance to involve the dental pulp.
- Defective atypical restoration is present.
- Esthetic concerns are expressed by the patient or parent.

Difference between hypomineralization, mild fluorosis and hyper mineralization

Diagnosis

Examination was carried under clean wet teeth of 8 years age, as at this age all permanent first molars and most of the incisors are erupted.

Hypomineralization	Dental Fluorosis	Hyper mineralization
Less mineralized tissue when ameloblast do not undergo complete maturation.	Overexposure to fluoride during the time of enamel formation.	During maturation when there is excessive mineralization tissue.
Presents as whitish/ yellow or brown in color.	Presents as white spots or brown mottled enamel.	Presents as staining and pitting of teeth.

Table 2

Permanent first molar should have good condition and without excessive post-eruptive breakdown [6-8].

Diagnostic criteria for FPMs currently available are the modified defect of dental enamel (DDE) index given by federation dentaire international.

Mild	<30% of enamel surface area is visibly disrupted (this encompasses the entire range reported in most other studies).
Moderate	31 to 49% of the tooth's enamel surface area visibly disrupted.
Severe	>50% of the tooth's enamel surface area visibly disrupted.

Table 3

Definitions of the criteria used for diagnosing MIH (Wertheim 2001)

Opacity

Any defect involving an alteration in the translucency of the enamel, variable in degree. The defective enamel is of normal thickness with a smooth surface and can be white, yellow or brown in color. The border is demarcated.

Pre-eruptive breakdown

A defect indicated by deficiency of the surface after eruption of the tooth. This may be caused by such factors as trauma and attrition.

Atypical restoration

Size and shape of restoration do not conform to typical restorative characteristics. In most cases, restorations will be extended to the buccal or the palatinal smooth surface. At the border of the restoration, opacity may be noticed [6].

Extraction due to MIH

Absence of a molar should be related to the other teeth of the dentition. Absence of a first permanent molar in a sound dentition is suspected to have been an MIH.

Demarcated opacity.

Post eruptive enamel breakdown (PEB)

The defective enamel is of normal thickness with a smooth surface and can be white, yellow, or brown in color. Alterations in the translucency of the enamel, variable in degree.

Loss of initially formed surface enamel after tooth eruption. A defect that indicates deficiency of the surface after eruption of the tooth. The loss is often associated with a preexisting demarcated opacity.

Differential diagnosis: Milder forms of dental fluorosis and enamel opacities of non-fluoride origin (from frejescov., *et al.* 1988)

Atypical restoration

Extracted molar due to MIH

The size and shape of a restoration are not matching to the temporary caries picture. At the border of the restorations frequently an opacity can be noticed. In incisors a buccal restoration can be noticed not related to trauma.

Absence of a first permanent molar should be compared to the other teeth of the dentition. Suspected for extraction due to MIH are opacities or atypical restorations in the other first permanent. Also the absence of first permanent molars in a sound dentition in combination with demarcated opacities on the incisors is suspected for MIH [2-4].

It is rare incidence that incisors will be extracted due to MIH.

Differential diagnosis

- **Enamel Hypoplasia:** Defect is quantitative and is associated with reduced localized thickness of enamel whereas hypo mineralization is a qualitative defect affecting enamel translucency.
- **Fluorosis:** The enamel opacities of Fluorosis are diffuse, in contrast to the well-demarcated borders of hypomineralized enamel seen in MIH.
- **Amelogenesis Imperfecta:** In Amelogenesis Imperfecta, all teeth are affected and may be detected pre-eruptively on radiograph. There is usually a positive family history in cases of Amelogenesis Imperfecta.

Characteristics	Dental fluorosis	Enamel opacities
Area Affected	The entire tooth surface (all surfaces) often enhanced on or near tips of cusps/incisal edges.	Usually centered in smooth surface of limited extent.
Lesion Shape	Resemble line shading in pencil sketch which follows incremental lines in enamel (perikymata). Lines merging and in score 3 a cloudy appearance. At cusps/ incisal edges formation of irregular white caps (snow cap).	Round or oval.
Demarcation	Diffuse distribution over the surface of varying intensity.	Clearly differenciated from adjacent normal enamel.
Color	Opaque white lines or clouds, chalky appearance. 'Snow caps' at cusps/incisal edges. Score 3 may become browinsh discolored at mesio-incisal part of central upper incisors after eruption.	White opaque or creamy- yellow to dark reddish- orange at time of eruption.
Teeth affected	Always on homologous teeth. Early erupting teeth (incisors/ 1st molars) least affected. Premolars and second molars (and third molars) most severely affected.	Most common on labial surfaces of single or occasionally homologous teeth. Any tooth may be affected but mostly incisors.

Table 4

Management of molar incisor hypomineralization

A clinical management approach MIH

1. **Risk identification:** Medical history will be taken for putative etiological factor.
2. **Early diagnosis:** Monitor at-risk molars on radiographs if possible. Examine these teeth during eruption of teeth.
3. **Remineralization and desensitization:** Apply localized topical fluoride.
4. **Prevention of dental caries and PEB:** Instruction on oral hygiene maintenance at home care program should be given.
    - Reduce cariogenicity and erosivity of diet.
    - Place pit and fissure sealants.
5. **Restorations or extractions:** Place intracoronal (resin composite) bonded with self-etching primer adhesive or extracoronal restorations (stainless steel crowns).
6. **Maintenance:** Examine margins of restorations for Pre-Eruptive Breakdown.
    - Examine full coronal coverage restorations in the long-term.

Preventive treatment

1. Topical fluoride application.
2. Children should start using high fluoride content tooth paste.
3. Apply a CPP-ACP topical cream daily using a cotton bud.
4. Desensitizing toothpaste.

Curative treatment

1. **Fissure Sealant:** Provides temporary protection to the teeth against caries and sensitivity.
2. **Restorative treatments:** This is an initial approach for complete removal of the defective enamel. The second approach is the removal of the porous enamel while leaving the enamel with increased resistance to drill intact.
3. **GIC's:** Due to the adhesive property, GICs are good isolators and also which release fluoride. However, use is restricted in hypomineralized permanent teeth.
4. **Resin composite materials:** Compared with other restorative material, the composite resin material provides longer stability, Self-etching adhesive have better bond strength than to MIH affected enamel.
5. **Amalgams-** Amalgams mechanically adhering to the restoration cavity and demonstrating frequent leak in restored edges. They are also proved as bad isolators not chemically bound to the dental tissues [4].
6. **Stainless steel crowns:** Stainless steel crowns (SSC) has the best option for restoration of teeth with severe coronal malformation and can be implemented temporarily until the adjacent permanent tooth erupts and reaches the occlusion plane.
7. **Laboratory fabricated crowns:** Not preferred in clinical applications due to short crowns, large pulps of young permanent teeth, expensiveness, causing wear of the opposing teeth and difficulty of finishing the crown edges in permanent teeth not completely erupted.
8. **Incisor treatment:** Composite laminates with or without removal of a certain amount of enamel tissue is more effective in improving the esthetic appearance of tooth [5,6].

Conclusion

Molar Incisor Hypomineralization occurs as a result of the multifactorial reasons during the child's prenatal term putting the health in danger such as systemic diseases and malnutrition during the first 3 years of children age.

Clinical MIH is a serious problem for children and dentists. Tooth become sensitive to cold and hot food and cause mild to severe pain. The probability of maintaining tooth structure integrity in molars affected, can be restored with GIC (mainly in single-surface tooth restorations). It can be concluded that invasive treatment – like complete removal of the affected area – should be postponed until the child is mature enough to understand and cooperate with more complex rehabilitation and treatment procedures.

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