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Case Report

Molar Incisor Hypomineralization: A case report

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Abstract

Molar Incisor Hypomineralization (MIH) is a developmental enamel defect affecting one or more permanent first molars, frequently associated with similar defects in the permanent incisors. This case report presents a 19-year-old patient with severe MIH affecting multiple first permanent molars and incisors, demonstrating the clinical challenges in diagnosis and management. The patient presented with increased dental sensitivity, aesthetic concerns, and post-eruptive enamel breakdown. The purpose of this case report is to discuss the clinical presentation, differential diagnosis, and comprehensive management approach for MIH, highlighting the importance of early detection and preventive strategies.

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INTRODUCTION

In 2001, Weerheijm introduced the term molar incisor hypomineralization (MIH), which refers to a developmental enamel defect of systemic origin affecting one or more first permanent molars and, in some cases, the permanent incisors.[1] These defects appear as clearly demarcated areas of poor enamel mineralisation.[1] MIH was initially described by the European Academy of Paediatric Dentistry as a qualitative enamel formation defect. Initially, the condition was reported to involve the first permanent molars (FPMs) and incisors; however, more recent evidence suggests that hypomineralization defects may occur in any primary or permanent tooth.[2] In healthy teeth, intact enamel usually has a smooth and hard surface, and after eruption, the outer enamel layer undergoes further mineralisation, becoming even more resistant. In contrast, enamel affected by MIH is porous and less mineralised, making it weaker and more prone to post-eruptive breakdown (PEB).[3] In MIH, the first permanent molars are especially vulnerable, often developing rapid tooth decay soon after they erupt.[2] As a result, the affected individual may experience pain, sensitivity, and difficulty in maintaining oral hygiene, while dentists often face challenges in providing long-term, successful treatment.

CASE REPORT

A 19-year-old female patient presented with the chief complaint of tooth sensitivity and discoloured teeth since early childhood. The onset of symptoms was noted around age 6, shortly after eruption of permanent teeth, when the parents observed that the molars & incisors appeared yellowish brown. The discolouration had progressively worsened since then, and the patient experienced tooth sensitivity while eating cold and hot foods. No history of trauma to the teeth or similar conditions in immediate family members. There were no associated symptoms of fever, swelling or systemic illness, but the patient reported self-consciousness about the appearance of her teeth, impacting social interactions.

Extraoral examination revealed no significant findings. Intraoral examination demonstrated maxillary & mandibular permanent incisors exhibit well-demarcated yellowish brown to creamy white opacities affecting the labial surfaces. The enamel appeared rough, porous, & irregular in contour. Marked post-eruptive enamel breakdown (PEB) is evident in several anterior teeth, particularly the mandibular incisors, with loss of enamel structure resulting in sharp, chipped, & irregular incisal edges. The maxillary first permanent molars showed extensive enamel defects with yellowish brown discoloration & severe structural loss involving the occlusal surfaces. The affected enamel appeared soft, friable & collapsed. Similar hypomineralized lesions with enamel breakdown were also noted in the mandibular first permanent molars. The remaining dentition appeared normal with no signs of caries. Oral hygiene was satisfactory. Gingival tissues were pink and healthy. The patient reported hypersensitivity to thermal stimuli on the affected teeth.

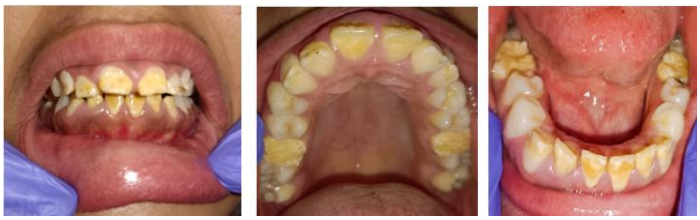

Fig 1: Intraoral pictures

Radiographic assessment with periapical radiographs revealed that the affected teeth demonstrate irregular, poorly mineralised enamel with reduced radiodensity compared to normal enamel, producing a mottled appearance. Radiographically, the incisors show thinning & loss of enamel at the incisal edges, correlating clinically with post-eruptive enamel breakdown. The first permanent molars exhibit extensive coronal enamel defects with areas of enamel loss & altered crown morphology. There was no evidence of caries in any of the affected teeth with normal pulp chamber morphology.

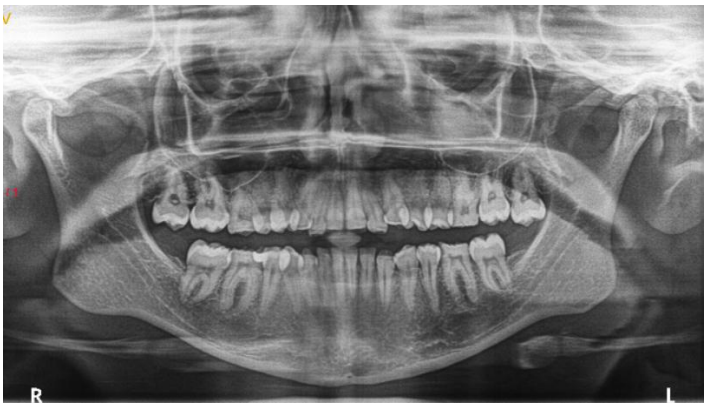

Fig 2: Orthopantomogram of the patient

Based on clinical and radiographic findings, a diagnosis of severe Molar Incisor Hypomineralization (MIH) was established. The following conditions were considered in the differential diagnosis:

Condition	Distinguishing Features
Amelogenesis Imperfecta	Affects all teeth in both dentitions; genetic inheritance pattern; ruled out due to selective involvement of first molars and incisors.
Fluorosis	Presents with diffuse opacities; affects multiple teeth symmetrically; no post-eruptive breakdown; patient had no history of fluoride exposure.
Enamel Hypoplasia	Characterised by quantitative defects with reduced enamel thickness, grooves or pits; ruled out as enamel thickness was normal in unaffected areas
Dental Caries	No evidence of demineralisation at cavity margins; lesions were demarcated; no bacterial involvement; ruled out radiographically

DISCUSSION

Molar incisor hypomineralization (MIH) is recognised as a common developmental enamel defect, affecting as many as of 25% children globally.[4] Despite its high prevalence, the precise underlying causes remain unclear, with current evidence suggesting a multifactorial origin involving systemic influences during enamel formation.[4] Exposure to certain pharmacological agents—particularly antibiotics and anticonvulsants administered during pregnancy or early childhood—has been implicated as a potential risk factor, with associations reported between their use and an increased likelihood of enamel developmental defects.[5] Other contributing factors include a history of childhood illnesses, episodes of birth-related hypoxia, and the occurrence of hypomineralized second primary molars.[6] These conditions are thought to compromise enamel formation, thereby increasing the likelihood of defects in the permanent dentition. Recent studies highlight a strong genetic contribution to molar-incisor hypomineralization (MIH). Variants in enamel-related genes such as MMP20, AMBN, ENAM, and AMELX, along with immune response genes like IL4, IL17A, and TGFBR1, have been linked to increased susceptibility and severity.[7] Studies further support heritability, while genome-wide analyses suggest novel associations (e.g., SCUBE1), underscoring the complex interplay of gene-gene interactions in MIH development.[8,9]

The diagnostic guidelines proposed by *Weerheijm et al.*[10] remain central to identifying and classifying molar–incisor hypomineralization (MIH). These criteria emphasise the clinical appearance of clearly demarcated enamel opacities, which may vary in shade from white to yellow or brown. A hallmark of MIH is the tendency for post-eruptive enamel breakdown (PEB), leaving teeth vulnerable to structural loss. In advanced cases, this fragility can necessitate atypical restorations in molars or, when damage is extensive, even the extraction of the affected teeth.[11]

Management of molar incisor hypomineralization (MIH) remains challenging due to its variable severity and associated sensitivity. Preventive strategies such as fluoride varnishes, casein phosphopeptide–amorphous calcium phosphate (CPP-ACP), and dietary counselling aim to strengthen enamel and reduce discomfort.[11] Minimally invasive restorative approaches, including glass ionomer cement, composite resins, and resin infiltration, have shown varying success depending on lesion severity.[7] Advanced cases often require crowns or vital pulp therapies to preserve function. Adjunctive methods like low-level laser therapy and biomimetic hydroxyapatite pastes have demonstrated desensitising effects.[7,11] Overall, individualised treatment tailored to severity and patient needs is emphasised to ensure long-term success and improved quality of life.

CONCLUSION

Molar Incisor Hypomineralization represents a significant challenge in clinical dentistry due to its variable presentation, sensitivity issues, and propensity for rapid deterioration. Early recognition and diagnosis are paramount to successful management. A comprehensive, individualised treatment approach addressing both functional and aesthetic concerns, combined with aggressive prevention and regular monitoring, offers the best prognosis for affected patients.

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