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Missing links of Molar Incisor Hypomineralization: A review

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Abstract:

Dental enamel is an unusual tissue in that once formed it is not remodeled, unlike other hard tissues such as bone. Because of its non remodeling nature, alterations of enamel during its formation are permanently recorded on the tooth surface. As enamel formation can be affected by many factors, the changes induced in the enamel formation, can provide clues as to the timing and nature of these events. Enamel defects may thus be studied as a marker of many adverse biological events occurring during the time of its development. One such developmental defect of the enamel occurring due to changes in the environmental factors causing permanent damage of the enamel is Molar Incisor Hypomineralization (MIH). It describes the clinical picture of hypomineralization of systemic origin affecting one or more first permanent molars that are associated frequently with affected incisors. The treatment comprises of complex care including management of behavior and anxiety of the young child along with aiming to provide a durable restoration under pain free conditions. This paper aims at describing the various etiological factors of this condition and to identify the individuals at potential risk to develop MIH thereby attempting to prevent damage caused due to this condition.

Key words: Molar incisor hypomineralization, post eruption breakdown, permanent first molars, permanent incisors.

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Introduction:

Tooth enamel is unique among mineralized tissues because of its high mineral content. Enamel is made up of highly organized, tightly packed crystallites that comprise 87% of its volume and 95 of its weight. Other mineralized tissues have about 20% of organic matter whereas mature enamel has less than 1% of organic matter. Enamel crystallites contain more than 1000 times the volume of corresponding crystals in bone, dentin and cementum. Superior organization and mineralization give dental enamel its outstanding physical properties, making it hardest tissue in the vertebrate body. Despite its hardness, tooth enamel can be destroyed fairly rapidly by dental caries. Additionally enamel is also afflicted by various structural defect which could be inherited or acquired.[1]

Developmental defects of enamel are disturbingly prevalent, potentially afflicting over 10% of population with multiple burdens including dental pain, disfigurement and increased caries risk. Classically, attention has centered on rare genetic disorders termed Amelogenesis Imperfecta and on dental fluorosis, a widespread defect acquired from excessive fluoride intake. Over the past decade, however another acquired defect has been causing increasing concern to clinicians worldwide. Primarily disrupting mineralization of permanent first molars and incisors, this condition is commonly termed as Molar Incisor Hypomineralization (MIH).[2]

This condition has been described by various terms in the literature like hypomineralized permanent first molars, idiopathic enamel hypomineralization in permanent first molars and cheese molars. The term molar incisor hypomineralization was introduced in the year 2001. Although the denominations differ, the clinical description of the phenomenon remains similar.[3]

Diagnosis and clinical appearance:

Criteria for the diagnosis of demarcated opacities, post eruption breakdown (PEB), atypical restorations and extracted permanent first molar (PFM) due to MIH were developed by Weerheijm et al. Clinical appearance (Table 1) in less severe cases is characterized by well demarcated opacities. Defective enamel is white cream or yellow brown in color, of normal thickness with smooth surface and has distinct boundary adjacent to normal enamel (Fig 1). The opacites are limited to the incisal or cuspal third of the crown rarely involving the cervical one third. Surface enamel is hypermineralized due to post eruption mineralization (Fig 2). More severe cases are characterized PEB with soft porous enamel which looks like discolored chalk or Old Dutch cheese.[4,5]

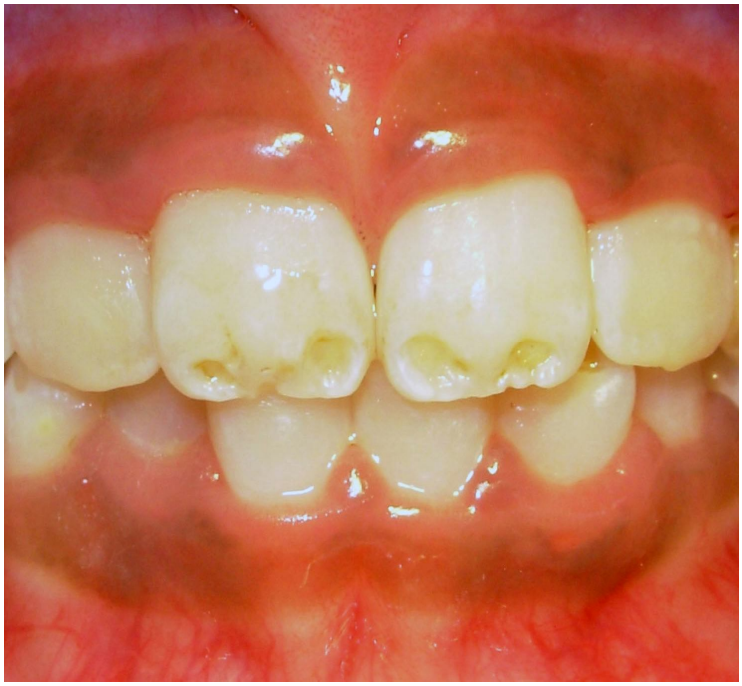

Fig 1: Affected incisors with well demarcated yellow brown areas.

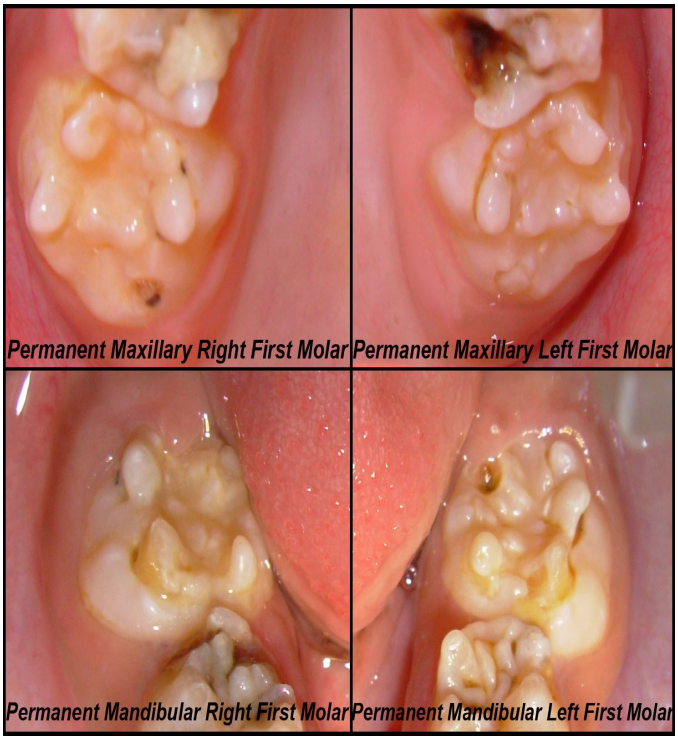

Fig 2: Affected Permanent First Molars.

Predisposing factors:
Hypomineralization is thought to be due to disturbed resorptive potential of ameloblasts and proteolytic enzyme inhibition leading to protein retention and interference with crystal growth and enamel maturation. Most common predisposing factors for disrupted amelogenesis of PFM include systemic and environmental insults influencing natal and early development.

1) Systemic illness:
Conditions common in first 3years, such as upper respiratory tract diseases, asthma, otitis media, tonsillitis, chicken pox, measles and rubella are associated with MIH. Antibiotic usage has also been implicated. Due to the concurrence of disease and antibiotic therapy, however, it is difficult to ascertain whether MIH is associated with the disease or antibiotic therapy.[6] Other systemic illnesses associated with MIH are nutritional deficiencies, brain injury, cystic fibrosis, syndromes of epilepsy and dementia, lead poisoning and repaired cleft lip and palate.

2) Gestational age:
Premature delivery or preterm birth has been associated with increased prevalence of enamel defects in the permanent dentition. Preterm birth can be associated with respiratory difficulties, hyperbilirubinaemia, metabolic disturbances including hypocalcemia and hypoglycemia, hematological disorders and intracranial hemorrhage. The enamel defect severity increases and decreasing gestational age and lower birth weight.[7]

3) Affect of low pH:
Regulation of pH during mineralization is considered necessary for normal apatite deposition and crystallite growth. Sui et al reported that reduced enamel matrix pH disrupted the crystal growth and proteinase function which can result in protein retention and hypomineralization. Speculatively conditions affecting matrix pH during enamel maturation may predispose MIH. A medical condition affecting the pH like cystic fibrosis has been found to be associated with MIH. Studies have shown statistically significant correlation between cystic fibrosis and MIH.[8,9]

4) Lack of calcium phosphate:
An optimal serum calcium level is important for initial dentin mineralization and proper enamel matrix secretion and mineralization. Impaired calcium metabolism plays a role in development of hypomineralized enamel. Studies using secondary ion mass spectrometry and x – ray microanalysis revealed that increased severity of hypomineralization correlated positively with increasing carbon concentration and decreasing concentration of calcium and phosphorus. This resulted in significantly lowered calcium / phosphorus ratios in enamel. Moreover proteins like amelogenin, ameloblastin and enamelin which are essential for enamel matrix formation all belong to the secretory calcium-binding phosphoprotein gene family and are controlled by vitamin D[10] and also certain proteinases processing amelogenins during enamel

mineralization at the secretory and early maturation stages like Enamelysin (MMP-20), is also calcium-dependent matrix metalloproteinase. Hence hypocalcemia in any form can predispose a child to develop MIH.

5) Duration of breast feeding:

Various studies have been conducted to study the association between duration of breast feeding and occurrence of hypomineralization. Hypomineralization due to prolonged breast feeding is thought to be due to the exposure to polychlorinated dibenzo-*p*-dioxins (PCDDs). The PCDDs belong to a class of environmental pollutants known as polyhalogenated aromatic hydrocarbons. PCDDs, polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs) are collectively called dioxins and dioxin-like compounds, although the term "dioxins" strictly refers only to PCDDs. The most toxic and widely studied of this general class of compounds is 2,3,7,8-tetrachlorodibenzo-p-dioxin, which is often called simply "dioxin" and which represents the reference compound for this class of compounds. PCDDs are ubiquitous in the environment and liposoluble, thus they accumulate in fat and enrich in food chains. In infancy, children can be exposed to these compounds mainly via breast-feeding. An infant can get even 25% of the mother's dioxin load via lactation and the accumulation of dioxins and dioxin-like compounds in fat may prolong the duration of their action. In human adults, most of TCDD is stored in the adipose tissue and has a half-life of approximately 7 years. Studies on Finnish children has shown increase in severity and number of defects in those exposed to higher amounts of PCDD and furan via mother's milk compared to those less exposed. Similar results were obtained by Alaluusua et al (1996). In this study two child populations were selected, one with frequency of severe hypomineralization and another with prolonged breast feeding. The results of the study indicated association between duration of breast feeding and hypomineralization. However few other studies do not indicate any significant association between duration of breast feeding and occurrence It thus appears that in study groups selected on the basis of either frequent hypomineralization or prolonged breast of hypomineralization in the PFM. feeding the association between exposure to harmful agents and hypomineralization becomes more evert than in unselected child population.[5,11-13] However many researches are described (Table 2), more research is still required in this area to prove any correlation.

Table 1: Clinical criteria for differentiation between MIH, Amelogeneis Imperfecta and Fluorosis:

Condition	Findings
Amelogeneis Imperfecta (AI)	involves all the teeth, family history is present, teeth may appear taurodont on radiograph
Fluorosis	Diffuse opacities which are caries resistant. Number of teeth involved depends on the time of exposure.
Molar incisor hypomineralization	Involves PFM and incisors, well demarcated opacities will be present which will be caries prone. Only severe cases may resemble AI. No appearance of taurodont on radiograph.

Table 2: Review of the Studies related to the Molar Incisor Hypomineralization

Reference	Aim of the study	Type of study	Outcome
Commision on Oral Health Research and Epidemiology – Report of an FDI working group, Leader: Clarkson J 1992	Review the acceptability of Developmental Defects of Dental Enamel Index (DDE Index)	Review and technical report	1) Recommendations on classification of developmental enamel defects. 2) Guidelines for clinical examination, recording, analysis and presentation of data corresponding to developmental dental defects.
Van Amerongen W E et al 1995	Pilot study to identify the possible etiology of cheese molars.	Retrospective study	Positive correlation of birth related condition and childhood systemic conditions like bronchitis, pneumonia, infections of upper respiratory organs, high fever, Gastro Intestinal disorders with occurrence of hypocalcified molars.
Kim Seow W 1996	Study of development of permanent dentition in very low birth weight children.	Longitudinal cohort	1) Very low birth weight (VLBW) prematurely born children have delayed dental development. 2) Higher prevalence of enamel defects in first permanent molars and incisors in VLBW children.
Simmer PJ et al 2001	Impact of dental enamel formation on clinical dentistry	Literature review	1) Discussion on genetic control of enamel formation, dentino enamel junction formation, enamel crystal elongation and crystal thicknening as related to enamel properties. 2) Quantitative effect of different enamel proteins on enamel properties.
Weerheijm K L et al. 2001	Prevalence of cheese Molars in eleven-year-old Dutch Children	Longitudinal Standardized Epidemiological survey	10% of the Dutch eleven-year-old children showed the presence of cheese Molars.
Weerheijm K L et al. 2001	Describe briefly the phenomenon of	Short communication	A common consensus was adopted and the type of

	Specific type of Enamel Hypomineralization and find a common name for the condition		enamel Hypomineralization with typical features of affecting the permanent first molars and permanent Incisors was named as "Molar Incisor Hypomineralization"
Sui W et al 2003	Effect of altered pH regulation on mineral content during enamel development	Animal study (Mice) – Biochemical pH estimation study	Reduced pH results in hypomineralization due to altered calcium influx during enamel maturation.
Weerheijm K L 2004	Relate the knowledge of MIH to the Etiology, clinical presentation and Management	Literature Review	1)Consider childhood diseases as a precursor for MIH and plan frequent monitoring of erupting first permanent molars 2)Management of MIH includes Pain management followed by functional management with long term favorable prognosis
Vanessa William et al 2006	Review and recommendations for clinical management of MIH	Literature review	1)4-25% prevalence of MIH 2) No. of affected permanent first molars can vary from 1 – 4 depending on the severity. 3) Risk of involvement of permanent maxillary incisors appears to increase when more permanent first molars are affected.
Helen D Rodd et al 2007	Pulpal status of hypomineralized permanent molars.	Indirect immunofluoresecence of extracted hypomineralized permanent molars	1) Overall increase in neural density with highly varicose morphology of the nerve fibers. 2) leucocyte common antigen – immunoreactive (LCA – ir) cells significantly increased. 3) No vascular changes.
sMangum J E et al 2010	Protein composition of hypomineralized enamel.	Proteomic analysis and mineral binding assay	1) Increased amelogenins is pathognomonic of MIH. 2) 3- 15 fold higher content of protein in hypomineralized enamel than normal.

			3) greater amount of accumulated proteins from saliva in hypomineralized enamel
Chan Y L et al 2010	Evaluation of microstructure of enamel from specific region of MIH teeth.	Focused ion beam and nano indentation technique.	Notable alteration in the prism sheath of enamel in MIH teeth at its transitional region as a possible contributing factor to lowered mechanical properties of affected teeth.
Farah R A etal 2010	Comparison of relative amounts and nature of proteinous content of sound and MIH enamel.	Electrophoretic profiling and Mass spectrometric study.	1) 8 – 21 fold higher protein content in MIH enamel. 2) Increase in serum albumin and Anti Trypsin and presence of serum anti thrombin in MIH enamel. 3) Albumin mediated inhibition of crystal growth during enamel formation as a probable cause for enamel hypomineralization.

Pathophysiology for hypomineralization caused due to PCDDs:

The majority of adverse effects of PCDD are mediated by the aryl hydrocarbon receptor (AhR), which, after binding to TCDD translocates to the nucleus, dimerizes with the aryl hydrocarbon receptor nuclear translocator (ARNT) and binds to DNA. This initiates the transcription of xenobiotic-metabolizing enzymes such as cytochrome P450 1A1 (CYP1A1), which is known to be the primary target for PCDD- inducible gene expression. CYP1A1 metabolizes PCDD compounds to active metabolites that are for the most part responsible for the toxic effects of PAHs All in all, the understanding of the connections between PCDD as a ligand, signaling mechanism and a certain toxic outcome remains minimal.[13] Dioxins and dioxin-like compounds are also suggested to increase the prevalence of orofacial clefts.

Reason for susceptibility of only PFM and Permanent Incisors:

The stage of development of the dentition is dependent on the age of the child and therefore, susceptibility of different teeth to developmental disturbances at different times varies. Development of the first permanent molars and incisors begins at the fourth gestational month and hard tissue formation in them starts around or soon after birth. Enamel formation in the upper first incisors has been completed by the end of the fifth year of life and in the first molars at about three years. Accordingly, human permanent incisors and first molars are at greatest risk for defects caused by systemic environmental factors up to the first years of life.[14]

Some characteristic features of teeth with MIH:

1. Pulpal status of hypomineralized molars:

Immunohistochemical study conducted by Rodd et al (2006) showed that non carious

hypomineralized molars have underlying pulpal inflammation, as demonstrated by an increase in the pulpal innervation density and immune cell accumulation.[15] In MIH teeth, porous hard tissues and exposed dentin may predispose to pulpal ingress of bacteria and other oral irritants. Following tissue inflammation, a variety of morphological and cytochemical neuronal changes may occur. Changes include neuronal branching and altered expression of neuropeptides and ion channels. These features are all indicative of peripheral sensitization, whereby the threshold of neuronal activation is reduced.[16]

Following administration of local anesthetic, some MIH patients continue to experience pain on instrumentation. Failure to achieve adequate levels of pulpal analgesia may be attributed to peripheral sensitization. Some studies on tissue inflammation have shown significant changes in neuronal expression of ioninc sodium channels. Increased expression of tetrodotoxin resistant sodium channels has been linked to hyperalgesia and altered sensitivity to local anesthesia.[17]

2. Bacterial invasion in the dentinal tubules:

Studies conducted by Tobias et al (2008) indicate the presence of bacteria in the dentinal tubules of the hypomineralized teeth. Bacteria do penetrate through hypomineralized enamel with a macroscopically intact surface into the dentin. Presence of highly microporous enamel in severe MIH constitutes the possible pathway for transport of the bacteria to the enamel. Because MIH is an enamel aberration which is seen at the time of eruption of the first molars, the dentinal tubules are still wide in which bacteria could easily penetrate. The study indicated that pulpal reactions were limited to the presence of reparative dentin, and in few cases presence of inflammatory cells indicating the response of the bacterial invasion in the dentinal tubules.[18]

3. Microstructure of enamel in and around the affected area:

The success of the restorations placed on affected teeth depends on the strength of the remaining enamel. The major problem with these teeth is the quality of the remaining enamel which often ruptures and leaves gap between restoration and tooth. Information on mechanical properties of enamel in MIH teeth would therefore be critical to their successful restoration. Studies have shown existence of transitional zone between the affected and the unaffected enamel. The enamel in this transitional region adjacent to affected enamel has notable alterations in the prism sheath which likely contributes to the lowered mechanical properties.[19]

4. Protein content of the Hypomineralized enamel:

It is reported that the classic etching patterns obtained with sound enamel were absent in MIH enamel. The increased protein content of the MIH enamel limits the access of acid to the hydroxyapatite crystallites. The high resistance of MIH enamel to acid etching is consistent with an increased organic content rather than carbonate substitution of the normal apatite lattice. MIH showed 8 – 21 fold higher protein content than sound enamel. Brown enamel has the highest protein content (15 – 21 fold greater), whilst the protein content of white/ opaque and yellow enamel are both markedly higher than sound enamel. Serum albumin, alpha 1 antitrypsin and type I collagen is found to be present in abundance in these teeth. Only brown and yellow enamel is shown to have antithrombin III.[20]

5. Syndrome associated with MIH:

22q11deletion syndrome is found to be associated with MIH. The patients with 22q11 deletion syndrome have many and complex medical problems including hypocalcemia and or/ hypoparathyroidism. Studies on these patients have shown a relationship between high numbers of medical problems in the patients and enamel deviations. More research is required in this field relate the association between medical problems and risk of MIH.[21]

Management:

Hypomineralized PFMs are at risk of post eruptive breakdown after erupting in the oral environment, where masticatory forces and dietary challenges lead to enamel chipping, dentin exposure and early dental caries. The restorative management usually depends on the defect's severity, child's cooperation and age. The severely affected teeth are soon in need of restoration due to disintegrating enamel and subsequent caries. The treatments can be complicated due to difficulties in achieving anaesthetia, managing the child's behavior, determining how much enamel to remove and selecting a suitable restorative material. The prismatic morphology in the porous enamel is altered and patients frequently report of loss of the filings as well as continuing disintegration of porous enamel. Consequently, the affected teeth often require repeated treatment.[22,23]

Children at risk of MIH should be identified prior to the eruption of PFM based on the history of putative etiological factors. As the teeth are susceptible to caries and erosion, the cariogenicity and erosivity of the diet should be assessed and appropriate recommendations can be made for dietary modifications. Remineralization and desensitization therapy should commence as soon as the defective surfaces are accessible. This can be accomplished with Casein phosphor peptide amorphous calcium phosphate (CPP-ACP) and fluoride. CPP-ACP enhances remineralization by creating state of super saturation followed by deposition of calcium and phosphate ions at the enamel surface. Topical fluorides, delivered as concentrated varnishes or gels, can remineralize enamel, reduce sensitivity and enhance resistance to demineralization by providing a reservoir of fluoride ions for deposition as fluorapatite during remineralization. As a part of preventive management GIC fissures sealants can be done in partially erupted molars where moisture control is sub optimal.[5]

Restoration of hypomineralized teeth:

The choice of the materials depends on the defect severity, the age and cooperation of the child. Adhesive materials are chosen due atypical cavity outlines following removal of hypomineralized enamel. For dentin replacement or as an interim restoration, GIC provides placement ease, fluoride release and chemical bonding. The resin modified GIC offer similar advantages and incorporation of resin and photoinitiators improves handling, wear resistance, fracture toughness and fracture resistance.[24,25]

The resin composites are material of choice in MIH where defective enamel is well demarcated and confined to 1 or 2 surfaces with supragingival margins and without cuspal involvement. Resin composites are not successful in large defects because the etch pattern shows preferential dissolution of rod peripheries, loss of inter rod enamel resulting in enlarged inter rod space and inter crystal space is minimal probably reducing surface area available for bonding. The enamel adhesive interface of hypomineralized enamel is porous with cracks without a uniform hybrid layer. Failures with composite restorations have been thought to be due to these reasons. Hence it is recommended to remove all the hypomineralized enamel prior to placement of resin composite restorations and it is also suggested to pretreat the enamel with 5% sodium hypochlorite to remove the protein encasing the hydroxyapatite prior to etching.[26,27]

Full coverage restorations with SSC are performed when PFMs have moderate to severe PEB. These crowns prevent further tooth deterioration, control tooth sensitivity, establish correct interproximal contact and proper occlusion, are not as technique sensitive and costly as cast restoration and require little time to prepare and insert. When PFM are severely hypomineralized, restoration may be impossible and extraction must be considered. In such cases, early orthodontic assessment is

recommended for the management of the developing occlusion.[28]

Conclusion:

The prevalence of the MIH appears to be increasing and managing affected children is a common problem. As the etiology most commonly is multifactorial, preterm children and those with poor general health at the first 3years of life are considered to be at risk to develop MIH. These children should be monitored frequently during the eruption of PFM so that remineralization and preventive measures can be instituted at the earliest. This area is still not clearly understood hence requires lot of research about the etiology of the disease so as to prevent the disease from occurrence at the earliest and to prevent the severity of the disease.

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