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Original Research

Molar Incisor Hypomineralization: A Current Update

Dr. Bidyut Seal[1], Dr. Chitrita Gupta Mukherjee[2], Dr. Anuranjan Maharaj[3], Dr. Anamika Rani[4], Dr. Prerna Anand[5], Dr. Milind Rajan[6]

[1]MDS, Pediatric and Preventive Dentistry, Private Practitioner, Goalpara, Assam, India;
[2]Head of Department, Department Pediatric and Preventive Dentistry, Buddha Dental College, Patna, Bihar, India;
[3]Postgraduate student, Department Pediatric and Preventive Dentistry, Buddha Dental College, Patna, Bihar, India;
[4]Postgraduate student, Department Pediatric and Preventive Dentistry, Buddha Dental College, Patna, Bihar, India;
[5]Postgraduate student, Department Pediatric and Preventive Dentistry, Buddha Dental College, Patna, Bihar, India;
[6]Postgraduate student, Department of Pedodontics and Preventive Dentistry, Coorg Institute of Dental Sciences, Virajpet, Karnataka, India

ABSTRACT:
Molar incisor hypomineralization (MIH) is a developmental defect affecting teeth. The condition is defined as a hypomineralization of systemic origin of one to four permanent first molars frequently associated with affected incisors. The comprehensive care involved in treating affected children must address their behavior and anxiety, aiming at early diagnosis, remineralization and desensitization, prevention of caries and posteruption breakdown; restorations and extractions under pain-free conditions and maintenance. Present review of literature aims to discuss clinical presentation, pathogenesis and management of MIH in detail.

Keywords: Developmental defects, Molar Incisor hypomineralization, MIH

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Corresponding address: Dr. Bidyut Seal, MDS, Pediatric and Preventive Dentistry, Private Practitioner, Goalpara, Assam, India

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INTRODUCTION

Developmental defects of teeth are caused by complex interactions between genetic and environmental factors during tooth development. Enamel is a unique hard tissue which does not undergo remodelling like bone and as a result the structure of enamel is affected during its formation permanently. The changes induced during amelogenesis can be correlated with the timing and the nature of adverse biological events occurred. One such developmental defect of enamel occurring due to changes in the environment, causing permanent damage is molar incisor hypomineralisation. Historically, MIH has been suggested to be first seen as early as the 17th-18th century archaeological extracts of sub-adult samples of a London cemetery.[1]

Weerheijm et al (2001) defined the condition as a hypomineralization of systemic origin of one to four permanent first molars frequently associated with affected incisors and suggested the name MIH for it.[2] Clinically the condition is defined as a hypomineralization of systemic origin of one to four permanent first molars frequently associated with affected incisors. The prevalence of MIH is reported to vary between 2.4 and 40.2% in normal child populations. Management consists of early diagnosis, prevention of caries or posteruptive breakdown and interception if caries or breakdown has already ensued. Management challenges include difficulty in obtaining adequate anesthesia, increased incidence of caries, early pulpal involvement and gross destruction of clinical crown of affected teeth.[3] Present review of

literature aims to discuss clinical presentation, pathogenesis and management of MIH in detail.

ETIOLOGY

Molar incisor hypomineralisation has no hypoplastic defects as there is no discernable reduction in enamel thickness. Any reduction in enamel thickness seen clinically is indicative of posteruption disintegration of enamel. MIH is a qualitative defect in enamel classified as hypomineralised type that follows the natural incremental lines of enamel formation, from cusp tip to cemento enamel junction.[4,5]

Authors 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.[6]

Willmott et al. (2008) suggested asthma, pneumonia, upper respiratory tract infections, otitis media, antibiotics, dioxins in mother's milk, tonsillitis and tonsillectomy, and exanthamatous fevers of childhood. [7] Crombie et al. (2009) opined polychlorinated biphenyl/dioxin nutrition, birth and neonatal factors, and acute or chronic childhood illness/treatment, fluoride or breastfeeding.[8]

PATHOPHYSIOLOGY

The pathogenesis of MIH is considered to be a combination of factors. The mineralization of first permanent molar begin before birth (32nd week) and is completed around 4year of age development of upper central incisor closely follows, hence the cause of abnormal enamel formation must be present in this particular period.

Maxillary lateral incisor, mandibular central and lateral incisor are less frequently affected, difference in developmental timetable partly explain this. An early insult of the ameloblast may influence the cell in three different ways:

- **First:** The disturbance of the ameloblasts ability to produce the correct deposition of proteins. If the protein deposition is incorrect, subsequent accurate maturation is impossible. An increase of 3-15 times more protein is hypomineralised enamel has been found. Most of the amelogenin was resorbed. However, other proteins such as albumin which is a protein known to inhibit tooth mineralization persisted.
- **Secondly:** Ameloblasts are affected during late secondary phase or early maturation stage. These stages are very sensitive process. In secretory phase partially mineralised enamel is deposited to the whole enamel thickness. Later organic material and water in the enamel are removed to allow an additional influx of mineral. An insult of ameloblast during this phase is considered to result in hypomineralisation. If the protein resorption does not occur, calcium and phosphate will not be able to enhance the prism development, crystal hydroxyapatite does not form.
- **Thirdly:** Ameloblast in a transistional stage during enamel mineralization are the most vulnerable type of ameloblast. An insult of ameloblast at a specific time of this sensitive transition may affect the ameloblasts and cause a disturbed function.[9,10]

Table no 1: Predisposing medical conditions leading to MIH

Prenatal	Perinatal	Postnatal
• High episodes of fever , common cold • Maternal diabetes • Hypertension • Prolonged use of medication • Malnutrition • Vomiting upto last month imbalance in (fluid and electrolyte balance, hypoxia) • Chicken pox • Renal deficiency	• Caesarean section • Prolonged / Complicated delivery (Respiratory distress and birth asyphxia) • Twins • Premature birth – low birth weight • Haemorrhage • Cyanosis	• Repeated episodes more than 5 episodes of high fever • Otitis media • Bronchitis/ Bronchiolitis • Asthma • Laryngitis/Tonsilitis • Seizures – afebrile/febrile • Urinary infection • Encephalitis • Gastroenteritis • Exanthematous diseases[Incubator] • Dioxin present in Mothers milk

CLINICAL PRESENTATION

Clinically there may be hypoplasia involving one or more permanent first molars and incisors. Lesions are more frequent in the upper jaw than in the lower jaw. Occlusal surfaces are most commonly affected. Affected teeth show demarcated enamel opacities, ranging from white to brown, according to the severity of the disease and the hypoplasia that can be associated hypomineralized enamel is soft and porous and occasionally it undergoes to posteruptive breakdown, resulting in anomalous noncarious cavities. This rapid breakdown of the teeth often calls

for extensive restorative procedures. Molar incisor hypomineralization molars are fragile, and caries can develop very easily in these molars. The affected teeth are very sensitive to air, cold, warm, and mechanical stimuli.[11,12]

DIAGNOSIS

Any examination for MIH should be undertaken on clean wet teeth and the age of 8 years is optimum, as at this age all permanent first molars and most of the incisors are erupted. In addition, the permanent first molar teeth will be in a relatively good condition without excessive posteruptive breakdown. Judgments related to individual teeth (all FPM and incisors) should be recorded, helping in the correct diagnosis of the condition. Diagnostic criteria for hypomineralization of FPMs currently available are the modified defect of dental enamel (DDE) index given by federation dentaire international.[13.14]

Table no 2: Modified defect of dental enamel (DDE) index	
Mild	<30% of the tooth's enamel surface area 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

MANAGEMENT

Management of MIH consists of early diagnosis, prevention of caries or posteruptive breakdown and interception, if caries or breakdown has already ensued. Treatment options include symptomatic treatment for the increased sensitivity, sealing the caries prone pits and fissures in a noncarious freshly erupted MIH molar, restoration of the carious molars and in a badly broken down molar, stainless steel crowns provide a very successful option in children.

SYMPTOMATIC TREATMENT

Treatment options include for the increased sensitivity, sealing the caries prone pits and fissures in a non-carious freshly erupted MIH molar, restoration of the carious molars and in a badly broken down molar, stainless steel crowns provide a very successful option in children.

Molar incisor hypomineralisation molars exhibit significant sensitivity, especially in the early posteruptive period. Five percent sodium fluoride varnish (Duraphat) is well tolerated by young children and can be applied sparingly directly to first permanent molars. Repeated application has been shown to reduce dentine sensitivity.

Remineralization of the defects also has an added advantage of desensitization. Remineralization must be started as soon as possible after the eruption so as to enhance mineralization in the superficial layer of enamel and prevent development of caries. Remineralizing agents like topical fluoride, CPP-ACP can be used to enhance the mineralization of affected teeth. Fluoride may be applied topically as varnishes (5% NaF varnish), gels (1.23% APF gel), or solutions (2% NaF, 8% SnF2 and 1.23% APF solution).[3,15,16]

PREVENTION

It is essential to apprehend children with MIH alongside with their parents, preferably from a young age. Adhesive restorations such as Fissure sealants seem to be suitable in MIH cases. In case of mild MIH, this therapy is suggested especially when teeth are fully erupted and when moisture control is adequate. Long-term frequent follow up is mandatory since chances of failure and replacement requirements are high. Preventive treatment should be started as soon as the defective areas are accessible. It can be accomplished with fluoride or Casein Phosphopeptide-Amorphous Calcium Phosphate (CPP-ACP).[17,18]

CONSERVATIVE TREATMENT

Restorations with glass ionomer cement, composite, stainless steel crowns, full veneer metal-ceramic crowns are the different treatment options that are discussed in various studies.

Restorative options for affected molar vary from adhesive intracoronal restorations (composite is the material of choice) to extracoronal restorations (e.g., preformed metal crown). Esthetic solutions for affected incisors include microabrasion with resin composite or porcelain veneer to full veneer crowns in childhood and metal/metal-ceramic full veneer crowns in adulthood. Furthermore, adhesion of composite resins to hypomineralized tissue is clinically significant. The acid etched hypomineralized enamel that appeared on SEM studies is shown to be covered with a structureless layer and, enamel prisms appeared disorganized with thick prism sheaths and loosely packed crystallites. Bacteria were also found deep in porous hypomineralized enamel close to the enamel–dentin junction.

Extraction should be considered if teeth are non-restorable. In extraction cases, moreover, an interdisciplinary approach with an orthodontist should be planned for space management and restoration of function in these young children.[1,19,20]

CONCLUSION

Molar incisor hypomineralization describes the hypomineralization of systemic origin affecting one or more first permanent molars and are associated with affected incisors. Clinically, the defect presents as opaque lesions varying in colour from white to yellow or brown, with a sharp demarcation between the affected and sound enamel to posteruptive enamel breakdown. The destruction of enamel is so rapid and clinically, it presents as if the enamel has not formed at all. Management primarily depends on the age of the patient and severity of MIH at the time of intervention. Early intervention of this condition helps in preventing first permanent molar morbidity and mortality so that remineralization as a preventive measure can be initiated at early as possible.

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