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Research Paper

MIH- Review of Literature

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I. INTRODUCTION:

Dental enamel is a unique, highly mineralized tissue of ectodermal origin. It is characterized by a lack of metabolic activity once formed, meaning disturbances during development can manifest as permanent defects in the erupted tooth. Disturbances in the initial matrix secretion phase of amelogenesis will most likely present as quantitative or morphologic defects (hypoplasia), whereas disruptions to the calcification or maturation processes may produce morphologically normal but structurally or qualitatively defective enamel (hypomineralization/hypomaturation).[1]

Molar Incisor Hypomineralization (MIH) was first noted in Sweden in the late 1970s [Koch et al, 1987][4] due to the chronological distribution of enamel defects. Also referred to as "hypomineralized" permanent first molars [Jalevik & Noren, 2000][9], "idiopathic enamel hypomineralization" [Koch et al, 1987, Fearne et al, 2004],[4 5] "dysmineralized" permanent first molars [Croll T. 1991],[6] "nonfluoride hypomineralization" [Holtta et al, 2001, Leppaniemi et al, 2001],[7 8] and "cheese molars" [Van Amerongen & Kreulen, 1995, Weerheijm et al, 2001], Weerheijm et al. [2001] stated that when discussing these developmental defects of dental enamel it would be desirable to use one name, one that made no reference to any possible etiology.[3] He suggested 'Molar-Incisor Hypomineralization' (MIH), and gave a definition as "hypomineralization of systemic origin of 1-4 FPM, frequently associated with affected incisors." This anomaly can involve only primary or permanent teeth, or may both dentitions.

Enamel defects tend to present in teeth that develop during pregnancy and in the first year of life and can be classified as:

1. Enamel hypoplasia (defect in quantity of enamel development)
2. Enamel hypomineralisation (defect in quality of enamel development), which commonly presents as:

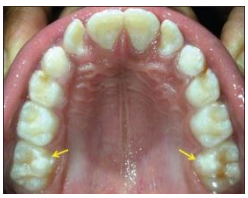
I. Deciduous Molar Hypomineralisation (DMH) affects the second baby molars, which emerge from 2 to 3 years of age

II. Molar Incisor Hypomineralisation (MIH) affects the first permanent molars and incisors, which emerge from 6 to 7 years of age.

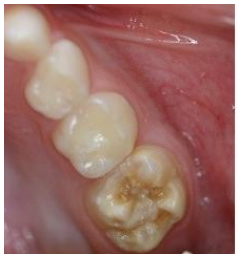
CLASSIFICATION OF MIH

Mathu-Muju and Wright in 2006 gave following clinical criteria in order to divide the defects in 3 severity levels:

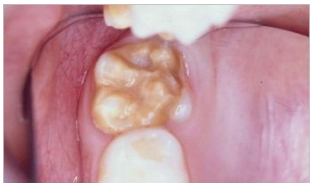
Mild MIH: Demarcated opacities are in non-stress-bearing areas of first primary molar, there are isolated opacities, no enamel loss and fracturing is present in opaque areas, there may be no history of dental hypersensitivity, there are no caries associated with the affected enamel and incisor involvement is usually mild if present



Moderate MIH: Intact atypical restorations can be present, demarcated opacities are present on occlusal/incisal third of teeth without post eruptive enamel breakdown, post eruptive enamel breakdown or caries are limited to one or two surfaces without cuspal involvement, dentinal sensitivity generally reported to be normal, aesthetic concerns are frequently expressed by the patient or parent.



Severe MIH: Post eruptive enamel breakdown is present and frequently occurs as the tooth is emerging, there may be history of dental sensitivity, often widespread caries is associated with the affected enamel, crown destruction can readily advance to involve the dental pulp, defective atypical restoration is present, aesthetic concerns are expressed by patient.



ETIOLOGY OF MIH

Etiology of MIH is somewhat complex with systemic and genetic factors disrupting normal amelogenesis in the affected teeth. A variety of systematically acting medical factors contributing to MIH are prenatal, perinatal and postnatal illnesses, low birth weight, antibiotic consumption and toxins from breastfeeding. The main possible important etiological factors to remember that between 28 weeks in uteroand the first 10 days of life ameloblasts initiate amelogenesis in the first permanent teeth to be formed, the first primary molar, followed by other teeth later in time. If the function of ameloblasts get disturbed, temporarily or permanently, then depending upon time of insult, enamel hypoplasia or enamel hypomineralization is produced.

Many conditions affecting the enamel matrix pH, i.e. respiratory acidosis and abnormal oxygen levels resulting from hypoventilation, resulting in enamel hypomineralization. Lack of calciumphosphate in the area of crystallites may affect as reduced calcium deposits and reduced ratio of calcium/phosphorus again leading to enamel hypomineralization. Maternal pyrexia has been shown experimentally to have much more influence on amelogenesis, ranging from ameloblasticdysfunction to complete cellular degeneration[47]. In case of maternal diabetes hypocalcemia in the mother and oxygen shortage problems to the infant may result in enamel hypomineralization[48].

Environmental factors - Some of environmental factors have been said to be capable of causing enamel defects. These systemic disturbances consist of prenatal, perinatal and postnatal problems, malnutrition, intoxications, infectious diseases and a range of other medical conditions.

Prenatal conditions -Such as prolonged maternal nausea and vomiting,different fluids and electrolytes as well as nutritional status sometimes leads to fetal biochemical disturbance. Use of myometrium spasmolytics medication may produce side effects such as nausea, vomiting and fetalhypocalcemia which may again disturb the amelogenesis[49].Perinatal medical conditions appear to be associated with hypocalcaemia and hypoxia.

During **perinatal period**, various medical conditions affect the infant's health may be in combination or individually. Prolonged delivery, preterm delivery and twin deliveries are among the very frequent perinatal problems/conditions. MIH is reported to be higher compared to normal children in such cases[50].

Postnatal -Special cause is mainly infectious childhood illnesses, high fever, medication (antibiotics), environmental toxicants, breastfeeding and use of fluorides. Illnesses such as otitis media,pneumonia, asthma, urinary tract infections and chicken pox been positively associated with MIH[51,52].

Other causativefactors to be considered are oxygen starvationof the child combined with a low birthweight, calcium and phosphate metabolicdisorders and frequent childhooddiseases.[10-12] vaccines given during earlychildhood have also been suggested as apossible cause. The use ofantibiotics has also been implicated butantibiotic use in most cases related tooccurrence of diseases, so it is difficult to distinguish whether the association with MIH is caused by the antibiotic or by theillness itself.

Sometimes, it was said that compared with new born delivered vaginally, those delivered by elective caesarean section, at around full-term had an increased risk of overall and serious respiratory illnesses, conditions often associated with hypoxia[53].Also, the commonly used spinal anaesthesia for caesarean section has a frequent complication of maternal hypotension that can be associated with severe nausea or vomiting which occasionally produces infant hypoxia[54].

Intoxications

The main responsible factor affecting people is probably fluoride. The relationship between DDE and fluoride consumption has been established for over 80 years[55,56]. The prevalence of enamel defects increases with increasing levels of fluoride in the drinking water [57]. Fluorotic defects can vary from minor white striations to small or extensive areas of opaque enamel [58]. However, fluorotic defects do not have unique characteristics which allow them to be differentiated from defects caused by other factors. The level of strontium in the drinking water has been shown to be associated with the frequency and severity of enamel mottling same as that of fluorosis[59]. It is said that, less severe the defect, greater the problem of positive diagnosis for fluorosis [60,61].

Recently, **Finnish** studies have focused on dioxins as being a causative agent for developmental defects of permanent first molars. Prolonged breast-feeding might increase mineralization defects in teeth because of environmental contaminants such as dioxins or dioxin-like compounds in breast milk interfering with enamel maturation. However, a Swedish study which identified similar levels of dioxins in breast milk failed to reproduce the findings[62.63]. Wozniak reported that elevated levels of chemical compounds, e.g. fluorine, ammonia and sulphur, in the atmosphere increased the incidence of diffuse white/creamy mottling and lines in the 14 to 15 years old schoolchildren[64]. Other intoxications include hypervitaminosis D, chronic lead poisoning, diphosphonate and polychlorinated biphenyl poisoning[65].

Malnutrition

Rugg-Gunn and his collegues reported that boys of 14 years classed as malnourished, by height for age percentage of the median of reference population and had a higher prevalence of enamel defects than those with well-nourished. Moreover, rickets due to vitamin D deficiency has been reported as a cause of enamel hypoplasia. Animal experiments have also demonstrated that a lack of vitamins A and D may cause enamel hypoplasia [66,67]. However, nutrition was not observed to be a significant aetiological factor in well-nourished New Zealand children[68].

PREDISPOSING FACTORS

Hypomineralization is thought to be resorptive potential of ameloblasts and proteolyticenzyme prevention which results in protein retention and interference with crystal growth and enamel maturation. Most common factors are natal and early post natal development.

Systemic illness- Condition common in first 3 years, as respiratory diseases, asthma, otitis media, tonsillitis, chicken pox, measles and rubella appears to be associated with MIH.Antibiotics may cause to occur which may cause difficulty in identification as whether it is associated with disease or antibiotic.[80] Children with poor general health and systemic conditions have more of the DDEs. Systemic conditions include nutritional deficiencies, brain injury, neurologic defects, cystic fibrosis, epileptic syndromes and dementia, atopia, lead poisoning, repaired cleft and palate, radiation treatment, opthalmic conditions, GIT[81].

Gestational age- Preterm birth also been associated with increased prevalence of enamel defects including hypomineralization and hypoplasia in permanent dentition[82].

Effect of low PH- PH regulation during mineralization is important for normal crystal deposition and growth.

Lack of calcium phosphate- A normal serum calcium level is important for initial dentin mineralization and proper enamel matrix secretion and mineralization. As disturbance in this metabolism play a role in development of hypomineralized enamel.Proteins like amelogenin, ameloblastin and enamelin are essential for enamel matrix formation which are secretory calcium binding phosphoprotein gene family, controlled by vitamin D[10] and also the amelogenins during enamel mineralization at secretory and early maturation stages like

enamelysin (MMP-20), calcium dependent matrix metalloproteinase. Means, hypocalcemia may predispose a child for development of MIH[83].

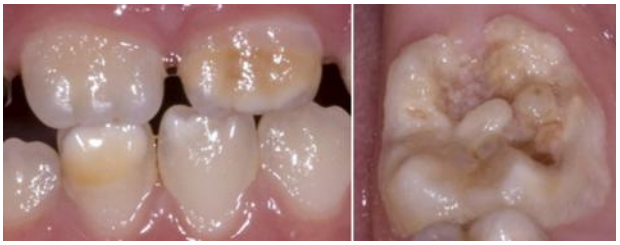
Duration of breast feeding-Association of Polychlorinated Dibenzo p-dioxins (PCDDs) present in breast milk and enamel hypomineralization have been made according to clinical and laboratory studies. PCCDs belongs to class of environmental pollutants are called as polyhalogenated aromatic hydrocarbons. Their accumulation in tissue lipids and in food chain may result in chronic low level exposure in humans. In infancy, children can be exposed to these compounds mainly via breast feeding. An infant may get 25% of mothers dioxin load via lactation[84].

PATHOLOGY OF HYPOMINERALIZATION

Enamel is a highly mineralized tissue of ectodermal origin, secreted from ameloblasts that differentiate from the internal dental epithelium. Hypomineralization is thought to follow deposition of full thickness of enamel matrix. The transitional ameloblast are considered to be most vulnerable and when these cells do not undergo complete maturation, full-thickness hypomineralization occurs[72].Enamel maturation involves: a) the removal of acid-labile mineral; b) replacement with more acid-resistant apatite; and c) an influx of calcium and phosphate ions, increasing the crystal width and thickness. Disturbed resorptive potential of ameloblasts and inhibition of proteolytic enzyme leading to protein retention and interference with crystal growth and enamel maturation may result in enamel hypomineralization.[73]Hypoplasia is a defect associated with a reduced quantity and thickness of enamel. Sarnat and Schour believed that, the morphologic unit of enamel hypoplasia was the enamel pit, which resulted from a cessation of ameloblastic activity. In a clinical investigation of anxious patients, whose childhood medical histories were available and said that a narrow zone of defect indicated a disturbance of short duration or an acute disease, while a wide zone indicated a disturbance of long duration or chronic disease [74,75].

Clinical features of MIH

Molars and incisors affected by MIH presents demarcated opacities, which are whitish-yellow or yellowish brown in colour. The affected Permanent First Molar may undergo post-eruptive enamel breakdown because of occlusal loading as incisors rarely presents post-eruptive enamel breakdown. MIH should not be mistaken for enamel hypoplasia, which is a quantitative developmental defect resulting from deficient enamel matrix formation[87]. Clinically, in cases of hypoplasia, the margins are smooth, while in MIH the borders are irregular[88].Affected molars may at times be hypersensitive and difficult to anesthetize. The structural defect of enamel of the molars affected by MIH may lead to early caries involvement and rapid progression which may be increased by difficulty in brushing those acutely sensitive teeth.



Older children may have more severe lesions than younger ones which may be because affected teeth have undergone post-eruptive enamel breakdown under masticatory load. Severity of MIH can vary between different individuals but it can also vary within the mouth of a single individual, as not all Permanent First Molar will be affected to the same degree, indeed some molars may apparently be unaffected in some cases.

B. Microstructure of hypo mineralized enamel

Severity of hypomineralization correlated positively with increasing carbon and decreasing calcium and phosphorus concentrations using secondary ion mass spectrometry and X-ray microanalysis. Yellow-brown defects have lower Knoop hardness values and greater porosity than white defects and normal enamel. Nano-indentation studies have shown significantly lower values for hardness and modulus of elasticity than seen in unaffected enamel[89]. Under scanning electron microscopic analysis, these defects revealed increased porosity and disorganized rod structure of fractured surfaces.

C. Surface protein content of hypomineralized enamel

Hypomineralized enamel was found to have from 3 to 15 fold higher protein content than normal but a near normal level of residual amelogenins. It was found to have accumulated various proteins from oral fluid and blood, with differential incorporation depending on integrity of enamel surface. Pathogenically, these results areresponsible for pre-eruptive disturbance of mineralization involving albumin and in cases with post-eruptive breakdown, subsequent protein adsorption on the exposed hydroxyapatite matrix[90].

D. Dentin characteristics of MIH affected teeth

Fagrell et al have found that, oral bacteria may penetrate through hypomineralized enamel into the dentinal tubules and create inflammatory reactions in the pulp, thus possibly contribute to hypersensitivity of teeth with MIH. In sections where bacteria were found in the cuspal areas or deeper in the dentin, a zone of reparative dentin was found, and in sections from one tooth, the coronal pulp showed an inflammatory reaction with inflammatory cells. The dentinal tubules with odontoblastic processes were mainly filled with bacteria.

E. Pulpal status of hypomineralized first permanent molars

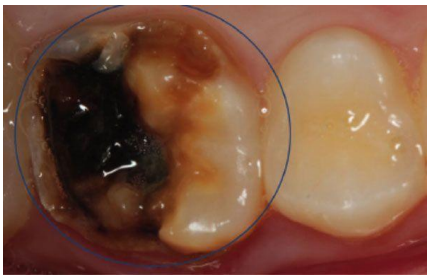
It was found that, the pulps of non-carious hypomineralized Primary First Molar present the changes indicative of inflammation. Innervation density was significantly greater in the pulp horn and sub odontoblastic region of hypomineralized teeth than in sound teeth. Immune cells were most abundant within pulps of hypomineralized teeth exhibiting enamel loss. Vascularity was found to be similar for both hypomineralized and sound teeth, but was significantly greater in hypersensitive hypomineralized samples[91].

DIAGNOSIS

As diagnostic criteria of molar-incisor hypomineralization according to EAPD

a) **Tooth hypersensitivity**: It often present from outset, which especially manifests itself through changes in temperature or contact & should be treated fast as it will determine whether any other problems will arise. In fact, as affected molars are sensitive to brushing, they will be avoided by children, which increases risk of decay[95].

b) **Rapidly developing decay**: Physiological immaturity of hard tissues, as well as fragile nature of enamel, associated with a bucco-dental hygiene problem presented previously, will cause serious and fast, irreversible damage to coronal structures. Moreover, these hypoplastic areas form a reservoir for plaque and food residues.



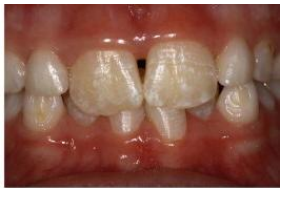
c) **Anaesthetic difficulties:** Hypersensitivity and rapid damage to the tissues will cause chronic pulpal inflammation, preventing effective local anaesthesia. Loco-regional techniques (Spix), osteo-central techniques or even electronically assisted local anaesthesia, such as Quick Sleeper or Wand may be useful. Quick Sleeper enables optimum performance of all anaesthesia, including osteo-central.

d) **Problem of restoration**: Adhesion of restorative material is low on soft, hypomineralised enamel, so the risk of early loss of restoration and development of secondary decay is higher. Clinical handling, treatment (short, medium and long-term) and progress as early MIH diagnosis (cleaning, magnifying glasses, microscope) and prevention of decay (fissure sealing) and loss of substance due to rapid erosion caused by chewing forces.

DIFFERENTIAL DIAGNOSIS OF MIH

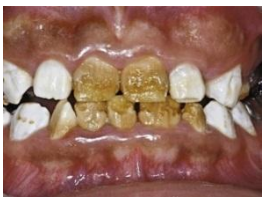
Teeth with developmental defects of enamel may present similar, regardless of etiology, and the developmental defects of enamel hypoplasia may be sometimes confused with MIH.

1. Fluorosis



This is associated with history of fluoride ingestion during enamel development. Number of teeth involved depending upon fluoride exposure time. Clinically, it presents as diffuse, linear, patchy or confluent white opacities without a clear boundary. The severity may range from barely perceptible striations in the enamel to more or gross disfiguration with almost complete loss of external enamel. It affects teeth in a symmetrical, bilateral pattern unlike MIH which is asymmetrical. Moreover, teeth affected by fluorosis are caries-resistant while in MIH they are caries-prone.

2. **Amelogenesis Imperfecta (AI)**



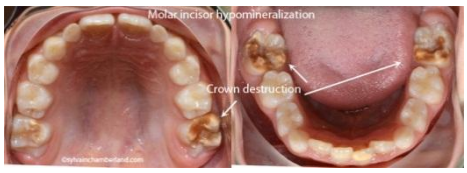
This is a genetic condition which results in enamel that is hypoplastic, hypomature or hypomineralised. In this condition, all teeth in both dentitions are affected and a familial history is often present.

3. Hypoplasia

This is a quantitative defect with reduced enamel thickness. The borders of hypoplastic enamel lesions are mostly regular and smooth, indicating developmental and pre-eruptive lack of enamel. The margins in MIH with post-eruptive enamel breakdown are sharp and irregular due to post-eruptive shearing of weakened enamel.MIH has increasing prevalence and have much impact on treatment required. Hence, it is highly recommended to increase the regular check up for children with history of repeated illness in the first year of life during the eruption of first permanent molars. In the same time, MIH represents a challenging disease, due to the serious problems that needs for both paediatric dentists and child and due to its interdisciplinary approach (general practitioner, paediatric dentist, physician, psychologist etc)[96].

4. Traumatic hypomineralization

This is associated with a history of dental trauma to the primary predecessor tooth. Periapical infection of primary tooth can disturb mineralisation of underlying tooth germ. It has a wide variety of clinical presentations differing in shape, outline, localisation and colour. It is often limited to one tooth and asymmetrical.



TREATMENT OPTIONS FOR MOLARS

It has been reported that, these teeth have 5 to 10 times more dental treatment need than molars without MIH. When managing these teeth, the first clinical consideration is whether to restore or extract. This depends on factors such as child's age, severity of MIH, pulp involvement, presence of third molar germ(s), restorability of the tooth/teeth, expected long-term prognosis, and long-term treatment cost.

1. Resin infiltrant: It improves the appearance of teeth by altering how light reflects off the teeth. Also known as erosion-infiltration, which uses a very low viscosity resin which is capable of penetrating demineralised enamel. Icon by DMG (Hamburg, Germany) is the only material available for this procedure. Its manufacturer recommends this material to treat incipient caries and/or carious white spot lesions reaching up to the outer third of dentine. Crombie et al, suggest that in MIH molars, the resin infiltrant has the potential to penetrate surfaces like hypomineralised cuspal inclines which are susceptible to post-eruptive enamel breakdown without interfering with occlusion or being broken by occlusal forces so this material can be

effective if used as 'fissure sealant', but the material here will be infiltrated into the hypomineralised enamel therefore this procedure, if done, is irreversible and it requires excellent isolation.[115]

2. Full or partial coverage

Preformed metal crowns (PMCs) can be used successfully in severely damaged MIH molars with high long-term survival rates. These crowns can prevent further post-eruptive enamel breakdown, manage sensitivity, and are not expensive, can establish correct interproximal and occlusal contacts, require no/little tooth preparation, and can be done in single visit. Non-precious metal, gold or tooth-coloured indirect onlays can be used in older children but the procedure is time-consuming, technique sensitive and expensive. Preformed malleable composite temporary crowns that come in different sizes (Protemp Crown Temporisation Material by 3M ESPE) can offer an aesthetic option. With this material some tooth preparation is required and the crown will require some adjustments but the process is considered easy and requires a single visit. There are as yet no studies that assess the performance of these crowns in MIH molars [117].

3. Extraction of severely affected molars

For severely affected First Permanent Molars with poor prognosis, extraction might be considered at the dental age of 8-10 years.[118]This will give the second permanent molars (SPM) an opportunity to drift into the First Permanent Molar position. Before a decision to extract the molars is made, full dental assessment should be carried out to check for presence, position and normal formation of the developing permanent dentition to ensure favourable orthodontic conditions.

TREATMENT OPTIONS FOR INCISORS

Aesthetic concerns are common in patients with MIH with incisor involvement. In young patients, these teeth should be treated in a conservative approach as they have immature anterior teeth with large and sensitive pulps.

1. Microabrasion
2. Tooth bleaching
3. Etch-bleach-seal technique
4. Resin infiltration
5. Composite restorations or veneers
6. Porcelain veneers

RESTORATIVE TREATMENT APPROACHES FOR MIH

1. GICs
2. Resin composite materials
3. Amalgam
4. Stainless steel crowns (SSC)
5. Laboratory fabricated crowns
6. Extraction and orthodontic approach

II. CONCLUSION

Despite a fall in the prevalence and in the speed of progression of dental caries disease.Often, the clinicians and the pedodontics can find first permanent molars and incisors withhypomineralised enamel defected. MIH is a public health problem which brings painful consequences,compromised aesthetic and it directly affect the quality of life of individuals suffering from MIH. It is a difficult and complex problem; hence all effort should be taken for proper knowledge of the MIH aetiology for more accurate diagnosis and more appropriate treatment.

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